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EVALUATION AGAINST STREPTOZOCIN INDUCED DIABETIES OF *ACALYPHA COMMUNIS AND BERGIA CAPENSIS* PLANTS EXTRACT

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ABSTRACT:

To evaluate the effects of Bergia capensis and Acalypha communis extracts on rats with diabetes brought on by streptozocin. The Euphorbiaceae family member Acalypha communis has historically been used in the treatment and/or management of a wide range of illnesses, including diabetes, jaundice, hypertension, fever, liver inflammation, dysentery, and respiratory issues. Bergia capensis is a tiny, annual herb that thrives in moist environments. It is a member of the Elatinaceae plant family, which is frequently found in seasonally flooded parts of tropical and subtropical countries. It is utilised in traditional medicine and folk rites. 42 male albino rats, weighing between 250 and 300 gms, were allocated into seven groups at random (n = 6). Both the aqueous and the ethanol extracts were diluted in tween 80 and then administered orally for a total of 14 days. On days 1, 7, and 14 of the treatment plan, blood samples will be taken by puncturing the retro orbital plexus while under mild ether anaesthesia. The estimation of glucose was done using plasma, while the estimation of the lipid profile, SGOT, SGPT, ALT, DB, TB, and creatinine was done using serum. The mean SE was used to express every piece of experimental data. The one-way analysis of variance (ANOVA) was used to examine the data. P values at or below the 0.05 threshold were deemed statistically significant. Liver enzymes such ALT,

AST, ALP, and bilirubin were assessed in albino rats to examine the role of liver markers in diabetes mellitus (DM). The liver indicators SGOT and SGPT are strongly elevated in diabetic control.

Keywords: *Acalypha communis*, *Bergia capensis*, diabetes, and liver markers.

INTRODUCTION:

Acalypha communis Müll.The Euphorbiaceae family[1-4], which includes herbs, shrubs, and small trees, includes Arg. Shrubs or suffrutex with simple or glandular hair indumentum and resinous brilliant drops on leaves and inflorescences. Male inflorescences are axillary, whereas female or androgynous inflorescences are terminal. Spicate inflorescences are mostly unisexual but can rarely be androgynous. Although some species can also be found in temperate regions, the majority of species are found in the tropics and subtropics. Nearly 350 chemical constituents of the Acalypha[7-16] genus have been reported, the majority of which are sesquiterpenoids, alkaloids, phenylpropanoids, butanolides, lucidones, flavonoids, etc. Plants from Acalypha genus are traditionally used in the treatment and/or management of diverse ailments such as diabetes, jaundice, hypertension, fever, liver inflammation, schistosomiasis, dysentery, respiratory problems including bronchitis, asthma and pheumonia as well as skin conditions such as scabies, eczema and mycoses. Traditionally used Acalypha species have been reported to possess at least one of the following biological activities: antimicrobial, anti-diabetic, antioxidant, anti-inflammatory, larvidal, pupicidal, hepatoprotective, anticancer, leishmanicidal, antihyperglycemic, antihypertensive, anti-venom, analgesic, anthelmintic, antiemetic, laxative, expectorant, diuretic, post-coital antifertility effects and wound healing. Small, wet-growing annual herb Bergia capensis[17-19] is native to Africa, southern China, and tropical Asia. A tiny plant family known as the Elatinaceae is often found in areas of tropical and subtropical climates that experience seasonal flooding. It flowers and produces fruit in India from August to November. Phenolic acids such delphinidin, ellagic acid, quercetin, cyanidin, and kaempferol have been shown to be present in Bergia members. Traditional medicine and rituals employ bergia capensis. In India, the leaves are administered to animals suffering from food illness. The leaves are also used to treat gastrointestinal worms. The objective of the current research is to evaluate the diabetic activity in rats with diabetes produced by streptozocin[5,6], Acalypha communis, and Bergia capensis[20] extracts. 42 male albino rats, weighing between 250 and 300 gms, were allocated into seven groups at random (n = 6). Both the aqueous and the ethanol extracts were

diluted in tween 80 and then administered orally for a total of 14 days. On the first, seventh, and fourteenth days of therapy, blood samples are taken by puncturing the retro orbital plexus while receiving a little ether anaesthetic. Estimates of SGOT, SGPT, ALT, DB, TB, and creatinine were made using serum, whereas estimates of glucose and the lipid profile were made using plasma.

