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EVALUATION OF ANTIDIABETIC PROPERTY OF KARCHIKAI (MOMORDICA CYMBALARIA) AN UNDERUTILIZED MEDICINAL CROP DISTRIBUTED IN NORTH KARNATAKA

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ABSTRACT
Background: Momordica cymbalaria, commonly known as Karchikai, is an underutilized wild vegetable with significant nutritional and pharmaceutical value, particularly in managing diabetes mellitus.
Objective: This study aims to elucidate the antidiabetic effects of Karchikai fruit powder using a streptozotocin-induced diabetic Wistar rat model.
Materials and methods: The investigation focused on the efficacy of Karchikai fruit powder sourced from distinct regions in Karnataka, India, i.e. Bagalkot, Vijayapur, Hungund and Raichur. After oven-drying and grinding, the resultant powder was orally administered at a dose of 400 mg/kg body weight to diabetic male Wistar rats per day (200-300 g) for 21 days. Body weight and some biochemical parameters like serum glucose, triglycerides and cholesterol were measured on day 0 (before) and day 7, 14 and 21 after the induction of diabetes.
Results: Treatment with Karchikai fruit powder caused a significant reduction in serum glucose and reduced weight loss in diabetic rats, with maximum antidiabetic activity shown by the Bagalkot sample. Additionally, Karchikai treatment reduced elevated levels of serum cholesterol and triglycerides in the diabetic group, thus strengthening its efficacy as an antidiabetic drug. Histopathological analysis revealed a substantial increase in beta cells of the pancreas among rats treated with Karchikai, indicating its possible role in insulin regeneration.
Conclusion: The fruit powder extracted from Karchikai has been seen to possess prominent antidiabetic properties and similarities to insulin, known as "m cym," to determine why it can be an efficient source for controlling diabetes. Seed cultivation needs to improve as a solution for low germination rates.

KEYWORDS: Antidiabetic properties, Karchikai fruit powder, Streptozotocin induced diabetes, Hypoglycemic effects, Wister albino rats, triglycerides.

1. INTRODUCTION:

Momordica belongs to a genus with 59 species [1], mainly found in the paleotropic regions and dominated within the warm regions of tropical Africa and South Asia [2, 3]. Also, there are eight species described for India, which include M. cochichinensis, M. dioca, M. cymbalaria, M. denudata and M. macrophylla. The genus shows monoecious as well as dioecious conditions. The list of monoecious species includes M. charantia (L.) var. muricata (Wild.) Chakrav. and M. charantia (L.) var. charantia (L.), M. balsamina (L.) and M. cymbalaria. However, M. dioca (Roxb), M. sahyadrica, M. cochichinensis and M subangulata. Out of these, Momordica cymbalaria, also known as Wild Gourd, is an evergreen twining vine belonging to Cucurbitaceae family with diploid numbers 2n = 18. It is thought to be native to the tropical area of India and Southeast Asia, but its original habitat still hasn't been identified [4]. It prefers environments within India's Karnataka, Madhya Pradesh, Maharashtra andhra Pradesh and Tamil Nadu [5]. It goes by several regional names like 'Kakrol' = Princep's yam = Common gourd '(India), 'Athalakkai' = Little gourd '(Srilanka/Tamil), 'Kasarakayee' = Little Wild Gourd '(Andhra Pradesh) and 'Karchikai' = Little Wild Gourd '(Kannada) [6]. Historically, Momordica cymbalaria has been valued for its nutritional benefits and has been incorporated into various traditional forms of Asian medicine, emphasizing its importance as a wild vegetable with underappreciated medicinal uses [7]. The different types of bitter gourd share a close resemblance with the unripe fruits of Momordica cymbalaria, which serves as a vegetable in the northern region of Karnataka. The plants of this species display a unique stem structure which grows vertically while developing multiple branches that show visible striped patterns. The leaves show an orbicular-reniform shape which combines deeply cordate base and five to seven obtuse lobes. The male flowers develop on 0.05 0.30 cm long peduncles which contain 2 to 5 flowers arranged in racemes that display a pale-yellow corolla and two stamens while the female flowers develop individually on 28 mm peduncles. The fruits have a pyriform shape which extends to 25 mm length and features eight sharp ridges that lead to a curved fleshy dark-green peduncle which measures 24 mm by 15 mm and tapers at its top. The seeds have a smooth shiny appearance and take an ovoid shape which measures 4.6 mm in length. The roots of the plant appear with light brownish-yellow color and reach a diameter between 4 to 8 centimeters while developing aromatic tuberous structures that break into fibrous pieces when fractured due to their intense bitterness which

enables the plant to grow as a perennial. Phytochemical analyses discover medicinally important compounds which include tannins, alkaloids, amino acids, vitamin C, carbohydrates, β-carotenes, flavonoids, triterpenes, sterols, glycosides, citric acid, maleic acid and fixed oils that have high palmitic and oleic and stearic and α-eleostearic and γ-linolenic acid content [6, 8, 10]. The plant M. cymbalaria (Karchikai) grows in fields while it extends its growth along fences and bunds and dies after the seasonal period ends [7, 9]. The green synthesis of silver nanoparticles occurs through the action of bioactive principles [11]. Research demonstrates that there exists unexploited potential to combat cancer and diabetes and diseases that stem from oxidative stress. The process of seed-based propagation suffers from two main challenges which include both the low germination rates and the limited number of seeds that can grow from each fruit [12-14]. M. cymbalaria provides multiple health benefits which include anti-allergic properties, antidiabetic effects, hypolipidemic activity, anti-diarrheal protection, anti-ulcer treatment, cardioprotective abilities, antimicrobial action, nephroprotective support and anticancer power [15-19]. Folk knowledge highlights Momordica species for preventing Type I/II diabetes, improving glucose tolerance and lowering postprandial blood sugar in rats. Diabetes management through diverse herbs exists worldwide according to sources [20, 21, 22]. The fruit of the plant produces M. cymbalaria which functions as an insulin-mimetic protein that assists glucose uptake and shows potential as an antidiabetic treatment yet scientists must conduct more research on phytochemical mechanisms [22, 23, 24]. The researchers conducted their study by gathering M. cymbalaria samples from three different locations throughout North Karnataka for testing its antidiabetic properties through in-vivo experiments.

