Int J Ayurvedic Med,Vol 17 (2), 2026; 370-375

Ayurveda Sahiti Prabha's

International Journal of Ayurvedic Medicine

ISSN No: 0976-5921

Research Article

Acute oral toxicity and antidiabetic evaluation of Phala Trikadi Kwath extract in Streptozotocin-induced Wistar rats

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Received: 14-05-2025 Accepted: 21-06-2026 Published: 30-06-2026

Abstract

This study aims to determine the safety and antidiabetic potential of Phala Trikadi Kwath extract in Wistar rats. For the acute toxicity study, three groups with three animals each were given oral dosages of the test drug at 50,300 and 2000 mg/kg body weight. Then each animal was checked for symptoms of toxicity, once in the first 30 minutes, every 24 hours and for the next 14 days. For the OGTT test, Wistar rats weighing 150-200 g were selected for induction of diabetes using streptozotocin. Body weights were measured then OGTT was performed in fasted normal rats, randomised into five groups (n=6). They were treated with drinking water, extract or standard (glibenclamide 5mg/kg). Glucose was fed 30 minutes after administration. Blood was withdrawn under anaesthesia in 0,30,60, and 120 minutes of glucose administration and glucose levels, serum HDL, LDL, Cholesterol and liver function estimated. Histopathology of the pancreas was done after euthanasia. In an acute toxicity study, no dose amounts resulted in death, while higher doses showed some alterations in skin and fur. Hence, 300mg/kg was selected to evaluate the therapeutic effect. Significant control of diabetes and other parameters was observed. The Islet cells of the 60 mg/kg body weight extract-treated group were found to be in a normal position.

Keywords: *Acute oral toxicity, Antidiabetic, Phala Trikadi Kwath, Oral glucose tolerance test, Streptozotocin, Wistar rats*

Access this article online

Website: https://ijam.co.in

DOI: https://doi.org/10.47552/ijam.v17i2.6146

Introduction

"*Phalatrikadi kwatha*", which is composed of Triphala (combination of *Amla- Embellica officinali*s Gaertn, *Harad -Terminalia chebula* Retz and *Baheda-Terminalia bellirica* Gaertn), *Motha* (*Cyperus rotundus* L), *Daruhaldi* (*Berberis aristata* DC) and *Indrayan* (*Citrullus colocynthis* L Schrad) root When decoctions prepared from the *Kwath Churna* and turmeric powder (*Curcuma longa* L) is added as *Prachep Dravya*, Consuming the decoction can cure all types of *Prameha* (diabetes) (1) as It has been mentioned in *Sarangdhar Sanhita shloka* number 111, from *Madhyam Khanda*. To develop scientific evidence in Ayurvedic principles, drug therapies by way of integrating ancient wisdom with modern technology and to bring Ayurveda to the people through innovations related to diagnostics, preventive, promotive as well as treatment methods and also introduce scientific research for sustained availability of quality natural resources, to translate them into product and process and in synergy with concerned organisations to introduce these innovations into public health systems. With this vision keeping in mind, the above combination of medicines was chosen to study its efficacy in curing diabetes. *Phala trikadi kwatha churna* (2) (3)is a combination of six potent drugs, such as *triphala*, composed of *Amla* (*Emblica officinalis* Gaertn.), *Baheda* (*Termininalia bellirica* (Gaertn.) (Roxb), and *Harad* (*Terminalia chebula* Retz) , along with *Motha* (*Cyprus rotundus*), *Indrayan* (*Citrillus colocynthus* (L) Schrad), *Daruhaldi* (*Berberis aristata* DC)and Haldi (Curcuma longa L) Although this combination of drugs has not been tested invivo previously but several animal studies has been conducted in the individual plants. Such as (4) (5)Gallic acid in *E. officinalis* has antidiabetic potential, which represented the upregulation of vPakt, PPAR-X and Glut 4 in gallic acid-mediated antidiabetic activity, thus providing potential remedies for diabetes. *Terminalia chebula* (6) showed antidiabetic activity and many other medicinal properties based on in vitro and in vivo studies. (7) *Terminalia Bellirica* fruit extract showed strong antioxidant and notable alpha-amylase inhibitory activity during in vitro evaluation and hypoglycemic effect in alloxan-induced diabetic rats, suggesting antidiabetic potential. It was noted that a

