



Research article

Extraction and evaluation for anti-diabetic effects of some Indian plant extracts

Narayana Methri¹, Rakesh kumar jat¹, Rambabu Boda*²¹ Department of Pharmacology, SJJT University, Jhunjhunu, Rajasthan, India² Department of Pharmacology, MLR College of Pharmacy, Dundigal (V, M), Telangana State, India**Corresponding author:** Rambabu Boda, ✉ rambabuboda34@gmail.com, **Orcid Id:** <https://orcid.org/4993-1964-8347-7893>

Department of Pharmacology, MLR College of Pharmacy, Dundigal (V, M), Telangana State, India

© The author(s). This is an open access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by-nc/4.0/>). See <https://jmpas.com/reprints-and-permissions> for full terms and conditions.

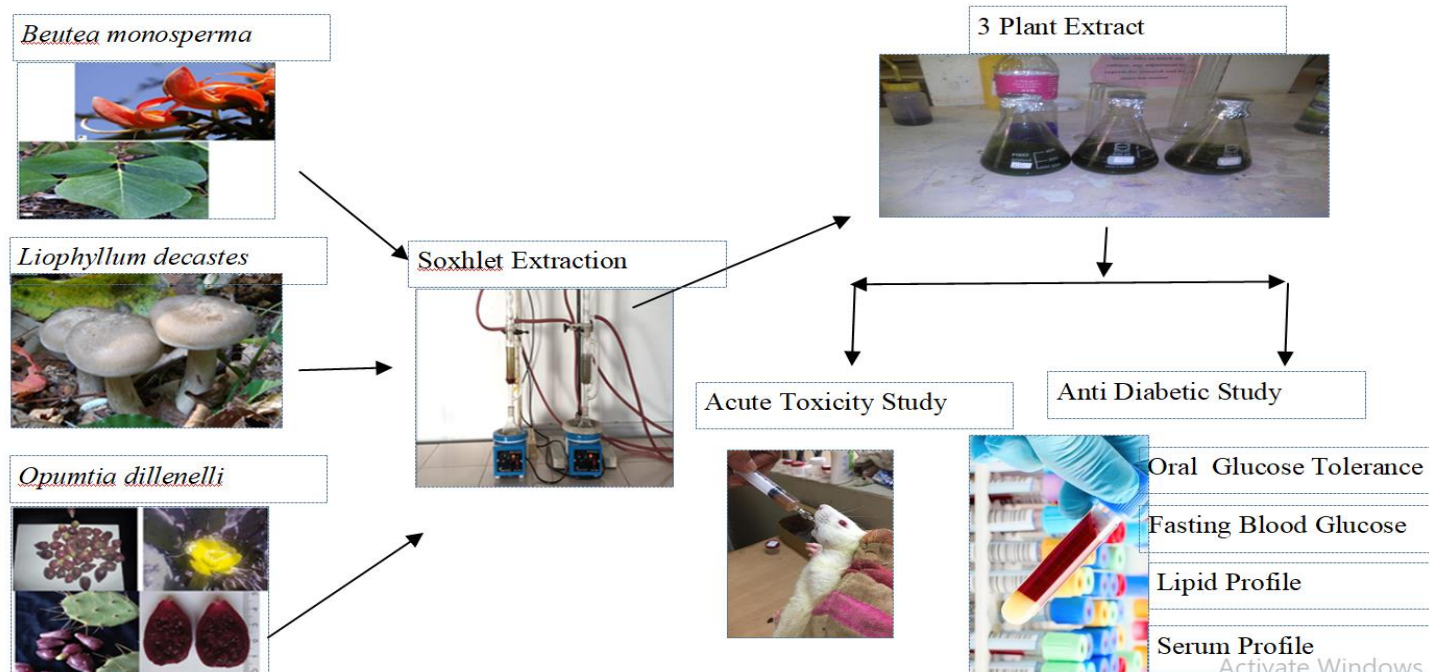
Received - 25-05-2024, Revised - 27-07-2024, Accepted - 03-09-2024 (DD-MM-YYYY)

Refer This Article

Narayana Methri, Rakesh kumar jat, Rambabu Boda, 2024. Extraction and evaluation for anti-diabetic effects of some Indian plant extracts. Journal of medical pharmaceutical and allied sciences, V 13 - I 5, Pages - 6755 – 6762. Doi: <https://doi.org/10.55522/jmpas.V13I5.6541>.

ABSTRACT

Diabetes is a metabolic disease associated with changes in the structure and function of several biological systems. The main cause of tissue injury is persistent hyperglycaemia. The past 20 years have seen increased expansion of the harmful effects of persistent hyperglycaemia, even if peripheral neuropathy and small and large vascular disease can also be used to explain diabetes-related organ failure. *Liophyllum decastes*, *Butea monosperma*, and *Opuntia dillenil* fruit extracts were shown to pass phytochemical testing. The study findings of *Butea Monosperma*, *Opuntia dillenil*, and *Liophyllum decastes* distilled (150 mg/kg) and (300 mg/kg) on STZ-tempted DM rats were paired with Glibenclamide. The glucometer method, serum and urine analysis, as well as factors including body weight, food consumption, glucose levels, and biochemical analysis, were used to determine the blood sugar rankings. *Liophyllum decastes* fungus fruit, *Opuntia dillenil* Planned fruit, and distilled *Butea monosperma* leaves all have anti-diabetic properties for diabetics.