2. MATERIALS AND METHODS

Karchikai fruits utilized in this study were sourced from four distinct locations within Karnataka, each identified accordingly. After harvest, the fruits underwent a drying process in a hot air oven for duration of 4 to 6 days. Subsequently, the dried samples were ground using a mixer for use in feeding the rats (refer to Figure 1).

2.1 Experimental animals

Male adult Wistar albino rats weighing 200-300 g were obtained from the Hanagal Shri Kumareshwar College of Pharmacy and Research Centre's Central Animal House at Bagalkot. The rats were kept under optimal conditions with a 12-hour light and dark cycle, clean drinking water and a standard laboratory pellet diet. All the experimental protocols were approved by the institutional ethical committee.

2.2 Treatment Protocols

The experimental design included four treatment groups, each representing one of the four collection sites: Bagalkot, Vijayapur, Hungund and Raichur, with three replicates for each group. The experimental design used in this study was Dunnett's test, which included a sample of 12 Wistar albino rats, weighing between 200 to 300 g (Table 1).

2.3 Induction of Diabetes in Rats

The induction of diabetes in rats was done through the intraperitoneal injection of 30 mg/kg body weight of streptozotocin, which was diluted in sodium citrate buffer. This resulted in the pancreatic swelling and subsequent degeneration of the beta cells in the islets of Langerhans, thus inducing experimental diabetes mellitus in 2 to 4 days. The confirmation of diabetes was done by measuring the fasting blood glucose levels, where rats with blood glucose levels above 200 mg/dL were considered diabetic and included in the study (Figure 2).

2.4 Oral Administration of Karchikai Powders

After an overnight fast, a dose of 400 mg/kg body weight of karchikai powder was given orally to each rat as per the group protocols. The powder was mixed with Tween 80 and an appropriate amount of distilled water before oral administration.

2.5 Blood sampling and serum separation

Blood samples were obtained from the retro-orbital plexus of diethyl ether-anesthetized rats on the 0^{th}, 3^{rd}, 7^{th}, 14^{th} and 21^{st} days. The blood samples were collected using sterile labeled Eppendorf tubes and allowed to clot for 10 minutes. After clotting, the samples were centrifuged at 4000 rpm for 15 minutes at room temperature and slanted positions to ensure separation of the serum. The clear and non-hemolyzed serum was then transferred to sterile labeled Eppendorf tubes for storage. The remaining serum was refrigerated at -20°C for subsequent analysis. The separated serum was immediately used for biochemical analysis of the important biological markers such as glucose, triglycerides and total cholesterol (Figure 3).

3. Observations recorded

3.1 Body weight

All individual animal body weights were recorded using an electronic scale

3.2 Serum glucose (mg/dL)

The Trinder method was employed to determine the serum glucose levels in blood samples, as described in reference [25]. The test tubes were labeled and underwent oxidation with glucose oxidase, yielding gluconic acid and hydrogen peroxide (H_2O). An adequate number of test tubes were prepared to hold standards, controls and actual samples for the experiment. Each test tube contained 10 ml of sample and 1000 ml of glucose reagent. The mixture in the test tubes was well mixed and then allowed to stand at room temperature for 30 minutes (ranging from 15 to 30°C). After the incubation period, a

UV spectrophotometer was employed to determine the absorbance readings at 505 nm for both the sample and the blank. The serum glucose level was determined using the following formula:

$$\text{Glucose}\left(\frac{\text{mg}}{\text{dL}}\right) = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{Concentration of standard}$$

3.3 Serum triglycerides (mg/dL)

The serum triglyceride concentrations were determined using the Trinder method [25]. For each set of standards, controls and test samples, an adequate number of labeled test tubes were prepared. In each test tube, 10 ml of the sample was mixed with 1000 ml of the triglyceride reagent. The mixture in the test tubes was well mixed and then incubated for 30 minutes at room temperature (15 to 30°C) or for 10 minutes at 37°C. The absorbance readings at 546 nm for both the sample and the blank, as described in the procedure in [26, 27], were determined using a UV spectrophotometer. The serum triglyceride level was calculated using the following formula:

$$\mathbf{Triglycerides}\left(\frac{\mathbf{mg}}{\mathbf{dL}}\right) = \frac{\mathbf{Absorbance\ of\ sample}}{\mathbf{Absorbance\ of\ standard}} \times \mathbf{Concentration\ of\ standard}$$

3.4 Serum cholesterol (mg/dL)

The measurement of the serum cholesterol level was done using the Tinder method [25]. An adequate number of labeled tubes were provided for each set of standard samples, control samples and test sample groups. In each test tube, 10 ml of the sample was mixed with 1000 ml of cholesterol reagent. The mixture in the test tubes was then thoroughly mixed and incubated for either 30 minutes or 10 minutes at room temperature, which ranged from 15 to 37°C. After incubation, the values of absorbance at 505 nm for both the blank and the sample were measured using a UV spectrophotometer, as explained in [27]. The measurement of serum total cholesterol was determined using the following formula:

$$\text{Cholesterol}\left(\frac{\text{mg}}{\text{dL}}\right) = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{Concentration of standard}$$

Verhoeff‟s van Gieson (VvG) and Periodic Acid–Schiff (PAS). These stains were used as per standard histological procedures.