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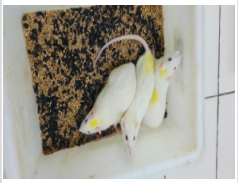
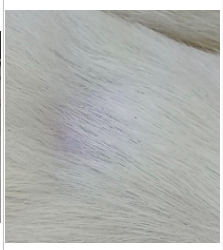
daily oral dose of 500mg/kg of the fruit extract of *Cyperus rotundus* (once a day for seven consecutive days) significantly lowered the blood glucose levels (8). The hydroethanolic extract of pulpy fruits and seeds of *Citrullus colocynthus* showed a marked decrease in serum blood glucose levels (9). In vivo berberine showed the same hypoglycemic effect as Metformin (10). Curcumin, found in Curcuma longa, a very common and effective medicine used extensively, has also shown antidiabetic properties in animal studies. (11)

Materials and methods

Animal care and selection

Albino rats, Wistar strain (150–200 g)) of either sex were obtained from the Lloyd Institute of Management and Technology, Greater Noida, Uttar Pradesh (India). Animals were housed under standard conditions of temperature, humidity, and light: dark cycle of 25 ± 2 °C, 65 ± 10%, 12 h respectively. Standard food pellets and drinking water were fed to animals ad libitum. The experimental protocol was approved by the IAEC of Lloyd Institute of Management and Technology, Greater Noida. All the animal activities were conducted according to the guidelines of "Guide for the care and use of laboratory animals" and CPCSEA (1206/ PO/Re/S/08/CPCSEA)

Figure 1: Wistar Albino rats	Figure 2: PTK extract	Figure 3: Fur alteration at 2000mg/kg Dose
		

Acute toxicity study (12)

The OECD Guidelines 423 (OECD Guidelines, 2001) were followed in the evaluation of the acute toxicity of the aqueous extract of *Phala Trikadi Kwath churna*, which was prepared by boiling the churna and water at a ratio of 1/16 and then reduced to 1/8. Then, 12 gms of haldi powder was added as per API (Part II, volume v). This PTK extract was further diluted with distilled water for the purpose of animal consumption. Three groups with three Wistar rats (Figure 1) were given oral dosages of PTK extract at 50, 300, and 2000 mg/kg body weight. Following dosing, each animal was checked separately for symptoms of toxicity (alterations in skin, fur, breathing, and motor activity) once in the first 30 minutes, once every 24 hours, and eventually, for the next 14 days, every day. The study was performed as per OECD Guidelines 423 to ensure that a higher dose of PTK extract might be significantly safe and free from symptoms of toxicity after observation of all parameters, such as alterations in skin, fur, breathing, and motor activity. No signs of toxicity in doses 50and 300/kg BW only some alterations in skin and fur in the rats with high dose of 2000/kg BW. Hence, a 300/kg BW dose was selected. (Figure 3 and Table 2) This study was further helpful to evaluate the dose-dependent pharmacological effect of the safe therapeutic dose of PTK extract, which was 1/5th and 1/10th of the selected dose of 300mg/kg, i.e., 30mg/kg (test drug I) and 60mg// kg (test drug II), which proved to be safest.