**Keywords:** Diabetes mellitus, Blood Glucose Level, *Opuntia dillenil*, *Liophyllum decastes*, *Butea monosperma*.

INTRODUCTION

Diabetes mellitus is a clinical condition marked by unfavourable hyperglycaemia brought on by either a whole or partial absence of insulin or a cellular resistance to the insulin surge. It's a typical endocrine condition. In the ecosphere, diabetes is the primary cause of mortality. Diabetes is a condition in which the body is unable to produce or utilize insulin for the breakdown of glucose. Instead, your blood sugar levels rise to dangerous levels because the glucose stays in your system. Millions of people worldwide, including millions in India, suffer from diabetes, and the number is quickly increasing ^[1]. ^[2]. Diabetes's long-term reliance on medicine is a big issue. Adverse responses and lasting impacts on the metabolic process result from the imbalance and overload of the body's systems caused by synthetic medications and chemically modified natural molecules/substances. Health professionals are required by law to transition to alternative therapies like homeopathy and Ayurveda due to the unique side effects and expense of allopathic pharmaceuticals. Natural medications are important in treating infections; the majority of them expedite the standard healing procedure. In addition to formal medicine, several medicinal plants and their substances are employed in Indian botanical medicine to treat a variety of illnesses ^[3]. A fungal fruit of *Liophyllum decastus* and leaves of *Butea monosperma*, fruit of *Opuntia dillenil* is widely used in medical systems for treating a variety of ailments, including bleeding, antitumor, anticancer, analgesic, anthelmintic, analgesic, and anti-inflammatory conditions. Seeds and leaves contain anti-ovulatory and anti-implantation properties and are beneficial as diuretics, astringents, and haemostatics. The blossoms possess aphrodisiac and tonic qualities ^[4].

MATERIALS AND METHODS

Materials

The bulk of the chemicals used in this investigation were bought from Mumbai-based Sygma Chemicals Private Limited.

Procedure for Gathering and Extracting Plants

According to Dr. M. Madhava Chetty of the Department of Biological Sciences at Sri Venkateswara University in Andhra Pradesh, the fruit of *Opuntia dillenil* and the robust, disease-free foliage of *Butea monosperma* and fungus from *Liophyllum decastus* survived intact from the hills of Tirupati, India. After two weeks of shade drying, each plant's leaves were chopped into a coarse residue using a chopper. For a full day, 500 millilitres of methanol were continually mixed with 100 grams of powdered plant components.

Animals

Albino man Wister rats weighing between 180 and 230 grams were used in the study. They were housed in typical ecological settings with access to water and pelleted feed. Plant Refinement Research: Fruits of *Liophyllum decastes*, *Opuntia dillenil*, and *Butea monosperma* were distilled using 200 ml of distilled water as part of

the extraction process. STZ the Made Diabetes in Rats experiment was approved in accordance with OECD guidelines, and the I A E Committee and C P C S E A obtained ethical approval.

Methodology

Acute Toxicity Study

The OECD - 423 (Acute Toxic Class Method) standards were followed in the performance of the acute oral toxicity investigation. Albino rats of the same sex (n=3) were employed and chosen at random for the acute toxicity investigation ^[5,6]. The animals were given just water to eat during their overnight fast. Following stomach intubation, the extracts were given orally at a dosage of 5 mg/kg body weight, and the subjects were monitored for 14 days. A dosage is considered toxic if it results in the death of two of the three animals. To confirm the hazardous dosage, the same dose is given to another animal if death is noted in that animal. The process was repeated at larger dosages, such as 50, 300, and 2000 mg/kg body weight, if no mortality was seen. Every animal was housed in plastic cages at room temperature, with unrestricted access to water and pelleted meal.

Anti-diabetic Activity

Experimental Design

The treatment regimen was administered as follows to nine groups of six rats each.

Group I: Normal control (saline).

Group II: STZ treated control (150 mg/kg.ip).

Group III: STZ (150 mg/kg.ip) + *Butea monosperma* leaf extract (150 mg/kg, p.o),

Group IV: STZ (150 mg/kg.ip) + *Iodocea sechellarum* Fruit extract (150 mg/kg, p.o),

Group V: STZ (150 mg/kg.ip) + *Opuntia dillenil* leaf extract (150 mg/kg, p.o).

Group VI: STZ (150 mg/kg.ip) + *Butea monosperma* leaf extract (300 mg/kg, p.o).

Group VII: STZ (150 mg/kg.ip) + *Iodocea sechellarum* Fruit extract (300 mg/kg, p.o).

Group VIII: STZ (150 mg/kg.ip) + *Opuntia dillenil* Fruit extract (300 mg/kg, p.o).

Group IX: STZ (150 mg/kg.ip) + Standard drug, Glibenclamide (5 mg/kg, p.o).

Using an oral catheter, the medication is given every morning on alternate days for duration of four weeks. All animals were given unrestricted access to water but were required to starve for the whole night after the medication treatment. The following values were examined in blood and urine the next morning. As part of the study, measurements of body weight, fasting blood glucose, lipid profile, and serum profile (from trace levels) were made. For each group, the average daily food and drink intake was determined using the pellets that were consumed ^[7,8].

Oral Glucose Tolerance Test

There were nine groups of six rats apiece that were created from the rats. Prior to receiving therapy, every animal was fasted. Group II got STZ only; groups III, IV, and V received methanolic extracts of 150 mg/kg; groups VI, VII, and VIII received methanol extracts of 300 mg/kg; and group IX received STZ plus Standard medication Glibenclamide just (5 mg/kg). Group I was retained as the vehicle control group, receiving 5% between 80 p. o. In every instance, solely inside the car a glucose injection (3 g/kg, p. o.) was given to rats in Groups III to VIII half an hour after the drugs were given. Prior to the delivery of the medication and 30, 90, and 150 minutes following the injection of glucose, blood samples were obtained by retro orbital sinus puncture. A glucose determination kit was used to determine the serum glucose levels right away. (Sigma Diagnostic, Mumbai, India).

Collection of Blood Sample and Blood Glucose Determination

Following the pharmacological treatment, the animals were maintained in metabolic cages for a full day in order to collect pee and sample blood. All animals got unrestricted access to water throughout their overnight fast. Under mild ether anaesthesia, a retro orbital puncture was used the next morning to obtain a blood sample. Every week until the study's conclusion, blood samples were drawn from the rat's tail tip (i.e. 2 weeks). On days 4, 7, 10, and 14 of the trial, fasting blood glucose levels and body weight assessments were carried out. Using glucose test strips, an electronic blood glucose meter (One-Touch) can measure blood sugar. On day 14, blood was taken from the retro-orbital plexus of overnight fasting rats under light ether

anaesthesia and fasting blood glucose was determined. Serum was separated and analysed for Estimation of Cholesterol ^[9,10], serum Triglycerides ^[11] using the enzymatic DHBS colorimetric method, serum HDL-Cholesterol ^[12], serum ALKALINE PHOSPHATASE ^[13] SGOT/AST ^[14,15], SGPT/ ALT ^[16,17,18], Estimation of Serum Total Proteins ^[19,20], Glucose ^[21] were estimated by the hydrolysed phenol-amino-antipyrine method.