3.5 Statistical analysis

Graph Pad Prism software was used to evaluate the experiment data using one- way analysis of variance (ANOVA), which was followed by Dunnett's test, which tested all treated groups against controls. The values shown are the mean SEM (n=6). A significant difference between experimental and control rats was defined as P0.

4 RESULTS

4.1 Anti-diabetic property of karchikai fruits

To assess the antidiabetic potency of M. cymbalaria (Karchikai) fruit powder, an in vivo rat experiment was performed. Four sets of rats were given oral doses of powder derived from fruits harvested from four different locations in North Karnataka. The data was collected for the normal, diabetic control and treated sets, which showed a dose-dependent decrease in blood glucose levels.

4.1.1 Body weight estimation (g)

The initial body weight (g) and body weight after treatment are shown in Table 2. There were no statistically significant differences in the body weights of all groups before the injection of streptozotocin. After streptozotocin injection, a reduction in body weight was recorded on day 3 in all groups (Normal: 223.8±4.3, Control: 221.54±6.7, G1: 213.8±6.3, G2: 215.8±3.3, G3:256.5±8.34, G4: 243.5±9.16), except in the normal group. Later, the body weight increased in all groups, except in the normal group. The oral treatment of methanol extract of karchikai fruit powder (400 mg/kg body weight) in diabetic rats effectively blocked the reduction in body weight. Based on these findings, it can be concluded that the body weight of rats reduced after the induction of diabetes, but an increase in body weight was recorded after the treatment with karchikai fruit powder.

4.1.2 Serum glucose estimation (mg/dL)

The effect of karchikai fruit powder on serum glucose concentrations (mg/dL) in normal and diabetes-induced rat models is presented in Table 3 and Figure 1. On day 7, the serum glucose concentrations were recorded as follows: Normal: 135.7±3.786, Control: 552.7±16.17, G1: 346.3±19.14, G2: 365.0±20.00, G3: 346.0±41.87, G4: 361.7±15.28. It is pertinent to mention here that the oral treatment of karchikai fruit powder caused a substantial decrease in serum triglyceride concentrations in all experimental groups, except the normal control group.

4.1.3 Serum triglycerides estimation (mg/dL)

The effect of karchikai fruit powder on serum triglyceride level of normal and diabetes induced rats are given in the Table 4 and Figure 2. On 7th day (Normal: 151.11±9.872, Control: 440.9±10.31, G1: 265.5±47.53, G2: 315.0±51.27, G3: 338.3±51.18, G4: 332.2±35.41), oral administration of karchikai fruit powder to different groups showed significant reduction in serum triglyceride level in all the groups except the normal group. Similarly it showed significant

reduction in serum triglyceride level on 14th (Normal- 155.8±11.17, Control: 372.5±5.828, G1: 142.0±5.123, G2: 121.0±21.04, G3: 155.9±37.17, G4: 169.5±25.21) and 21st (Normal: 132.2±6.025, Control: 322.5±12.63, G1: 129.3±6.083, G2:133.0±4.230, G3: 127.6±0.577, G4: 126.3±2.00) day except the normal group.

4.1.4 Serum cholesterol estimation (mg/dL)

The effect of karchikai fruit powder on serum cholesterol level of normal and diabetes induced rats are given in the Table 5 and Figure 3. On 7th day (Normal: 129.0±5.508, Control: 386.0±10.50, G1: 284.6±5.686, G2: 281.6±15.28, G3: 275.3±1.00,G4: 268.6±20.60), oral administration of karchikai fruit powder to different groups showed significant reduction in serum cholesterol level in all the groups. Similarly there was significant reduction in serum cholesterol level on 14th (Normal: 133.3±4.325, Control: 380.0±5.686, G1: 177.3±61.70, G2: 245.6±14.92, G3: 242.2±4.985, G4: 238.1±5.283) and 21st(Normal: 132.3±6.058, Control: 334.3±27.57, G1: 148.9±10.09, G2: 138.5±3.233,G3:146.1±12.97, G4: 136.0±1.293) day except the normal group.

4.2 Histopathology

Compared to the normal, control and treated appearance of pancreas shown in Figure 4, karchikai powder treatment had significant effect on treated pancreas. Pancreatic islet cells of normal whister male rats seemed to have a normal architecture (Fig 4A). Intra-peritoneal injection of streptozotocin, at a dose of 30 mg/kg of body weight resulted in morphological alterations of pancreatic islet cells and showed destructed β cells with decreased number and vacuolated cytoplasm in control pancreatic islet cells (Fig 4B). Treatments for 3 weeks of diabetic rats with karchikai powder stimulated recovery of the islet cells in all the groups 1 (Fig 4C), (Fig 4D), (Fig 4E) and (Fig 4F). The islets approximately regained their normal appearance with a marked increase of β cell number and fewer vacuolated cells when compared to the pancreas of untreated diabetic rat.

4.3 Discussion

With or without associated impairment of insulin action, it is primarily brought on by inadequate pancreatic insulin secretion. Around 143 million people worldwide have diabetes. By 2030, this number is anticipated to double, according to the WHO. According to [28], diabetes mellitus is a very serious public health issue that contributes to a wide range of complications as well as an increase in illness and death. Diabetes cannot be completely cured. Several methods, including a combination of anti-diabetic drugs, exercise, a healthy diet and herbal therapies, can be used to control it [29] [30]. Hence, in the present study, 4 groups representing source of diversity collected from different regions of Karnataka like Bagalkot, Vijayapur, Hungund and Raichur were examined for anti-diabetic property in a rat model. Rat's estimated body weights before and after treatment were showed in the Table 2.