Experimental design

Before the experiment, the selected animals were weighed and grouped into five groups with six animals each as shown in table1. The complete duration of the experiment was 21 days. Daily intake of food and water was measured. Body weight was studied for all the groups in the 7-day gap and on the last day of sacrifice. (12)

Table 1: Shows five groups with six animals each to study the toxicity of the PTK extract

Groups		Route & Dose of Drug
Group I	Normal Control	Orally with distilled water (1 ml/kg BW)
Group II	Diabetic Control	50mg/kg (i.p)
Group III	Standard	Orally with Glibenclamide (5 mg/kg)
Group IV	Test drug	Orally with Test I (30 mg/kg)
Group V	Test drug	Orally with Test II (60 mg/kg)

Experimental induction of diabetes in rats

An adult albino rat (Wistar strain) weighing 150–200 g was selected for the study. Blood samples were collected for the determination of baseline glucose levels. DM was induced in overnight fasted animals. Rats were initially injected with a single dose of intraperitoneal with 50 mg/kg of freshly prepared in 0.01 M citrate buffer, pH 4.5. Animals in the control group received an equivalent volume of the citrate buffer and served as non-diabetic controls. After the elapse of 1 week, animals in groups 2–5 were declared diabetic with hyperglycemia (blood glucose level >200–300 mg/dl). The blood glucose level of selected diabetic animals was determined for further experimental procedures.

Body Weight Changes

Body weight was measured at STZ-dosing, and after 21 days of STZ-dosing, the body weight of experimental animals was measured once a week without sacrifice. Before blood collection, at dosing and sacrifice day, experimental animals were fasted overnight (water was not restricted) to reduce the erratum of feeding.

Oral glucose tolerance test (OGTT) (13) (14)

The oral glucose tolerance test was performed in overnight (18-h) fasted normal rats. Rats divided into five groups (n = 6) were administered either drinking water or PTK extract, 30 and 60 mg/ kg, respectively. Glucose (2 g/kg) was fed 30 min after the administration of the extract. Glibenclamide (5 mg/kg) was used as the standard drug. Blood was withdrawn from the retroorbital sinus under ether inhalation anaesthesia at 0, 30, 60, and 120 min of glucose administration and glucose levels were estimated using glucose oxidase-peroxidase reactive strips and a glucometer (Accu-Chek, Roche Diagnostics, USA).

Estimation of serum HDL, LDL and Cholesterol

The activities of pathophysiological enzymes such as serum triglyceride (TG), Total Cholesterol (TC), LDL-cholesterol (LDL-c) and HDL-cholesterol (HDL-c) were estimated with Erba kits as per manufacturer instructions.

Histopathology of rat pancreas

Rat pancreas were removed immediately from the animals after sacrificing and rinsed with ice cold saline. The tissues were fixed

with 10% formaldehyde, dehydrated in a graded series of ethanol and embedded in paraffin wax before sectioning. The sections were stained in hematoxylin and eosin (H & E).

Results and Discussions

Acute toxicity study

The acute toxicity examination was conducted after a single oral administration of 50, 300, and 2000 mg/kg. PTK extract in all doses did not result in the death of the treated animals during the 21-day observation period (Table 1). It was observed that a higher dose of 2000 mg/kg body weight had some alteration in skin fur but did not show any mortality in the animals used in the experiment. The dose of 300 mg/kg body weight is safe and did not result in the death of the treated animals or alter their morphological or physical characteristics during the 21 -day observation period. Hence, 1/5th and 1/10th of the selected dose, i.e. 300mg/kg, were selected as the doses to evaluate the therapeutic effects of PTK extract by dose dependant manner.

Table 2: Acute toxicity investigation of PTK extract in mice

Groups	Dose (mg/kg B.W)	No of mice with signs of toxicity/ Normal behaviour ST/NB	No of mortality/ Survival D/S
1	50	0/3	0/3
2	300	0/3	0/3
3	2000	2/3	0/3

B.W = Body weight, ST = Sign of toxicity, NB = Normal behaviour, D = Dead, S = Survive