MATERIALS AND METHODS:

Induction of diabetes: Prior to use, streptozotocin will be kept on ice after being freshly dissolved in citrate buffer. One intraperitoneal dose of STZ (60 mg/kg) will induce type 2 diabetes in the overnight starved rats. To get through the early hypoglycemia phase, 5% glucose will be given orally in a feeding bottle during the next day after 4 hours of STZ administration. After 72 hours of STZ treatment, rats with plasma glucose levels exceeding 250–300 mg/dL will be deemed diabetic and employed in the experiment.

Experimental animals: Forty two male albino rats weighing 250-300 gms were randomly divided into seven groups (n=6). Both the aqueous and ethanol extract was dissolved in distilled water and tween 80 respectively and given orally for 14days.

Experimental design: Male Albino rats were randomly divided into seven different groups of six rats each.

Group I: Control rats fed with saline.

Group II: Rats were administered with STZ to induce diabetes and fed with saline.

Group III: Diabetic rats were treated with 5 mg/kg of Glibenclamide for 14 days

Group IV: Diabetic rats were treated with CSAE1 (200mg/kg) for 14 days

Group V: Diabetic rats were treated with CSAE2 (400mg/kg) for 14 days

Group VI: Diabetic rats were treated with CSEE1 (200mg/kg) for 14 days

Group VII: Diabetic rats were treated with CSEE2 (400mg/kg) for 14 days

Blood samples will be collected by puncturing the retro orbital plexus under light ether anesthesia on 1, 7 & 14th day of treatment schedule. Plasma was used to estimate the glucose; serum was used for estimating the lipid profile, SGOT, SGPT, ALT, DB, TB, and creatinine. Antioxidant such as catalase activity, lipid peroxidation, and glutathione peroxidase were estimated.

Glucose measurements: Blood glucose was measured on day 1, day 7 and day 14 by GOD-POD method.

Lipid profile

Total cholesterol (TC)

TC was determined in the serum using the CHOD-POD technique and the standard kit. The fatty acids will initially be separated from the cholesterol in this technique by the enzyme cholesterol esterase. In the presence of peroxidase, the cholesterol will combine with cholesterol oxidase to produce free radicals (H2O2), which will then interact with 4-aminoantipyrine and phenol to make quinoneimine.

Hi**gh density lipoproteins (HDL)**

HDL and LDL help in the transport of cholesterol from the liver to various parts of the tissue. The HDL in the serum was measured using the standard kit according to the precipitation end point method. The serum was precipitated using the phosphotungstic acid and magnesium chloride. After centrifugation, the samples were measured at the absorbance of 505 nm. **Estimation of liver markers**

Aspartate Transaminase (AST):

AST activity in plasma was determined by the UV kinetic method. The reaction was measured at the absorbance of 340 nm with the rate of decrease in absorbance.

Alanine aminotransferase (ALT)

The activity of ALT in plasma was determined using the UV kinetic method.

Estimation of alkaline phosphatase (ALP)

The UV kinetic technique was used to ascertain the activity of ALP in plasma. Colourless 4-nitrophenoxide is created in an alkaline environment by converting colourless -nitrophenol, which takes on a very bright yellow hue.

Bilirubin

Using the usual kit, direct and total bilirubin were measured. This assay's basic working concept is that sulfanilic acid combines with sodium nitrite (NaNO2) to make diazotized sulfanilic acid (p-diazo

benzene sulfonic acid), which then reacts with conjugated direct bilirubin at an alkaline pH to produce a compound of blue colour. The sample was combined with sodium nitrite and DMSO to test the total bilirubin, and the absorbance was determined at 546 nm.