Streptozotocin administration after the third day resulted in a decrease in body weight, with the exception of the normal group. At the conclusion of the trial, the diabetic rats in the control group displayed a further reduction in body weight (113.6±5.4g on day 21 against 221.54±6.7g on day 3). Weight loss is a primary indicator of diabetes, but its mechanism is unclear [31]. Streptozotocin induced diabetes is accompanied by a visible decrease of body weight, which is due to accelerated muscle atrophy and protein loss brought on by insulin deficiency [32, 33, 34]. Although diabetic rats consumed more food than normal rats did, a reduction in body weight was still feasible due to the catabolism of proteins and fats. Proteolysis reduced the total protein content in muscle tissue as a result of insulin inefficiency [35]. Weight loss was reported in streptozotocin induced diabetic rats [36]. Our results are supported with the previous literature [37] that significant reduction in bodyweight was noticed in diabetic rats. However, the diabetic rats treated with 400 mg/kg body weight of karchikai showed significant check on the loss of body weight. The accurate measurement of serum glucose in bodily fluids is crucial for the analysis and treatment of hypoglycemia associated with diabetes. Many medical issues, including adrenal dysfunction. High serum glucose levels can occur in diabetics, people receiving glucose- containment fluids intravenously at times of extreme stress and other cases. In our investigation, diabetic rats in the control group displayed a further rise in serum glucose levels at the end of the experiment (at 0th day: 566.0±15.10 mg/dL vs at 21st day: 593.3±21.20 mg/dL) (Table 3). The primary mechanisms producing hyperglycemia include excessive synthesis (excessive hepatic glycogenolysis gluconeogenesis), decreased tissue glucose uptake and insulin in sufficiency [34] [38]. As demonstrated in our experiment, repeated oral administration of karchikai powder decreased the higher serum glucose level brought on by the injection of streptozotocin. The maximum hypoglycaemia effect was noticed in Group 1 (Bagalkot) rats followed by Group 2 (Vijayapur), Group 3 (Hungund) and Group 4 (Raichur) respectively. This hypoglycemic effect of karchikai may be caused by the insulin-like action exhibited by extract and encapsulate on peripheral tissues, which either facilitates glucose uptake by restraining gluconeogenesis in the liver or may result from glucose assimilation in the skeletal muscles and adipocytes, through encouraging the regeneration of beta cells [39, 40]. Tables 4 and 5 illustrate the effects of karchikai extract and encapsulation on the blood total cholesterol and triglycerides of diabetic rats. Total cholesterol and triglycerides increased in streptozotocin-induced rats. Similar findings were made by [41 and 42] in hyper cholesterol rats caused by sterptozotocin. In addition to preventing the release of free fatty acids into the bloodstream, insulin is a powerful inhibitor of hormone-sensitive lipase in adipose tissue [43]. As a result of increased beta-oxidation of fatty acids and increased fatty acid concentration, diabetic patients have higher levels of acetyl CoA and cholesterol [44]. Following catabolization, acetyl CoA is produced from the enhanced free fatty acid plasma. Blood cholesterol levels can rise as a result of the acetyl CoA being directed toward cholesterol synthesis [45]. Surprisingly in our investigation, diabetic rats treated with karchikai powder which had high levels of total cholesterol and triglycerides returned to nearly normal levels. For 21 days, diabetic rats were given continuous treatment with karchikai powder and the results showed a strong hypoglycemic impact. Rats of group 1 fed with karchikai powder of bagalkot location reported highest hypoglycaemia effect, followed by groups 2, 3 and 4. Regarding the histopathology

studies, karchikai powder had no significant effect on insulin release from isolated beta cells of the pancreas or on intestinal glucose absorption, while it increased glucose uptake significantly by rat diaphragm only at high concentration. In addition, karchikai powder treatment was found to increase number of beta cells in the pancreas of diabetic rats. These findings are in agreement with earlier findings [44, 45, 46] in their study also confirmed that serum cholesterol and triglyceride levels rise in tandem with blood glucose levels in alloxan-induced diabetes mellitus. When karchikai powder was administered to diabetic rats, the levels of triglycerides and cholesterol were brought close to normal. Similar results were confirmed in our study.

5 CONCLUSIONS AND FUTURE LINE OF WORK

M. cymbalaria (Karchikai), an under-exploited wild cucurbit of North Karnataka, was tested for its antidiabetic properties in streptozotocin (STZ)-induced diabetic Wistar rats. Fruits of four different locations, Bagalkot, Vijayapur, Hungund and Raichur, were oven- dried, powdered and orally administered (400 mg/kg body weight) for 21 days to male rats (200-300g, n=12/group). STZ induction resulted in severe diabetic symptoms in which body weight drastically dropped (control: 221.5±6.7g on day 3 to 113.6±5.4g on day 21). Also, theserum glucose significantly increased (566.0±15.1 mg/dL on day 0 to 593.3±21.2 mg/dL on day 21), triglycerides reached a peak (440.9±10.3 mg/dL on day 7) and cholesterol reached a maximum (386.0±10.5 mg/dL on day 7). Karchikai powder caused a dramatic reversal of these symptoms. Bagalkot (G1) showed better efficacy: glucose reduced to 147.4±19.1 mg/dL, triglycerides to 129.4±6.1 mg/dL, cholesterol to 148.9±10.1 mg/dL on day 21, with body weight maintained at 216.3±7.1g. All groups arrested weight loss and normalized lipid levels. Histopathology showed STZ-damaged pancreatic β-cells (vacuolated and reduced) compared to the restored islet structure following treatment, indicating β-cell regeneration. The antidiabetic action may be due to the insulin-simulating protein of Karchikai, increased peripheral glucose uptake (skeletal muscle and adipocytes), inhibition of hepatic gluconeogenesis and phytochemicals (flavonoids, alkaloids and triterpenes). This confirms the traditional antidiabetic property of Karchikai, making Bagalkot varieties a potential natural remedy. Isolation of M. cymbalaria for clinical studies and improving propagation difficulties (low germination rate) would be areas of future work. The importance of Karnataka's unexplored crop diversity for managing diabetes is highlighted. We can conclude from this study that the fruits of M. cymbalaria improves hyperlipidaemia caused by diabetes and has positive effects on blood glucose levels. To clarify the mechanism of M. cymbalaria fruit's antidiabetic and hypolipidemic effects, more pharmacological and biochemical studies are being conducted.