Table 3: Body weight changes comparing the control group with the diabetic group

Group	Body weight at 0 days (gm)	Body weight at 7 days (gm)	Body weight at 14 days (gm)	Body weight at 21 days (gm)
Normal Control	190.23 ± 5.19	187.34 ± 8.97	185.43 ± 7.87	185.67 ± 7.54
Diabetic Control	201.15 ± 11.41#	140.45 ± 19.42#	135.97 ± 16.67#	131.33 ± 12.43#
Standard	198.53 ± 13.43	182.21 ± 14.24[a]	178.76 ± 17.31 [a]	177.28 ± 13.15 [a]
Test drug I	198.75 ± 8.52	172.88 ± 6.12 [a]	176.73 ± 9.83 [a]	174.42 ± 6.71 [a]
Test drug II	197.53 ± 9.71	173.47 ± 9.23 [a]	184.87± 4.78 [a]	185.67 ± 9.49 [a]

Results are displayed as mean ± SD (n = 6 rats/group). The statistical significance of differences was determined by using one-way ANOVA followed by Dunnett's test.

[#]p<0.01 versus the control group, [a]p<0.01 versus the diabetic group

This table 3 details the average body weight, in grams, of Wistar rats across five distinct experimental groups over 21 days. The figures are expressed as Mean ± Standard Deviation.

Evaluating the Treatment Effects (Standard, Test drug I, and Test drug II):

- All treatment groups (Standard, Test drug I, and Test drug II) began with weights comparable to both the Normal and Diabetic Control groups, around 198 gm.
- The Standard Treatment group experienced significantly less weight loss than the Diabetic Control group, with their weight declining from 198.53 gm to 177.28 gm. The 'a symbol denotes that this difference is statistically significant, indicating that the standard drug effectively mitigated weight loss associated with diabetes.
- Test drug I also demonstrated a notable reduction in weight loss compared to the Diabetic Control, concluding with a final weight of 174.42 gm. The 'a symbol suggests it was statistically superior to the Diabetic Control, underscoring its effectiveness as an anti-diabetic agent regarding body weight.
- Test drug II appears to excel in maintaining or restoring body weight. It reached a final weight of 185.67 gm, nearly matching that of the Normal Control group. The 'a symbol confirms this effect is statistically significant compared to the Diabetic Control, indicating that Test drug II is highly effective in normalising body weight in diabetic animals - potentially even more so than the Standard treatment.

Table 4: Results of fasting blood glucose test in wistar rats

Group	Blood Glucose at 0 days (mg/dl)	Blood Glucose at 7 days (mg/dl)	Blood Glucose at 14 days (mg/dl)	Blood Glucose at 21 days (mg/dl)
Normal Control	85.17 ± 6.10	85.17 ± 7.98	85.17 ± 8.54	85.17 ± 7.63
Diabetic Control	87.56 ± 18.43	275.87 ± 29.31#	289.62 ± 21.53#	296.56 ± 28.46#
Standard	86.49 ± 12.34	200.19 ± 11.12 [a]	147.76 ± 15.23 [a]	106.28 ± 13.65 [a]
Test drug I	87.75 ± 7.44	243.54 ± 5.87 [a]	195.16 ± 4.67 [a]	149.65 ± 9.76 [a]
Test drug II	86.91 ± 8.18	204.23 ± 6.52 [a]	154.63 ± 5.98 [a]	104.21 ± 8.43 [a]

Results are displayed as mean ± SD (n = 6 rats/group). The statistical significance of differences was determined by using one way ANOVA followed by Dunnett's test.

[#]p<0.01 versus the control group, [a]p<0.01 versus the diabetic group

Discussion: There is the Long-term Blood Glucose Monitoring, which spans 21 days. This information, which we have previously discussed, assesses the ability of these drugs to maintain stable blood sugar levels over an extended period. Here's a summary of the findings for day 21: Normal Control: 85.17 mg/dL - This indicates a healthy and stable blood sugar level.

- Diabetic Control: 296.56 mg/dL - This signifies severe hyperglycemia in the absence of treatment.
- Standard: 106.28 mg/dL - This demonstrates excellent control, nearing normal levels.
- Test drug I: 149.65 mg/dL - This shows a moderate decrease, but remains elevated.