Analytical Methods

Fasting Blood Glucose Level

Following fifteen test days, all groups of rats were fasted, and blood samples were obtained from the tail area to measure blood glucose levels. At the end of the trials, data on body weight, food, and water intake were also noted and examined ^[22]. The findings demonstrated that the diabetes control group's fasting blood glucose levels were considerably higher than those of the normal control group over the study period (day 0–15). When compared to the diabetic control group, there was a notable drop in blood glucose levels in the Glibenclamide (5 mg/kg) group from day 0 to day 15. Similarly, as compared to the diabetic control group, the methanolic extract of *B. monosperma*, *Iodocea sachellarum*, and *Opuntia dillenil* fruits significantly lowered blood glucose levels in the treated rats (at doses of 150 and 300 mg/kg). The Glibenclamide group had the blood glucose level that was statistically lowest. Groups IV through VIII showed a dose-dependent impact on their fasting blood glucose levels (Table 1).

Table 1: Fasting Blood glucose Level

S. No	Treatments	Dose	Day 0	Day 7	Day 14	Day 21	Day 28
Group I	N Saline	0.9% NaCl	91.6± 4.2	92.0± 1.7	93.3± 2.5	92.7± 4.4	90.4±2.3
Group II	STZ	150mg/kg	291.0± 5.13	287.0±9.77	283.3±6.56	281.8± 7.26	278.5±12.3
Group III	STZ+ BME	150+150	281.83± 9.6	251.0±7.72	221.8±19.65	175.3± 18.35	147.3±6.28
Group IV	STZ+ISE	150+150	282.7± 9.18	260.6±5.46	245.1± 3.71	242.3± 3.37	211.0±7.61
Group V	STZ+ODE	150+150	270.53± 9.6	253.6±6.46	216.8±14.65	186.3± 12.35	161.0±7.61
Group VI	STZ+ BME	150+300	290.3± 6.73	204.5± 13.03	187.0±15.47	146.5± 14.57	123.6±3.81
Group VII	STZ+ISE	150+300	271.67± 9.18	251.8±9.17	234.17±3.71	222.8± 3.37	201.3±19.65
Group VIII	STZ+ODE	150+300	284.3± 6.73	242.8±9.17	213.8± 3.37	182.0±15.47	132.6±3.81
Group IX	Glibenclamide	5 mg/kg	293.83± 5.02	260.83±9.17	164.67±9.52	126.8± 6.85	103.8±3.76

Values are expressed as Mean SEM; n¼6. One-way ANOVA; followed by Tukey-Kramer multiple comparisons test: a P<

0.001incomparisonwithnormalcontroland ***P < 0.001 in comparison with the diabetic control.

Table 2: Body weight, food intake and water intake

S. No	Treatments	Dose	Day 0	Day 7	Day 14	Day 21	Day 28
Group I	N Saline	0.9% NaCl	121.2 ± 0.28	122.8 ± 0.20	123.6 ± 0.25	125.4 ± 0.47	127.5 ± 0.45
Group II	STZ	150mg/kg	129.8 ± 0.49	127.6 ± 0.38	126.4 ± 0.49	127.2 ± 0.38	126.9 ± 0.33
Group III	STZ+ BME	150+150	128.3 ± 0.33	131.6 ± 0.93	133.4 ± 0.36	134.1 ± 0.41	135.1 ± 0.27
Group IV	STZ+ISE	150+150	128.5 ± 0.28	128.3 ± 0.36	129.2 ± 0.16	129.4 ± 0.22	129.7 ± 0.20
Group V	STZ+ODE	150+150	132.6 ± 0.83	130.2 ± 0.16	129.5 ± 0.28	130.7 ± 0.20	131.4 ± 0.63
Group VI	STZ+ BME	150+300	127.8 ± 0.27	128.5 ± 0.28	128.2 ± 0.24	127.9± 0.27	126.7 ± 0.3
Group VII	STZ+ISE	150+300	128.5 ± 0.29	127.8 ± 0.31	126.4 ± 0.23	125.7 ± 0.22	125.9 ± 0.27
Group VIII	STZ+ODE	150+300	129.4 ± 0.28	128.8 ± 0.27	130.8 ± 0.16	132.4 ± 0.63	134.3 ±0.38
Group IX	Glibenclamide	5 mg/kg	134.3 ± 0.31	132.4 ± 0.38	133.7± 0.43	134.4 ± 0.31	135.3 ± 0.22

Values are expressed as Mean SEM; n ¼ 6. One-way ANOVA; Tukey-Kramer multiple comparisons test: aP< 0.001 in comparison with normal control; and ***P < 0.001 in comparison with the diabetic control.

Body Weight, Food Intake and Water Intake

On the first day of animal selection, at the onset of experimental diabetes, following seven days of treatment, and at the conclusion of the experiment, measurements were taken of body weight, food consumption, and water intake. Water consumption was evaluated using calibrated water bottles, and food intake was determined by counting the quantity of pellets ingested daily during the trial ^[23].

Lipid Profile

Following blood collection using retro-orbital techniques, the lipid profile was ascertained after 28 days of the trial. When

comparing the diabetic control group to the normal control group, there was a substantial rise in total cholesterol, triglycerides, low density lipoproteins, and very low density lipoproteins. In the treatment groups, the consumption of *Beutea monosperma* and *Iodocea Sachellarum*, *Opuntia Dillenil* fruit (150 and 300 mg/kg) resulted in a dose-dependent substantial rise in HDL levels and a significant drop in serum TC, TG, LDL, and VLDL levels. Conversely, the diabetes control group exhibited a substantial drop in high-density lipoprotein levels when juxtaposed with the normal control group (Table 3).