Author Contributions: RC: conceptualization, methodology, supervision, original draft preparation; KM: conducted the experiment, investigation; PG: review and editing; YAS: manuscript editing; RG: formal analysis; VMC: pharmacology studies, supervision; NBR: germplasm collection; VH: preparation of product, Post-harvest studies; SD: review; JN: review.

Ethical approval and consent: It is permitted to carry out the experiments on animals as per the institute of animal ethics committee as per the provisions made by CPCSEA.

Declaration of generative AI in scientific writing: The corrective AI tools were used to support language refinement, grammar correction and the organization of ideas. All content was reviewed and approved by the authors, who take full responsibility for the integrity and accuracy of the final manuscript. The AI was not used to generate original scientific content, analyzed data, or interpret results.

Funding sources: Nil.

Conflict of interest: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement: The authors would like to thank University of Horticultural Sciences, Bagalkot and College of Pharmacy, BVVS, Bagalkot, for providing the facilities for conducting the experiments.

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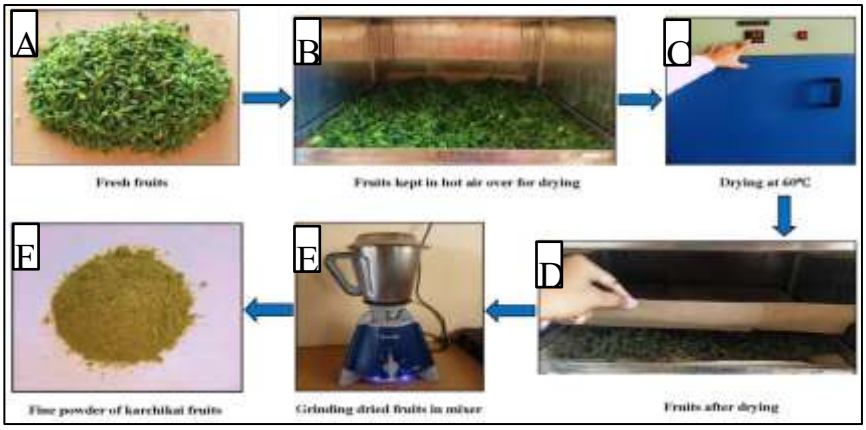

Figure 1. Flow chart of drying and powdering of Karchikai fruits 1A. Fresh fruits collected and cleaned, 1B. Fruits kept in hot air oven for drying, 1C. Set the drier at 60^{o}C, 1D. Oven dried fruits, 1E. Grinding the dried fruits in grinder, 1F. Fine powder of karchikai fruits

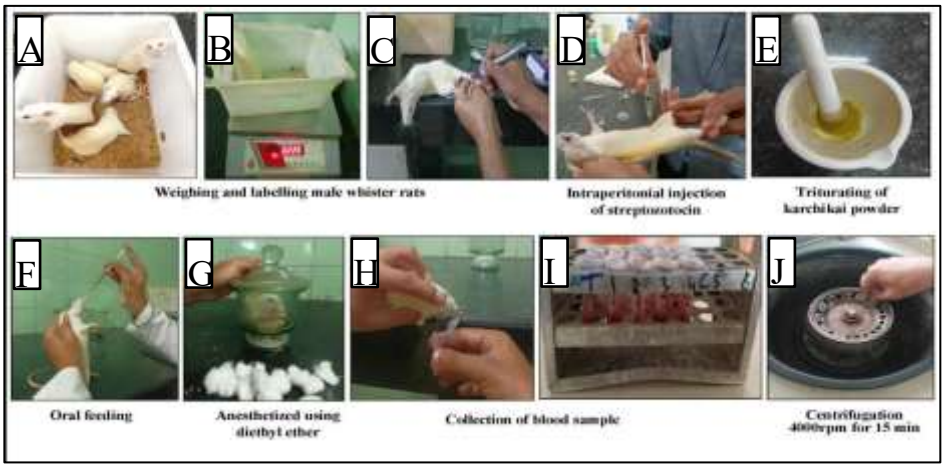

Figure 2. Pictures indicating activities carried out during rat studies: 2A. Male whister rats, 2B. Recording their weight, 2C. Labelling the rats, 2D. Injecting streptozotocin, 2E. Karchikai powder for feeding, 2F. Oral feeding to rats, 2G. Anesthetized rats usingdiethyl ether, 2H. Collection of rat blood samples, 2I. Storing the blood samples, 2J. Centrifugation.