Creatinine

Creatinine in the serum was measured using Jaffe method. The principle is creatinine forms an orange colored red complex in alkaline picrate solution. The difference in absorbance at fixed times during the conversion is directly proportional to the creatinine in the sample.

Estimation of antioxidant enzymes

Estimation of Superoxide dismutase (SOD)

To the tubes containing 0.75 mL ethanol and 1.5 mL of prechilled chloroform, 0.1 mL of the tissue homogenate was added. After centrifugation, 1mL of carbonate-bicarbonate (pH-10.2) buffer and 0.5 mL of 0.6 mM EDTA solution were combined with the supernatant. The addition of 0.5 mL of newly generated, 1.8 mM epinephrine started the reaction, and the UV spectrophotometer was used to quantify the rise in absorbance at 480 nm. The quantity of protein needed to provide 50% inhibition of epinephrine autoxidation was equal to one unit of SOD activity.

Estimation of GSH

500 mg of tissue sample was homogenized in 5mL of ice cold trichloroacetic acid in a homogenizer. Glutathione measurement was performed according to the modification of the Ellman procedure. Briefly, after centrifugation at 3000 rpm for 10 minutes, 0.5 mL of the supernatant was added to 2 mL of 0.3 M disodium hydrogen phosphate solution. Then 0.2 mL of dithiobisnitrobenzoate was added and the absorbance was measured at 412 nm immediately after mixing.

Estimation of lipid peroxidation

Thiobarbituric reactive material generation was observed in order to gauge the level of lipid peroxide formation. 15% w/v TCA, 0.375% thiobarbituric acid (TBA), and 0.25N hydrochloric acid make up the TCA-TBA-HCL reagent stock solution. TBA was dissolved in this solution using a small amount of heat. 2 mL of the TCA- TBA-HCL reagent were added to 1 mL of the sample and carefully mixed. The solution is cooked in a bath of boiling water for 15 minutes. After cooling, centrifugation at 2500 rpm

for 10 minutes was used to separate the flocculent precipitate. At 535 nm, the sample's absorbance was compared to a blank value.

Determination of glucose-6-phosphatase

The reaction mixture contained 3 mL of potassium citrate (0.1 M, pH 6.8), 5 mL glucose-6- phosphate (0.01 M, pH 6.8) and 2 mL homogenate. Blank was prepared using 5 mL of distilled water instead of glucose-6-phosphate. Both the tubes were incubated at 30°C. After 1 hr, 1 mL of 10% trichloroacetic acid was mixed and filtered. Inorganic phosphate was determined in 1 mL of the filtrate

Determination of fructose-1, 6-diphosphatase

The reaction mixture at a final volume of 2 mL contained 1.2 mL of Tris-HCl buffer, 0.1 mL of fructose-1,6-diphosphatase, 0.25 mL of MgCl2, 0.1 mL of KCl, 0.25 mL of EDTA and 0.1 mL of the homogenate. Incubate the reaction mixture for 5 min at 37°C. 1mL of 10% TCA was added and centrifuged. Inorganic phosphate was determined in 1 mL of the filtrate.

Determination of phosphoglucose isomerase enzyme

1mL of the freshly prepared buffered substrate (glucose-6-phosphate in 10 M borate buffer, pH 7.8) was pipetted into two large test tubes named “test” and “blank”.0.1 mL of the homogenate was added to both the tubes with or without incubation exactly for 30 min at 37°C in a water bath. At the same time, in the test tubes named “standard” and “standard blank,” 1 mL of the standard solution (fructose in 0.25% benzoic acid) and 1 mL of distilled water were added. 9 mL of color reagent (HCl, resorcinol-thiourea reagent and distilled water, 7:1:1) was added to four tubes and placed in a 75°C water bath for 15 min. The absorbance was measured at 410 nm.