Table 3: Lipid Profile

S. No	Treatments	Dose	TC	TG	LDL	HDL	VLDL
Group I	N Saline	Saline	144.3±3.2	86.32±2.64	48.5± 4.6	36.8± 2.5	91.32± 3.1
Group II	STZ	150mg/kg	272.6±10.5	128.8±3.2	85.12±4.8	32.0± 1.9	169± 4.6
Group III	STZ+ BME	150+150	185.2±2.5	115.8±3.5	73.5± 3.7	35.12±4.3	120.27± 3.2
Group IV	STZ+ISE	150+150	159.6±5.6	119.7 ± 0.31	66.2± 1.9	37.63±2.1	93.6± 4.8
Group V	STZ+ODE	150+150	148.6±5.6	113.9 ± 0.31	68.5± 3.7	31.2± 1.9	97.3± 3.1
Group VI	STZ+ BME	150+300	131.4±5.03	127.2 ± 0.19	66.8± 0.25	22.1± 1.71	83.11± 2.4
Group VII	STZ+ISE	150+300	125.7±3.77	107.3±3.5	56.4± 0.23	28.3± 1.48	72.5± 0.54
Group VIII	STZ+ODE	150+300	127.4±5.03	104.3±4.5	54.8± 0.25	29.3± 1.12	94.1± 4.8
Group IX	Glibenclamide	5 mg/kg	147.2±5.3	93.20±2.62	50.35±4.34	37.7± 1.6	92.35± 2.68

Values are expressed as Mean SEM; n ¼ 6. One-way ANOVA; Tukey-Kramer multiple comparisons test: aP< 0.001 in comparison with normal control; and ***P < 0.001 in comparison with the diabetic control.

Table 4: Serum Profile

S. No	Treatments	Dose	Creatinine	Urea	ALT	AST
Group I	N Saline	0.5 ml	0.54±0.3	30.84±2.0	34.6±2.62	57.59±3.1
Group II	STZ	150mg/kg	2.5±0.1	63.6±1.7	55.7±3.4	68.5±2.3
Group III	STZ+ BME	150+150	0.99±0.3	44.3±3.7	43.4±3.0	66.4±1.7
Group IV	STZ+ISE	150+150	1.60±0.2	33.3±2.0	39.1±1.9	65.9±1.2
Group V	STZ+ODE	150+150	1.81±0.3	48.3±4.7	46.2±3.0	63.4±0.7
Group VI	STZ+ BME	150+300	0.79±0.3	34.3±3.8	33.4±3.0	56.4±1.1
Group VII	STZ+ISE	150+300	1.50±0.2	32.5±2.0	29.1±1.0	55.9±1.2
Group VIII	STZ+ODE	150+300	2.14±0.2	32.3±3.6	37.4±2.0	54.3±1.2
Group IX	Glibenclamide	5 mg/kg	2.58±0.1	32.4±4.0	35.8±2.7	56.7±2.9

Values are expressed as Mean SEM; n ¼ 6. One-way ANOVA; Tukey-Kramer multiple comparisons test: aP< 0.001 in comparison with normal control; and ***P < 0.001 in comparison with the diabetic control.

Serum Profile

The diabetes control group had considerably higher serum levels of both aspartate aminotransferase (AST) and alanine aminotransferase (ALT) than the normal control group. When comparing the Glibenclamide group (5 mg/kg) to the diabetic control group, there was a substantial drop in both ALT and AST. Similar to this, groups treated with *B. monosperma* and *Iodocea sachellarum* fruit extract, *Opuntia Dillenil* (150 and 300 mg/kg) showed a dose-dependent drop in ALT and AST levels in comparison to the diabetic control group. (Table 4)

RESULTS

Phytochemical Screening

Several qualitative phytochemical screening assays revealed the presence of carbohydrates, phytosterols, glycosides, lipids, tannins, and flavonoids, fixed oils, and alkaloids in the methanolic extract of *Opuntia dillenil* mushroom fruit. These fruits have increased flavonoid and glycoside contents.

Several qualitative phytochemical screening assays revealed the presence of alkaloids, carbohydrates, fixed oils, lipids, glycosides, flavonoids, tannins, and phytosterols in the methanolic extract of *Liophyllum decastes* mushroom fruit. These fruits have increased flavonoid and glycoside contents.

Several qualitative phytochemical screening assays revealed the presence of carbohydrates, glycosides, fixed oils, tannins, phytosterols, flavonoids, and alkaloids in the methanolic extract of *Butea monosperma* leaves. The methanolic extract under investigation has a greater concentration of flavonoids and glycosides.

Fasting Blood Glucose

The outcomes were noted and are displayed in Figure 1. On the 28th day of the trial, the blood glucose level of the treated group 2 was lower than that of groups IV, VI, and VIII, which were 211.0±7.61, 123.6±3.81, and 132.6±3.81 mg/dl, respectively. When

compared to group II's (the diabetes control group) findings from day 28, which were 278.5 ± 12.3 , the results are noteworthy.

Body Weight, Food Intake and Water Intake

From Day 7 to Day 28, the rats in groups IV, VI, and VIII gained weight. The weight increase contrasts with the weight loss symptom associated with diabetes, indicating an improvement in the treated groups' health state. The diabetic group weighed 126.9 g on average, whereas the other groups' average weights ranged from 128.3 to 129.7 g and 128.8 to 134.3 g. Figure II presents the findings.