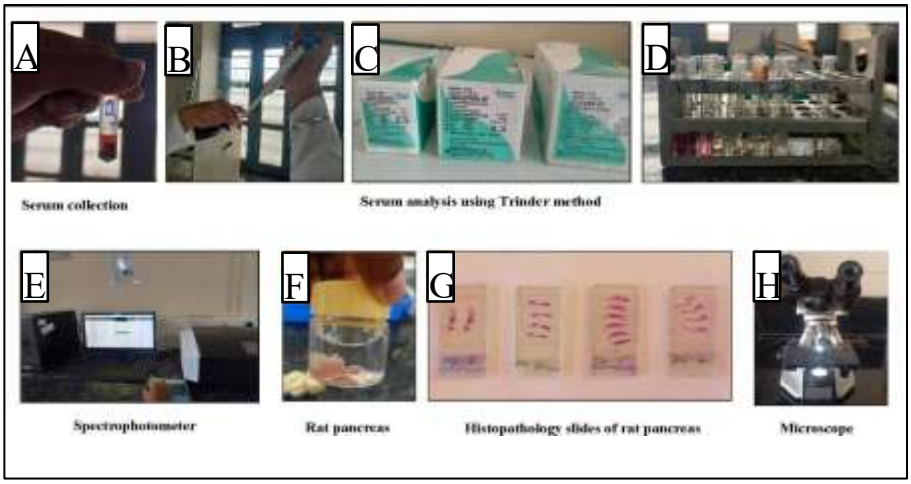

Figure 3. Various activities carried out during biochemical estimation: 3A. Serum collection, 3B. Pipette out required quantity, 3C. Serum analysis using Trinder method, 3D. Chemicals required for the study, 3E. Spectrophotometer, 3F. Rat pancreas, 3G. Histopathology study of rat pancreas, 3H. Microscope.

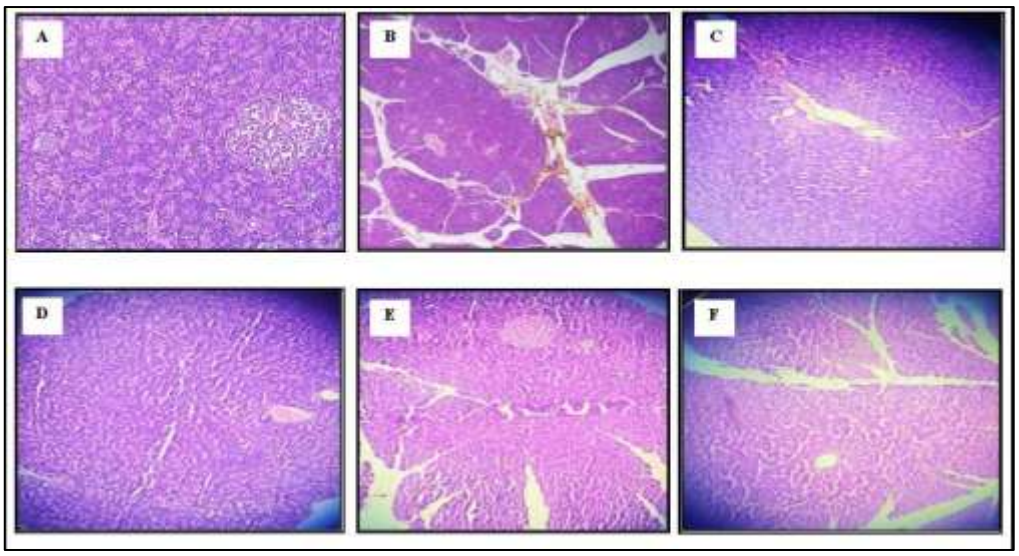

Figure 4. Histopathology results of rat pancreas 4A. Pancreas of normal male whister rats 4B. Pancreas of control group rat after 3 weeks experimental period 4C. Pancreas of group 1 diabetic rat 4D. Pancreas of group 2 diabetic rat 4E. Pancreas of group 3 diabetic rat 4F. Pancreas of group 4 diabetic rat

Table 1. The rats were divided into 4 groups (n=3) as follows

Treatment	Collections	Single-dose (mg/kg body weight)	Number of rats	Duration
Group 1(G_1)	Bagalkot	400mg/kg	3	3 weeks
Group 2(G_2)	Vijayapur	400mg/kg	3	3 weeks
Group 3(G_3)	Hungund	400mg/kg	3	3 weeks
Group 4(G_4)	Raichur	400mg/kg	3	3 weeks

Table 2. Effect of oral administration of karchikai powder on body weight (g) of diabetic rats

Groups (Landraces)	Days of estimation				
	0^{th} day	**3^{rd} day**	**7^{th} day**	**14^{th} day**	**21^{st} day**

Normal	218.3±6.8	223.8±4.3	227.8±7.5	230.2±7.88	232.5±7.6
Control	224±6.5	221.54±6.7	118.1±6.09	115.5±5.8	113.6±5.4
Group 1 (G_1)	209±3.4	213.8±6.3	215.8±3.3	216.2±4.7	216.3±7.1
Group 2 (G_2)	225.7±4.6	215.8±3.3	231.5±4.8	236.7±4.55	242.0±5.9
Group 3 (G_3)	239.3±8.02	256.5±8.34	243.5±9.16	260.5±9.47*	273.8±10.82**
Group 4 (G_4)	236.8±3.52	243.5±9.16	235.5±4.97	242±4.96	242.0±4.76

All values presented as a Mean ± SEm. The minimum value of p<0.05was considered as significant. p<0.05, **<0.01, *<0.001 as compared with control group. Group 1: Bagalkot collection Group 2: Vijayapur collection Group 3: Hungund collection Group 4: Raichur collection**

Table 3. Effect of oral administration of karchikai powder on serum glucose (mg/dL) ofdiabetic rats

