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Pharmacological Investigation of *Asparagus racemosus* and *Boerhavia diffusa* for Antidiabetic Activity in Dexamethasone-Induced Diabetic Rats

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Abstract

Diabetes is a disorder with improper metabolism, characterized by higher blood glucose levels. insulin resistance or deficiency of insulin. *Boerhavia diffusa, Asparagus recemosus* well well-known herbs in traditional medicine. We have evaluated its potential for claim for anti-diabetic action. *Boerhavia diffusa*, *Asparagus racemosus* were collected from the local region of Raipur. Extracted by using the hydroalcoholic method of extraction. Diabetes was induced by using dexamethasone, and different pathological markers were determined. Rats weighing between 200 - 250 g are selected for the study and divided randomly into six groups: normal, diabetic, standard (glibenclamide, 2.5 mg/kg), *Boerhavia diffusa* (200mg/kg), *Asparagus racemosus* extract (200mg/kg), and one group with both the test drugs. Dexamethasone was initially given for 10 days, followed by on day 11, treatment started for the next 14 days. Treatment was given for 14 days. The result shows a significant ($P < 0.05$) improvement in glucose, lipid profile and liver function, creatinine, and antioxidant potential parameters. Results indicate that *Boerhavia diffusa*, *Asparagus recemosus* posses anti diabetic and lipid-lowering potential, findings which support its traditional claim for therapeutic uses

Keywords: *Boerhavia diffusa, Asparagus recemosus*, Dexamethasone, Glibenclamide, Lipid profile, Glycogen.

Introduction

From ancient literature, it has been observed that diabetes was found from ancient time about 700-200 B.C. Ancient scriptures indicate that the term 'Madhumeha' for diabetes was used by Sushruta. It can be defined as it is a metabolic disorder that leads to chronic hyperglycemia because of abnormal metabolism of carbohydrate due to a deficiency in insulin secretion or resistance to insulin action. [1, 2]

According to the World Health Organization, 80% of the remote area population depends on traditional medicine as medication (WHO, 2000).[3] This amazing herb is known as the “Queen of herbs”. It is the key tonic for the female as a rejuvenative use in Ayurveda, its biological source, *A racemosus*, belongs to the family Asparagaceae [4]

In traditional therapy, it has been observed that *A racemosus* extract has been reported to possess antioxidant, anti-abortifacient, antioxytoxic, spasmodic to uterus, hypoglycemic, anti-hypertensive, anticoagulant, antiviral, and anticancer.[5,6]

The genus Boerhavia L. (Family: Nyctaginaceae) contains 40 sub-tropical and tropical species[7]. The leaves *B diffusa* have traditionally been a useful antidote for liver complications, hypotension, skin diseases, night blindness, snake poisoning [8,9,10,] as well as in gonorrhoea, dropsy, asthma, night blindness, rheumatism, diabetes, liver disorder, and diseases of the kidney and heart.[11,12,13]

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Aim of present research work is to evaluate the anti-diabetic effect of *B diffusa* and *A racemosus* in dexamethasone induced diabetic rats.

Methods

Identification

The entire plant of *B diffusa* and *A racemosus* tubers were gathered from Raipur, India. *And* authenticated by Prof. NK Dubey, "Centre of Advanced Study in Botany, BHU, Varanasi, UP, India." voucher specimen was deposited (no: Liliaceae 2023/01 and Nyctageneceae 2023/01).

Extractions [14]

The entire plant of *B diffusa* and the tuber of *A racemosus were* gathered and rinsed with tap water. Dried in oven (40-50°C). Coarsely grinded. Soaked with pet ether for the removal of fatty acids. The substance was extracted using a mixture of ethanol and water (8:2), and the process involved maceration for 3 days using orbital shaking. Solvent was removed by using the vacuum drying technique.

Induction of diabetes

Dexamethasone (Dexa) 10 mg/kg/day injected s.c. for 10 days; on day 11, keep the animal overnight fasting and monitor blood glucose level, select those animals whose fasting and postprandial blood glucose levels were higher than those of the normal controls chosen for further study. From day 11 to day 24, groups 2, 3, 4, 5, and 6 continued to receive dexamethasone 10 mg/kg/day/sc (Table 1).[15]

Blood glucose estimation

It was determined by tail vein pricking method, by using Gluco-One glucose measurement strips and Dr. Morepen, glucometer.