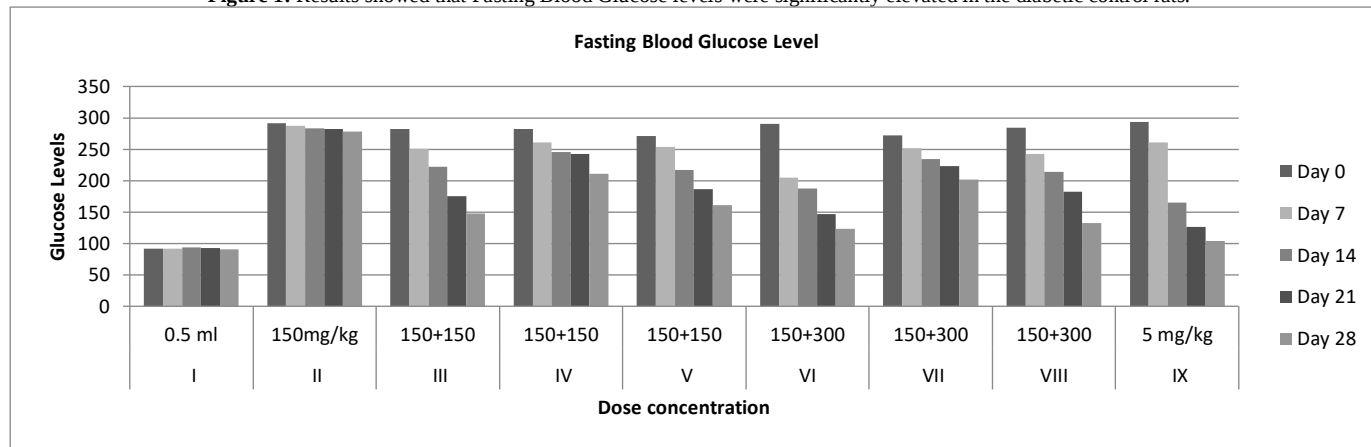
Lipid Profile

When compared to the diabetic control group, the lipid levels of groups VI, VII, and VIII demonstrate superior outcomes. In group VI and VII, VIII, the total cholesterol and triglyceride levels were reported as 131.4 and 125.7, 127.4, the LDL levels as 56.4 and 54.8, 66.8, the HDL levels as 28.3 and 29.3, 22.1, and the VLDL levels as 83.11 and 72.5, 94.5 mg/dl, respectively. When compared to group II's (the diabetic control group's) findings starting on Day 28, the results are noteworthy. The outcomes are displayed in Figure III.

Serum Profile

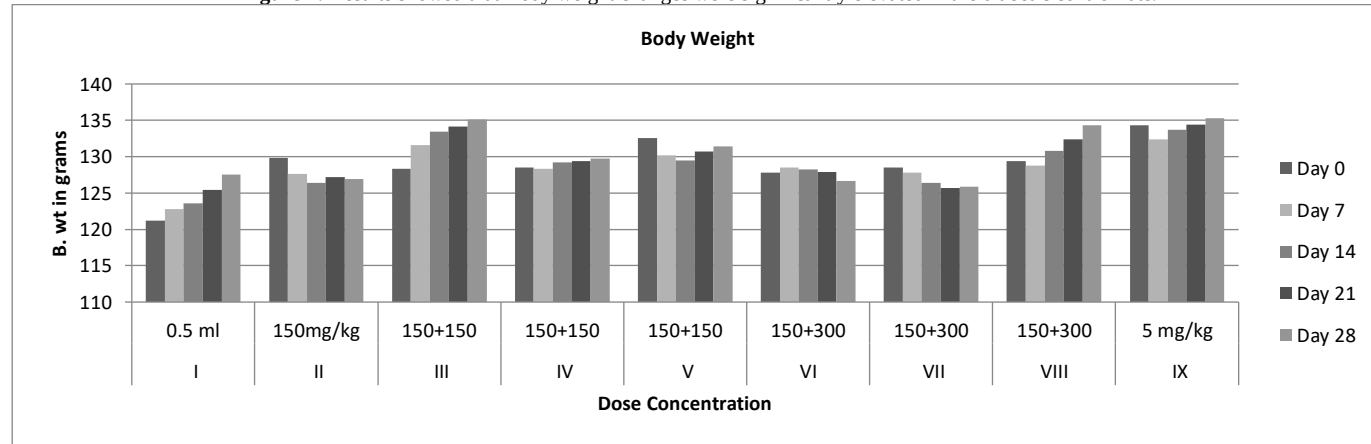
After 14 days of therapy, the plant extract at high dose (300 mg/kg) had a greater anti-hyperglycaemic impact than the entire plant extract at low dose (150 mg/kg). Treatment with streptozocin raised serum enzyme levels of cholesterol, LDL, creatinine, urea, and alkaline phosphatase while lowering HDL levels; however, Glibenclamide (5 mg/kg), *B. monosperma* plant leaf extracts, and *Iodocea sachellarum* fruit extract, *Opuntia Dillenil* reversed the effects of streptozocin (Table No IV). Figure 4 illustrates how the use of streptozocin affects the levels of AST and ALT plasma in diabetic rats. The results demonstrated that the diabetic control rats had considerably higher plasma levels of ALT and AST. In comparison to diabetic control rats, the administration of STZ (150 mg/kg) dramatically reduced the levels of AST and ALT. When compared to diabetic control rats, the administration of Glibenclamide (2 mg/kg) also decreased the levels of ALT and AST.

Figure 1: Results showed that Fasting Blood Glucose levels were significantly elevated in the diabetic control rats.