Groups (Landraces)	**Days of estimation**			
	0^{th} day	**7^{th} day**	**14^{th} day**	**21^{st} day**
Normal	133.7±7.095***	135.7±3.786***	142.3±11.68**	145.7±6.028***
Control	566.0±15.10	552.7±16.17	572.0±9.165	593.3±21.20
Group 1(G_1)	517.7±49.20	346.3±19.14***	465.5±84.36*	147.4±19.14***
Group 2(G_2)	484.0±20.45**	365.0±20.00***	244.9±177.9	161.9±8.568***
Group 3(G_3)	456.5±25.87**	346.0±41.87***	276.6±135.4	165.7±20.47***
Group 4(G_4)	459.5±17.65**	361.7±15.28***	321.0±193.2	179.2±7.252***

All values presented as a Mean±SEm. The minimum value of p<0.05was considered as significant. *p<0.05, **<0.01, *<0.001 as compared with control group Group 1: Bagalkot collection Group 2: Vijayapur collection Group 3: Hungund collection Group 4: Raichur collection**

Table 4. Effect of oral administration of karchikai powder on serum triglycerides (mg/dL) of diabetic rats

Groups (Landraces)	**Days of estimation**			
	0^{th} day	**7^{th} day**	**14^{th} day**	**21^{st} day**
Normal	137.9±3.843***	151.11±9.872***	155.8±11.17***	132.2±6.025***
Control	487.0±9.126	440.9±10.31	372.5±5.828	322.5±12.63
Group 1(G_1)	390.3±5.160***	265.5±47.53***	142.0±5.123***	129.3±6.083***
Group 2(G_2)	378.4±6.011***	315.0±51.27**	121.0±21.04***	133.0±4.230***
Group 3(G_3)	382.0±10.50***	338.3±51.18*	155.9±37.17***	127.6±0.577***
Group 4(G_4)	385.6±11.03***	332.2±35.41*	169.5±25.21***	126.3±2.00***

All values presented as a Mean±Sem. The minimum value of p<0.05 was considered as significant. p<0.05, **<0.01, *<0.001 as compared with control group**

Group 1: Bagalkot collection Group 2: Vijayapur collection Group 3: Hungund collection Group 4: Raichur collection

Table 5. Effect of oral administration of karchikai powder on serum cholesterol (mg/dL) of diabetic rats

(Landraces)	Days of Estimation			
	0th day	7th day	14th day	21st day
Normal	151.9±10.55***	129.0±5.508***	133.3±4.325***	132.3±6.058***
Control	437.2±12.48	386.0±10.50	380.0±5.686	334.3±27.57
Group1	370.9±22.01***	284.6±5.686***	177.3±61.70***	148.9±10.09***
Group2	369.9±19.51***	281.6±15.28***	245.6±14.92***	138.5±3.233***
Group3	381.6±6.351**	275.3±1.00***	242.2±4.985***	146.1±12.97***
Group4	355.0±15.01***	268.6±20.60***	238.1±5.283***	136.0±1.293***

All values presented as a Mean±Sem. The minimum value of p<0.05 was considered as significant. p<0.05, **<0.01, *<0.001 as compared with control group Group 1: Bagalkot collection**

Group 2: Vijayapur collection Group 3: Hungund collection Group 4: Raichur collection

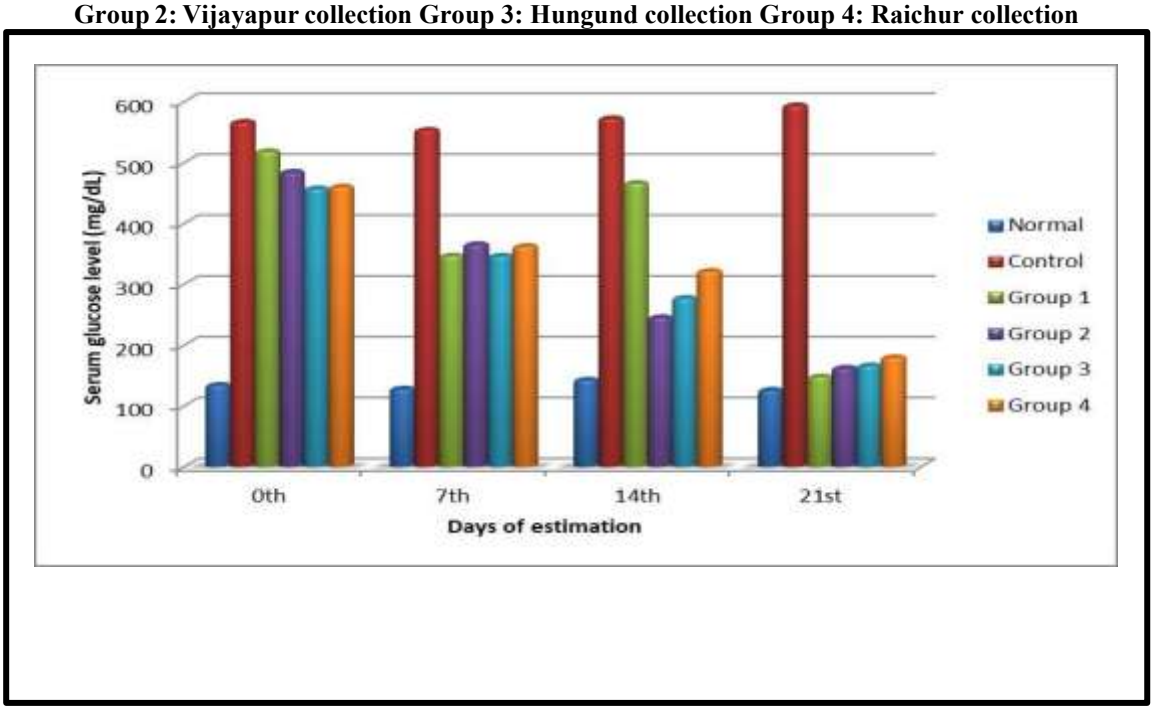

Figure 1: Pictorial representation of serum glucose levels in different groups of rats representing: Group 1: Bagalkot collection, Group 2: Vijayapur collection, Group 3: Hungund collection, Group 4: Raichur collection