Determination of aldolase enzyme

In a test tube set at 38 degrees Celsius, the reaction mixture included 1 mL of Tris buffer (0.1 M, pH 8.6), 0.25 mL of fructose-1,6-diphosphatase solution (0.05 M, pH 8.6), and 0.25 mL of hydrazine solution (0.56 M, pH 8.6). This was then diluted with water to a level of 2.5 mL. After adding 0.5 mL of the homogenate to the mixture, 2 mL of 10% trichloroacetic acid was added to halt the reaction after 30 minutes. Similar steps were taken to make the blank, except the fructose-1,6-diphosphatase was added

after the trichloroacetic acid. It centrifuged the tubes. In 1 mL of the supernatant, 0.75 N of NaOH was added. Dinitrophenylhydrazine was added after 10 minutes, and the tubes were submerged in 38°C water for 10 min. 7 mL of NaOH was added to give a total volume of 10 mL. The absorbance was read at 540 nm after 10 min of the addition of alkali.

RESULTS:

Table 1. - The effect leaf extract of Acalypha communis Mull.Arg and Bergia capensis L, on glucose in diabetic rats.

S.No	Parameter	Groups	Mean ± SE
1.	Glucose (Day-1)	Control	91.28 ± 11.78
		STZ	356.73 ± 26.13
		Glib	356.67 ± 16.28
		AE1	376.06 ± 26.99
		AE2	426.38 ± 22.76
		EE1	405.28 ± 21.46
		EE2	456.69 ± 14.90
2.	Glucose (Day-7)	Control	63.07 ± 4.47
		STZ	398.3 ± 14.34
		Glib	96.13 ± 2.0=35
		AE1	128.24 ± 15.83
		AE2	173.53 ± 13.93
		EE1	185.313± 18.56
		EE2	234.44 ± 23.86
3.	Glucose (Day-14)	Control	91.56 ± 3.49
		STZ	344.91 ± 18.40
		Glib	68.76 ± 4.46
		AE1	104.58 ± 7.59
		AE2	152.43 ± 15.79
		EE1	94.9 ± 7.17
		EE2	90.42 ± 2.52

Table 2. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on lipid profile in diabetic rats.

S. No	Parameter	Groups	Mean ± SE
1.	TC	Control	86.33 ± 7.56
		STZ	262.54 ± 17.99
		Glib	236.54 ± 18.56
		AE1	199.22 ± 14.56
		AE2	198.52 ± 10.93
		EE1	234.26± 11.78
		EE2	267.88 ± 6.56
2.	TG	Control	64.23 ± 8.57
		STZ	202.56 ± 13.69
		Glib	156.57 ± 3.46
		AE1	178.19 ± 3.86
		AE2	206.35 ± 10.96
		EE1	160.56 ± 4.56
		EE2	222.36 ± 11.36
3.	HDL	Control	53.36 ± 3.91
		STZ	32.42 ± 1.56
		Glib	121.33 ± 14.56
		AE1	56.69± 8.72
		AE2	107.12± 9.63
		EE1	171.26 ± 14.79
		EE2	192.62 ± 8.66
4.	LDL	Control	16.33 ± 6.56
		STZ	189.73 ± 15.22
		Glib	93.56 ± 19.23
		AE1	107.13 ± 11.56
		AE2	39.96 ± 12.26
		EE1	40.86 ± 5.23
		EE2	34.76 ± 11.83
5.	VLDL	Control	12.67 ± 1.70
		STZ	40.26 ± 2.61
		Glib	31.94 ± 0.56
		AE1	35.66 ± 0.76
		AE2	41.69 ± 2.16
		EE1	32.16 ± 0.99
		EE2	40.36 ± 2.15

Table 3. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on AI, CRI and % Protection in diabetic rats.