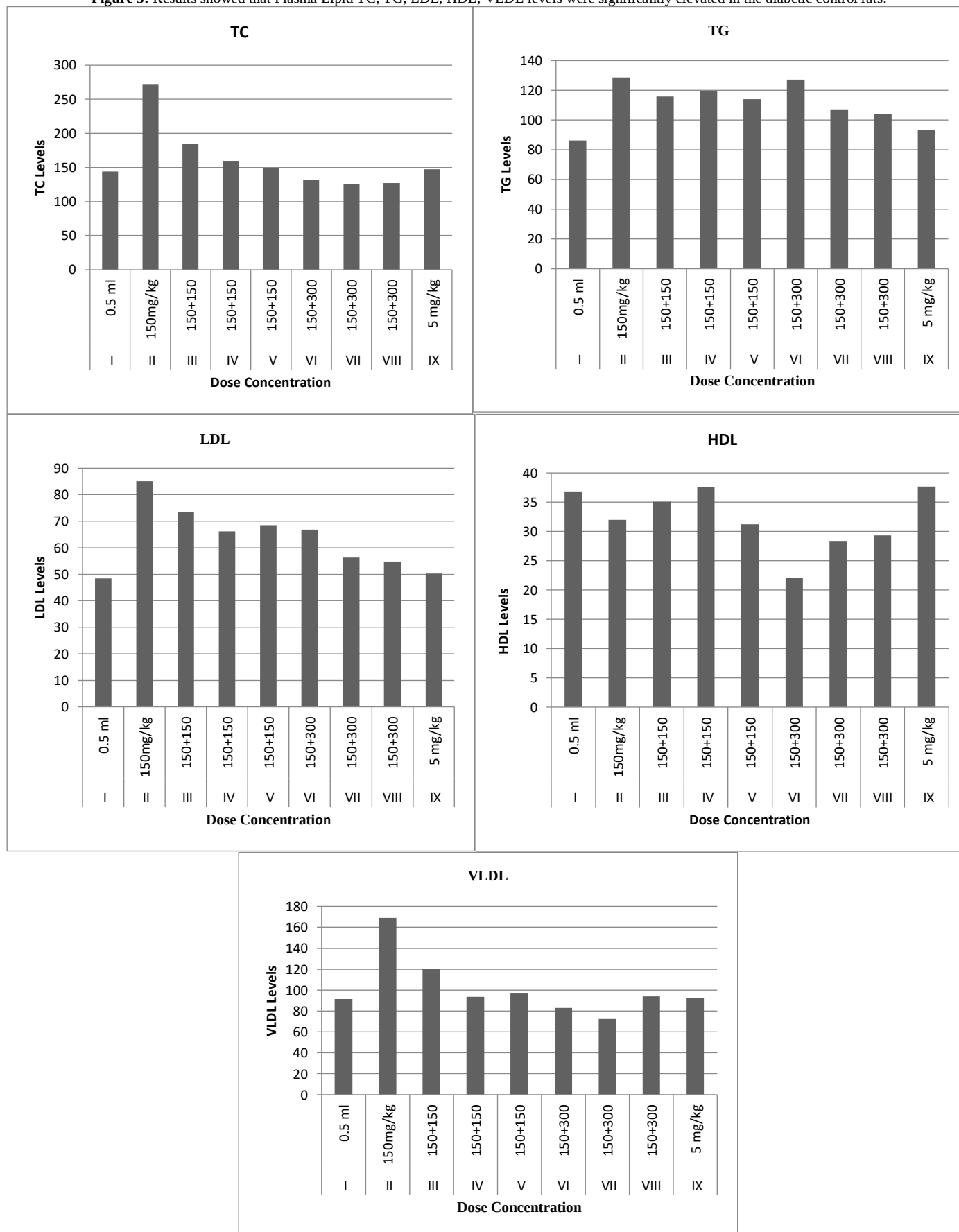


The values are expressed in terms of mean $\pm$ SEM (n = 9). ND nondiabetic, ND + PE-150 nondiabetic rats treated with PME at 150 mg/kg STZ-D diabetic, STZ-D + PE-150 diabetic rats treated with PME at 150 mg/kg, STZ-D + PME-300 diabetic rats treated with PME m. PE = Plant Methanolic Extract, ND = Nondiabetic, STZ-D = Streptozotocin-diabetic

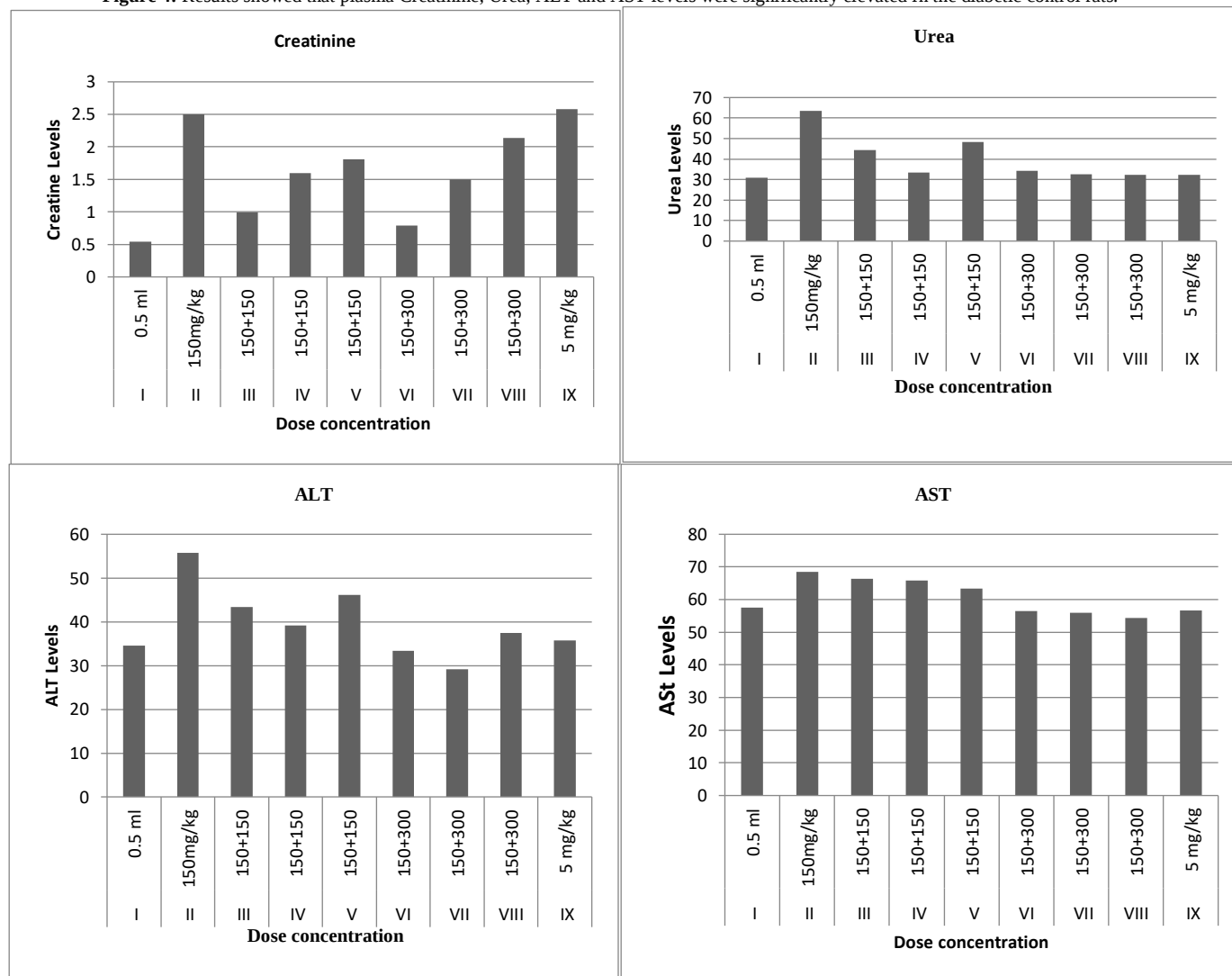
Figure 2: Results showed that Body weight changes were significantly elevated in the diabetic control rats.