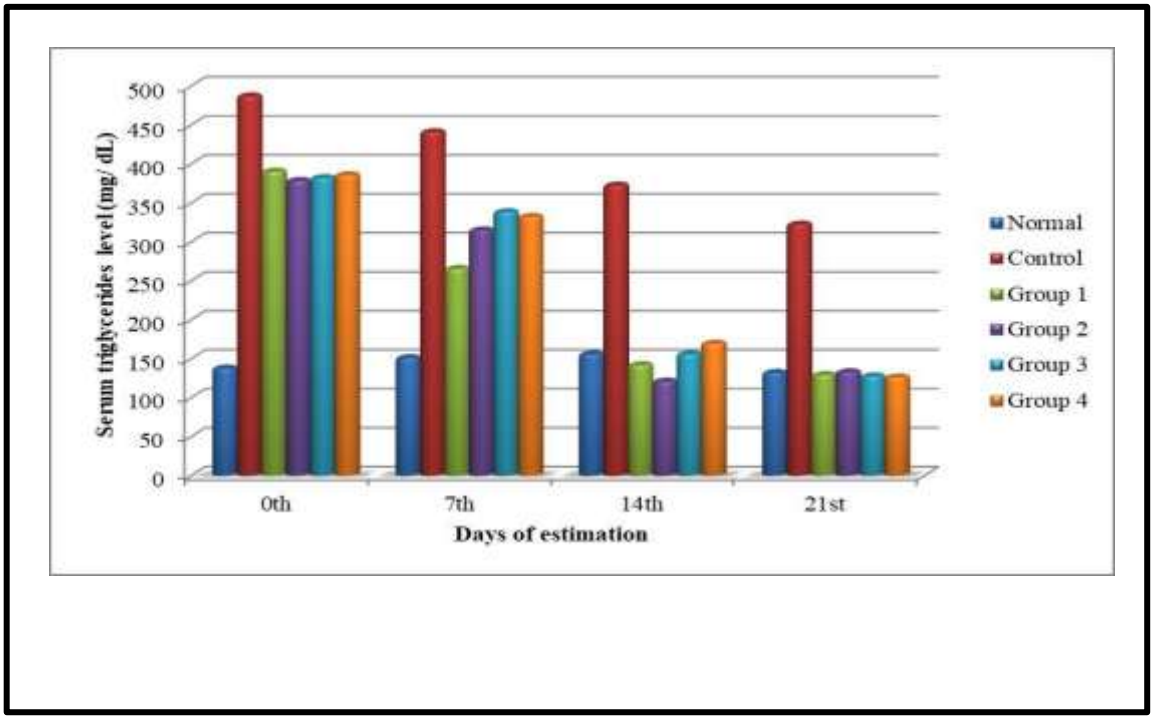

Figure 2: Pictorial representation of serum triglycerides levels in different groups of rats representing: Group 1: Bagalkot collection, Group 2: Vijayapur collection, Group 3: Hungund collection, Group 4: Raichur collection

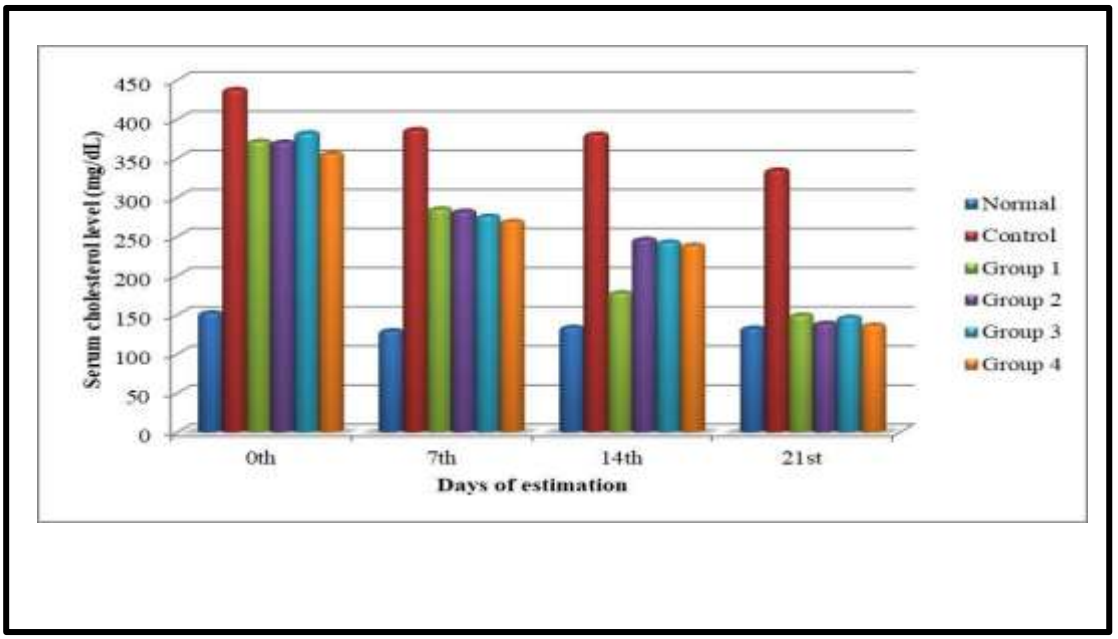

Figure 3: Pictorial representation of serum cholesterol levels in different groups of rats representing: Group 1: Bagalkot collection, Group 2: Vijayapur collection, Group 3: Hungund collection, Group 4: Raichur collection