S.NO	Parameter	Groups	Mean ± SE
1.	**AI**	Control	0.57 ± 0.69
		STZ	7.12 ± 0.41
		Glib	6.33 ± 0.44
		AE1	3.06 ± 0.56
		AE2	0.81 ± 0.14
		EE1	0.46 ± 0.11
		EE2	0.41 ± 0.08
2.	**CRI**	Control	1.57 ± 0.15
		STZ	8.12 ± 0.37
		Glib	2.06 ± 0.25
		AE1	4.06 ± 0.83
		AE2	1.81 ± 0.14
		EE1	1.46 ± 0.09
		EE2	1.41 ± 0.08
3.	**% Protection**	Control	--
		STZ	--
		Glib	85.65 ± 2.7
		AE1	56.11± 12.20
		AE2	88.32 ± 2.34
		EE1	93.708± 0.66
		EE2	94.06 ± 1.44

Table 4. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on AST, ALT, ALP and creatinine in diabetic rats.

S.NO	Parameter	Groups	Mean ± SE
1.	**AST**	Control	44.43 ± 11.38
		STZ	152.73 ± 17.10
		Glib	83.33 ± 12.34
		AE1	113.57 ± 15.36
		AE2	69.72 ± 14.46
		EE1	126.68 ± 14.17
		EE2	66.78 ± 13.60
2.	**ALT**	Control	48.43 ± 10.67
		STZ	105.78 ± 9.78
		Glib	65.13 ± 5.41
		AE1	61.13 ± 4.55
		AE2	46.77 ± 10.55
		EE1	33.88 ± 9.88
		EE2	27.33 ± 3.59
3.	**ALP**	Control	65.12 ± 5.41
		STZ	317.22 ± 11.44
		Glib	80.64 ± 10.5
		AE1	407.23 ± 20.35
		AE2	179.09 ± 18.49
		EE1	377.15 ± 16.78
		EE2	369.3 ± 20.53
4.	**Creatinine**	Control	1.27 ± 0.26
		STZ	2.47 ± 0.28
		Glib	1.35 ± 0.19
		AE1	1.91 ± 0.07
		AE2	1.94 ± 0.04
		EE1	1.05 ± 0.24
		EE2	1.82 ± 0.04

Table 5. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on TB, DB, SOD, LPO and GSH in diabetic rats.

S.No	Parameter	Groups	Mean ± SE
1.	**TB**	Control	0.79 ± 0.56
		STZ	3.14 ± 0.56
		Glib	1.99 ± 0.80
		AE1	0.55 ± 0.14
		AE2	0.47 ± 0.44
		EE1	0.47 ± 0.07
		EE2	0.45 ± 0.05
2.	**DB**	Control	0.22 ± 0.09
		STZ	5.55 ± 1.77
		Glib	6.67 ± 2.23
		AE1	1.65 ± 0.23
		AE2	1.39 ± 0.51
		EE1	1.89 ± 0.37
		EE2	1.01 ± 0.06
3.	**SOD**	Control	39.58 ± 1.88
		STZ	24.65 ± 1.77
		Glib	36.33 ± 1.83
		AE1	31.88 ± 1.9
		AE2	32.93 ± 0.79
		EE1	34.9 ± 1.84
		EE2	37.53 ± 1.96
4.	**LPO**	Control	15.03 ± 1.11
		STZ	32.10 ± 1.55
		Glib	24.98 ± 2.50
		AE1	23.57 ± 1.24
		AE2	22.93 ± 2.25
		EE1	21.40 ± 1.16
		EE2	18.0 ± 1.18
5.	**GSH**	Control	3.63 ± 0.38
		STZ	1.70 ± 0.18
		Glib	2.05 ± 0.16
		AE1	2.16 ± 0.13
		AE2	2.74 ± 0.44
		EE1	3.16 ± 0.54
		EE2	3.31 ± 0.14

Table 6. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on G-6-P, F-6-P, G- 6-PDH, PGI and Aldolase in diabetic rats.