The values are expressed in terms of mean $\pm$ SEM (n = 9). ND nondiabetic, ND + PE-150 nondiabetic rats treated with PME at 150 mg/kg STZ-D diabetic, STZ-D + PE-150 diabetic rats treated with PME at 150 mg/kg, STZ-D + PME-300 diabetic rats treated with PME m. PE = Plant Methanolic Extract, ND = Nondiabetic, STZ-D = Streptozotocin-diabetic

Figure 3: Results showed that Plasma Lipid TC, TG, LDL, HDL, VLDL levels were significantly elevated in the diabetic control rats.

The values are expressed in terms of mean $\pm$ SEM (n = 9). ND nondiabetic, ND + PE-150 nondiabetic rats treated with PME at 150 mg/kg STZ-D diabetic, STZ-D + PE-150 diabetic rats treated with PME at 150 mg/kg, STZ-D + PME-300 diabetic rats treated with PME m. PE = Plant Methanolic Extract, ND = Nondiabetic, STZ-D = Streptozotocin-diabetic

Figure 4: Results showed that plasma Creatinine, Urea, ALT and AST levels were significantly elevated in the diabetic control rats.

The values are expressed in terms of mean $\pm$ SEM (n = 9). ND nondiabetic, ND + PE-150 nondiabetic rats treated with PME at 150 mg/kg STZ-D diabetic, STZ-D + PE-150 diabetic rats treated with PME at 150 mg/kg, STZ-D + PME-300 diabetic rats treated with PME m. PE = Plant Methanolic Extract, ND = Nondiabetic, STZ-D = Streptozotocin-diabetic

DISCUSSION

The findings of this study demonstrate that in Wistar rats, experimentally induced diabetes and diabetic activity are reduced by chlorogenic acid. Thus, this relief is ascribed to the fact that experimentally generated hyperglycaemia and diabetes study are attenuated by chlorogenic acid obtained from the leaves of *Butea monosperma*, the fruit of the fungus *Liophyllum decastes*, and the fruits of *Opuntia dillenil*. Diabetes is a frequently recognized consequence that affects around 50% of people with the disease. This issue seems to be mostly brought on by the oxidative stress brought on by free radicals that is seen with the onset of diabetic activity [26].

A significant decline in diabetes cases was seen in this investigation [24]. Over the course of four weeks, there was a noticeable drop in glucose levels in this research when compared to the control group. This study validates the Antidiabetic properties of the leaves of *Butea monosperma*, the fruits of *Opuntia dillenil*, and the *Liophyllum decastes* mushrooms. According to experimental studies, giving *Butea*

monosperma leaves, *Liophyllum decastes* fruit, and *Opuntia dillenil* to Wistar rats that had been given streptozotocin-induced diabetes had a beneficial impact. Since diabetes is often diagnosed with weight loss, we kept an eye on the animals' body weight to verify the isolated action of chlorogenic acid. Diabetes is characterized by decreasing body weight, which was observed in the animals in the control group; in contrast, the treated group's body weight increased. This demonstrates how the treated groups' healing effects [25].

CONCLUSION

This study supported the traditional assertion of the tribal people and demonstrated the anti-diabetic efficacy of *Butea monosperma* leaves, *Liophyllum mushrooms*, and *Opuntia dillenil* fruit extracts in Wistar rats.

ACKNOWLEDGEMENTS

The author is Grateful to SJIT University, Churela, and Rajasthan.

Conflict of interest

The authors clearly point out that there is no conflict.