- Test drug II: 104.21 mg/dL - This drug is notable for its significant reduction, almost reaching normal levels, and even surpassing the Standard. In conclusion, Test drug II is very effective in maintaining long-term blood glucose levels, performing comparably to or slightly better than the Standard drug. Test drug I is also beneficial, but does not match the others.

Table 5: Oral Glucose Tolerance Test of all five groups

Group	0 min (mg/dl)	30 min (mg/dl)	60 min (mg/dl)	120 min (mg/dl)
Normal Control	85.17 ± 6.10	85.98 ± 8.06	85.46 ± 7.37	84.76 ± 6.24
Diabetic Control	87.56 ± 18.43	144.75 ± 11.42#	139.76 ± 15.67#	134.53 ± 11.43#
Standard	86.49 ± 10.97	122.21 ± 9.04[a]	116.76 ± 8.32 [a]	104.28 ± 6.23 [a]
Test drug I	87.75 ± 7.44	128.43 ± 8.22 [a]	125.61 ± 6.56 [a]	123.53 ± 7.72 [a]
Test drug II	86.91 ± 8.18	121.43 ± 7.41 [a]	118.76± 10.91 [a]	109.41 ± 7.44 [a]

Results are displayed as mean ± SD (n = 6 rats/group). The statistical significance of differences was determined by using one-way ANOVA followed by Dunnett's test.

[#]p<0.01 versus the control group, [a]p<0.01 versus the diabetic group

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In this test, animals receive glucose after a fasting period, and blood sugar levels are recorded at 0, 30, 60, and 120 minutes. Here are the results:

- Normal Control: Maximum glucose level at 30 minutes was 85.98 mg/dL and 84.76 mg/dL at 120 minutes - minimal variation, indicating that a healthy body manages glucose effectively. Diabetic Control: Maximum glucose reached 144.75 mg/dL and remained elevated at 134.53 mg/dL, suggesting difficulty in glucose clearance.
- Standard: Maximum glucose was 122.21 mg/dL, dropping to 104.28 mg/dL by 120 minutes - excellent clearance, nearly returning to normal.
- Test drug I: Maximum glucose was 128.43 mg/dL, with a final reading of 123.53 mg/dL - moderate clearance, but still relatively high.
- Test drug II: Maximum glucose peaked at 121.43 mg/dL and concluded at 109.41 mg/dL - also demonstrating excellent clearance, approaching normal levels.

Table 6: Estimation of serum HDL, LDL and Cholesterol

Group	HDL (mg/dl)	LDL (mg/dl)	Cholesterol (mg/dl)
Normal Control	23.61 ± 2.92	45.76 ± 8.06	88.61 ± 3.44
Diabetic Control	20.87 ± 2.42	84.98 ± 8.12#	127.65 ± 6.92#
Standard	24.49 ± 1.98	47.34 ± 4.04[a]	90.66 ± 4.67[a]
Test drug I	25.75 ± 2.43	51.98 ± 4.22 [a]	97.11 ± 6.86 [a]
Test drug II	23.91 ± 2.18	44.32 ± 3.41 [a]	91.26± 8.87 [a]

Results are displayed as mean ± SD (n = 6 rats/group). The statistical significance of differences was determined by using one-way ANOVA followed by Dunnett's test.