S.No	Parameter	Groups	Mean ± SE
1.	**Glu-6ptase**	Control	0.24 ± 0.06
		STZ	0.46 ± 0.07
		Glib	0.25 ± 0.03
		AE1	0.34 ± 0.01
		AE2	0.30 ± 0.02
		EE1	0.28 ± 0.04
		EE2	0.27 ± 0.01
2	**Fru-6ptase**	Control	0.47 ± 0.09
		STZ	0.70 ± 0.03
		Glib	0.49 ± 0.04
		AE1	0.54 ± 0.02
		AE2	0.53 ± 0.02
		EE1	0.51 ± 0.02
		EE2	0.49 ± 0.02
3	**G-6PDH**	Control	3.27 ± 0.26
		STZ	0.99 ± 0.28
		Glib	3.10 ± 0.89
		AE1	2.56 ± 0.05
		AE2	2.62 ± 0.49
		EE1	2.72 ± 0.39
		EE2	3.09 + 0.46
4	**Phosphogluco-isomerase**	Control	30.03 ± 0.86
		STZ	18.53 ± 0.87
		Glib	28.73 ± 0.89
		AE1	22.63 ± 0.88
		AE2	24.77 ± 0.97
		EE1	25.68 ± 0.56
		EE2	27.98 ± 0.59
5.	**Aldolase**	Control	0.23 ± 0.05
		STZ	0.47 ± 0.05
		Glib	0.26 ± 0.03
		AE1	0.33 ± 0.03
		AE2	0.32 ± 0.02
		EE1	0.30 ± 0.02
		EE2	0.28 ± 0.03

Table 7. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on glucose, SGOT and SGPT in pre-treated rats.

S.NO	Parameter	Groups	Mean ± SE
1.	**Glucose**	Control	85.23 ± 4.55
		STZ	393.12 ± 8.69
		Glib	56.69 ± 6.56
		AE2	138.68 ± 4.54
		AE4	107.37 ± 6.07
		EE2	101.29 ± 6.82
		EE4	96.64 ± 4.33
2.	**SGOT**	Control	42.15 ± 6.65
		STZ	138 ± 7.55
		Glib	78.05 ±9.46
		AE2	104.12 ± 4.15
		AE4	60.66± 6.21
		EE2	100.75 ± 4.58
		EE4	58.02 ± 6.61
3.	**SGPT**	**Control**	51.69± 3.89
		STZ	100.22 ± 5.41
		Glib	59.56 ± 4.86
		AE2	60.21 ± 3.56
		AE4	40.39 ± 1.11
		EE2	31.86 ± 1.65
		EE4	28.11 ± 1.66

Table 8. - The effect of leaf extracts of Acalypha communis Mull.Arg and Bergia capensis L, on TB and DB in pre-treated rats.

S. No	Parameter	Groups	Mean ± SE
1.	**TB**	Control	0.99 ± 0.11
		STZ	4.01 ± 0.43
		Glib	2.08 ± 0.21
		AE2	0.67 ± 0.06
		AE4	0.52 ± 0.07
		EE2	0.49 ± 0.06
		EE4	0.46 ± 0.07
2.	**DB**	Control	0.23 ± 0.11
		STZ	5.59 ± 1.54
		Glib	6.54 ± 2.56
		AE2	1.66 ± 0.25
		AE4	1.38± 0.56
		EE2	1.96 ± 0.38
		EE4	1.01 ± 0.07

CONCLUSION:

According to the examination of the studies data, streptozocin has been shown to cause diabetes at the levels used, as shown by biochemical markers. Both the test drug and the reference standard reversed harmful effects. Our study provided experimental support for the assertion that extracts of *Acalypha communis and Bergia capensis* have anti-diabetic properties. In both of these models, antidiabetic activity at doses of 200 mg/kg and 400 mg/kg was observed, which was corroborated by biochemical parameters.

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