REFERENCES

- LM Xiu, A B Miura, K Yamamoto et al, 2001. Pancreatic islet regeneration by ephedrine in mice with streptozocin induced diabetes. *Am J Chin Med.* 29(3-4), Pages 493-500. Doi: 10.1142/S0192415X01000514.
- Mohammed Fazil Ahmed, Syed Mohammed Kazim, Syed Safiullah Ghorri, et al, 2010. Antidiabetic Activity of *Vinca rosea* Extracts in Alloxan-Induced Diabetic Rats. *Int J Endocrinol.* 6, Pages 500-516. 2010; Doi: 10.1155/2010/841090
- Namani Satyanarayana, Suresh V, Chinni, et al, 2022. Antidiabetic activity of *Solanum torvum* fruit extract in streptozocin-induced diabetic rats. *Front Nutr.* 9(10) Pages 3389-3394. Doi: 10.3389/fnut.2022.987552.
- Jun M, Foote C, Lv J. 2010. Effects of fibrates on cardio vascular outcomes: a systematic review and meta analysis. *Lancet.* 29, Pages 1875-84. Doi: 10.1016/S0140-6736(10)60656-3.
- OECD Guideline 423. 2001. Acute Oral Toxicity - Acute Toxic Class Method. OECD guideline for the testing of chemicals. France, Paris. Pages 1–14. Doi: <https://doi.org/10.1787/20745788>
- OECD. Test No. 420. 1996. Acute Oral Toxicity – Fixed Dose Procedure. Paris. 2002. Doi: <https://doi.org/10.1787/9789264071049-en>.
- Williams F, Saffran M. 2020. Insulin in the drinking water of rats. *Drug Delivery.* 3, Pages 81–85. 6(10), Pages 05137. Doi: 10.1016/j.heliyon.2020.e05137.
- Cpcsea. Compendium of CPCSEA 2018, 2018. Committee for the Purpose of Control and Supervision of Experiments on Animals. Ministry of environment, Forest and Climate Change. Govt of India. Pages 1–213.
- Slater T F, Sawyer BC. 1971. The stimulatory effects of carbon tetrachloride and other halogen alkanes on per oxidative reactions in rat liver fractions in vitro. General features of the systems used. *Biochem J.* 123(5), Pages 805-14. Doi: 10.1042/bj1230805.
- Abourbih S, Falcon KB, Joseph L. 2009. Effect of fibrates on lipid profiles and cardio vascular outcomes. A systematic review. *Am J Med.* 122(10), Pages 962-968. Doi: 10.1016/j.amjmed.2009.03.030.
- Davidson MH. 2000. Safety profiles for the HMG-CoA reductase inhibitors: treatment and trust. *Drugs.* 61(2), Pages 197-206. Doi: 10.2165/00003495-200161020-00005
- Joy TR, Hegele RA. 2009. Narrative review: Statin-related myopathy. *Ann Intern Med.* 150(12), Pages 858-68. Doi: 10.7326/0003-4819-150-12-200906160-00009.
- Brugts JJ, Yetgin T, Hooks SE, et al, 2009. The benefits of statins in people without established cardio vascular disease but with cardio vascular risk factors: meta-analysis of randomised controlled trials. *BMJ.* 30, Pages 338:b2376. Doi: 10.1136/bmj.b2376.
- Murphy SA, Cannon CP, Wiviott SD, et al, 2007. Effect of intensive lipid lowering therapy on mortality after acute coronary syndrome: a patient level analysis of the Aggrastat to Zocor and Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis in Myocardial Infarction 22 trials. *Am J Cardiol.* 1; 100(7), Pages 1047-1051. Doi: 10.1016/j.amjcard.2007.04.053.
- Hardman, Lombard, Gilman. 2023. Goodman & Gilman's pharmacological basis of therapeutics. Mc Graw Hill medical publishers. 10, Pages 977-986.
- Rang, Dale, Ratter, Flower, and Henderson: A Text Book of Rang & Dale's Pharmacology. Elsevier Churchill livingstone publishers, 7: 604,285-293. Doi: eBook ISBN: 9780323873987.
- Lukacinova A, Mojzis J, Benacka R, et al, 2008. Preventive effects of flavonoids on alloxan induced diabetes mellitus in rats. *Acta Vet. Brno.* 77, Pages 175-182 Doi: <https://doi.org/10.2754/avb200877020175>.
- Claudia ENM, Julius EO, Dogbert T, et al, 1996. Anti diabetic and Hyperlipidemic effects of *Laportea ovalifolia* (Urticaceae) in alloxan-induced diabetic rats. *Afric J of Trad Complement and Altern Medic.* 3(1), Pages 36-43.
- Leonard Tedong, Théophile Dimo, Paul Desire Djomeni Dzeufiet et al 2006. Anti-hyperglycemic and renal protective activities of *Anacardium occidentale* (anacardiaceae) leaves in streptozotocin induced diabetic rats. *Afric J of Trad Complement and Altern Medic.* 3(1), Pages 23-35. DOI:10.4314/ajtcam.v3i1.31136.
- Faizal P, Suresh S, Satheesh Kumar R, et al, 2009. A study on the hypoglycaemic and Hyperlipidemic effects of a ayurvedic drug RAJANYAMALAKADI in Diabetic patients. *Ind J of Clinic Biochem.* 24(1), Pages 82-87. Doi: <https://doi.org/10.1007/s12291-009-0014-1>.
- Froude T.S, Y.S. Medeiros. 2008. Animal models to test drugs with potential anti diabetic activity. *J of Ethnopharmacology.* 115, Pages 173-183. Doi: <http://dx.doi.org/10.1016/j.jep.2007.10.038>.
- P Roeschlau, E Bernt, W Gruber et al, 1974. Enzymatic determination of total cholesterol in serum. *Z Klin Chem Klin Biochem.* 12(5), Pages 226. Doi: <https://pubmed.ncbi.nlm.nih.gov/4440114/>.
- B. W. Wilson, 1966. Automatic estimation of urea using urease and alkaline phenol," *Clinical Chemistry.* (12)6, Pages 360–368. Doi: <https://pubmed.ncbi.nlm.nih.gov/5911781/>.
- Choudhary SK, Chhabra G, Sharma D, et al 2012. Comprehensive Evaluation of Anti-hyperglycemic Activity of Fractionated *Momordica charantia* Seed Extract in Alloxan-Induced Diabetic Rats. *Evid Based Complement Alternat Med.* Doi: 10.1155/2012/293650.
- Faiyaz Ahmed, Siddaraju NS, Harish M, et al, 2012. Effect of *Butea monosperma* Lam. leaves and bark extracts on blood glucose in streptozocin-induced severely diabetic rats. *Pharmacognosy Res.* 4(1), Pages 33–36. Doi: 10.4103/0974-8490.91032.
- Gaikwad TA, Gawai N, Kale RH, et al, 2011. Evaluation of Antidiabetic Activity of *Butea monosperma* Preparation on Streptozocin Induced Diabetes in Rats. *Cur Pharmac Res.* (1)2, Pages 439-442. <https://jcpr.humanjournals.com/10.25166>.