[#]p<0.01 versus the control group, [a]p<0.01 versus the diabetic group

Discussion: Here are some notable insights regarding lipid levels:

- In the Normal Control group, HDL (often referred to as good cholesterol) measured 23.61 mg/dL, LDL (known as bad cholesterol) was at 45.76 mg/dL, with a total cholesterol level of 88.61 mg/dL.
- The Diabetic Control group presented alarming findings, with HDL decreasing to 20.87 mg/dL and LDL rising sharply to 84.98 mg/dL, resulting in a total cholesterol of 127.65 mg/dL. The Standard treatment yielded HDL at 24.49 mg/dL and LDL at 47.34 mg/dL, contributing to a total cholesterol of 90.66 mg/dL.
- Test drug I resulted in the highest HDL level at 25.75 mg/dL, while LDL was recorded at 51.98 mg/dL and total cholesterol at 97.11 mg/dL.
- Test drug II recorded HDL at 23.91 mg/dL, with LDL at 44.32 mg/dL (the best outcome), and total cholesterol at 91.26 mg/dL. The findings indicate that the Diabetic Control group exhibited classic dyslipidemia characteristics, showing elevated LDL and total cholesterol alongside reduced HDL compared to the Normal Control group. All treatments demonstrated significant reductions in LDL and total cholesterol levels.

Figure- 3 (a): Control 10X	Figure-3 (b): Control 40X	Figure-4 (a): Diabetic control 10X	Figuire-4 (b): Diabetic Control 40X

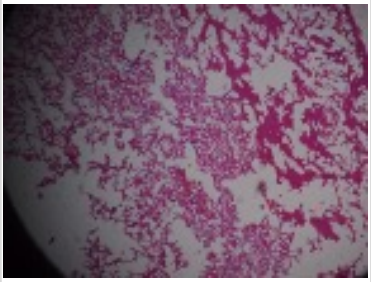
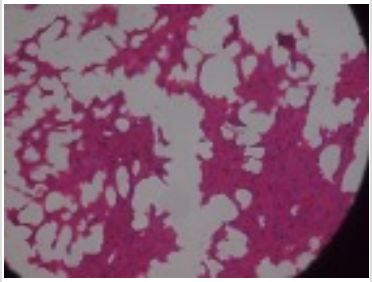
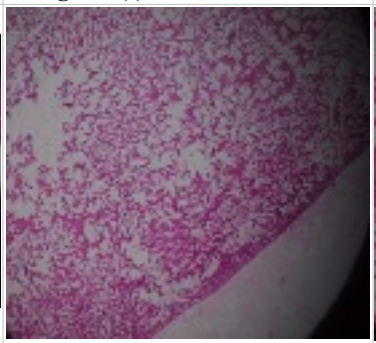
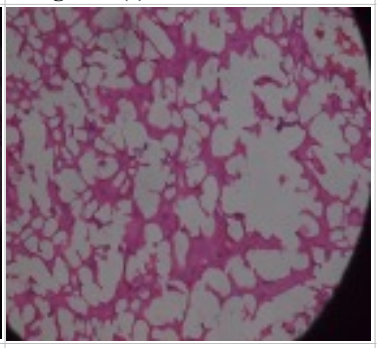
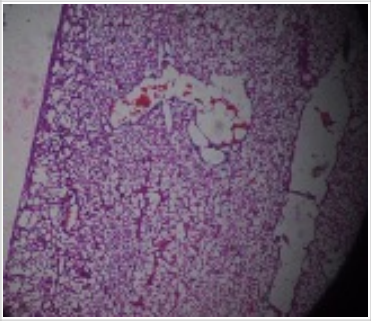
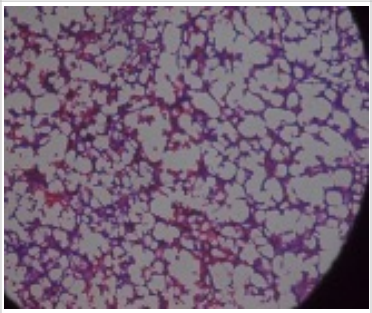
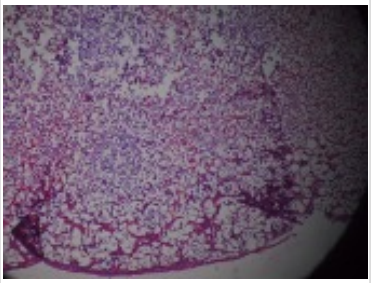
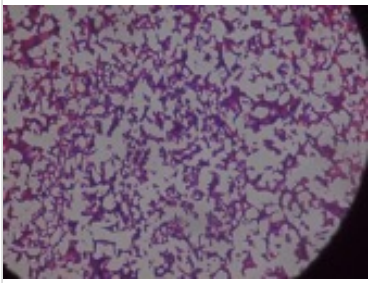

			

Figure-5 (a): Standard 10X	**Figure-5 (b): Standard 40X**	**Figure-6 (a): Test-1 10X**	**Figure-6 (b): Test-1 40X**
			

In the **normal group** as shown in figures 3 (a) and (b) the islets of Langerhans were unevenly distributed in pancreatic tissue, although they were often extremely abundant and of varied sizes in the same lobule of the pancreas. The strongly stained acinar cells were organised in lobules with prominent nuclei. The islets cells were found embedded within acinar cells and surrounded by a fine capsule.

Pancreatic islets of **diabetic control** rats, as in figures 4 (b)and (c) showed a significant decrease in the number and size of acinar cells around the islets, which appeared to be in normal proportion but did not look elegant. This revealed that the islets had been damaged and decreased in size, with lymphocyte infiltration. The degeneration of the beta cells was caused by the streptozotocin used to trigger diabetes.

The islets cells in the **glibenclamide-treated group** appeared to be normal in location, but in smaller numbers than the normal group, as shown in figures 5 (e) and (f). After 21 days of glibenclamide treatment, the cell size was expected to return to normal. The islet cells were organized compactly, with little intercellular space.

The number of islet cells was reduced in the treated group at **30 mg/kg** body weight as shown in Figures 6 (a) and (b). The cell size was reduced due to architectural disarray and hydrolysis as compared to the diabetes control group.

The islet cells of the **60 mg/kg** body weight extract-treated group were found to be in normal position, figure 7 (a)and (b). The islets were present with a large proportion of islet cells, though with a smaller volume compared to the normal group.

Conclusions

In the acute toxicity study, at any dose, it did not result in the death of any animal. While higher doses showed some alterations in skin and fur. Hence, 1/5th and 1/10th of the selected dose, i.e.,300mg/kg, which is 30mg/kg BW (Test drug I) and 60mg/kg BW (Test drug II)were selected to evaluate their therapeutic effect.

When assessing the effectiveness of both test drugs, we conclude: Test Drug I is effective but demonstrates moderate strength. It aids in reversing diabetic symptoms but does not perform as well as the Standard treatment or Test drug II. It successfully prevented severe weight loss and showed a positive increase in HDL cholesterol. Test Drug II is highly effective and comprehensive. It addresses all significant diabetes symptoms and surpasses the Standard treatment in multiple aspects. It completely restored normal body weight and achieved excellent management of blood glucose and lipid levels. In conclusion, Test drug II emerges as the leading compound among those evaluated. It not only reduces blood sugar levels but also enhances insulin utilisation, counteracts weight loss associated with diabetes, and significantly mitigates cardiovascular risk factors by normalising lipid levels. Significant control of diabetes and other parameters was observed with the test drug. The Islet cells of the 60 mg/kg body weight extract-treated group were found to be in normal position. Based on the data, there were no signs of acute toxicity and good control of diabetes in a fixed dose. (16)

Funding Sources: No other funding sources

Acknowledgement: The author has been thankful to Dr Lalit Tyagi, Lloyd Institute of Pharmacy, who permitted to conduct the experiments in their animal house

Declaration of ethical approval: The experimental protocol was approved by the IAEC of Lloyd Institute of Management and Technology, Greater Noida. All the animal activities were conducted according to the guidelines of "Guide for the care and use of laboratory animal" and CPCSEA (1206/PO/Re/S/08/ CPCSEA)

Prior informed consent: consent form has been attached with the submission

Abbreviations
OGTT
PTK

Conflict of Interest: none

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