Serum isolation: At the end of day 24, the animals were euthanized using chloroform according to CPCSEA guidelines, blood samples were collected from overnight fasted animals through cardiac puncture in plain vials, allowed to stand for 15 minutes, and serum was separated by cold centrifugation at 3000 rpm for 10 min. Blood was also collected in ethylene diamine tetraacetic acid (EDTA) coated vials for the analysis.

Table 1: Experimental Design

Group: I	Normal (CMC treated)
Group: II	Negative Control/ Dexamethasone (10 mg/kg/day)
Group: III	Dexa + Standard (Glibenclamide)
Group: IV	Dexa + HABD (200 mg/kg)
Group: V	Dexa + HAAR (200 mg/kg)
Group: VI	Dexa + HABD (100mg/kg) + HAAR (100mg/kg)
Number of animals in each group N= 5 HABD: Hydroalcoholic extract of *B. diffusa* HAAR: Hydroalcoholic extract of *A racemosus*	

Serum biochemical parameters: Serum Glutamate Pyruvate Transaminase (SGPT), Oxaloacetate Transaminase (SGOT), Alkaline phosphatase (ALP), serum albumin, bilirubin, and total protein were analysed using commercially available kits in a semi-automatic analyzer.

Hepatic antioxidant enzymes assay (estimation of MDA, GSH, SOD, and CAT)

Liver samples were dissected out and washed immediately with ice-cold saline to remove as much blood as possible. A Remi homogenizer prepared liver homogenates (5% w/v) in cold 50 mM Tris buffer (pH 7.4). The unbroken cells and cell debris were removed by centrifugation at 5000 rpm for 10 min using a Remi refrigerated centrifuge. The supernatant was used for the estimation of GSH,[16] malondialdehyde (MDA),[17]superoxide dismutase (SOD),[18] and catalase.[19]

Phytochemical Analysis[20]

The extracts were analyzed for phytochemical constituents using standard methods.

Results

Chemical tests for the hydroalcoholic extract of phytoconstituents of *Asparagus racemosus* and *Boerhavia diffusa* (Tables 2 and 3).

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Table 2: Phytoconstituents of *Asparagus racemosus*

Phytochemicals	Name of the test	HAAR
Carbohydrates	Molisch's test	+
Alkaloids	Dragendroff's test	-
Saponins	Frothing test	++
Phytosterols	Salkowski reaction	++
Glycosides	Killer-Killani's test	+
Triterpenoids	Liebermann-Burchard's test	-
Proteins & Amino acids	Ninhydrin test	-
Tannins & Phenolic compounds	Ferric chloride test	++
Presence: + and Absence - of phytoconstituents		

Table:3: Phytoconstituents of *Boerhavia diffusa*

Phytochemicals	Name of the test	HABD
Carbohydrates	Fehling's test	+
Alkaloids	Hager's test	++
Flavonoids	Ammonia test (modified)	+++
Tannins	$FeCl_3$ test	++
Saponins	Frothing test	++
Steroids	Liebermann-Burchard Test	++
Glycosides	Killer-Killani's test	-
Polyphenols	Folin ciocalteu test	++
Phlobatannins	Ring test	-
Presence: + and Absence - of phytoconstituents		

Statistical Analysis

Results are represented as Mean ± SEM. One-way ANOVA, followed by Dunnett's multiple comparison tests, was carried out. If (P<0.05), statistically significant. Comparison carried out between the mean of the normal and the NC group, the Test, and the NC group (Table 4). By using GraphPad Prism software version 5.0.

Table 4: Values are expressed as Mean ± SEM (n = 5); Values with * indicate SD (P < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (P < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Group	Initial Body weight	Final Body weight	Difference	% difference
Normal (CMC treated)	152.6	173.8	21.2	13.89
NC (Dexamethasone)	151.2	140.8	-10.4	-6.88
Dexa + Glib	154.8	171.6	16.8	10.85
Dexa + HABD	155.2	172.8	17.6	11.34
Dexa + HAAR	152.4	170.8	18.4	12.07
Dexa + HAAR+ HABD	155.2	173.2	18	11.60

Effect on Biochemical Profile

B. diffusa, A racemosus, and glibenclamide-treated rats significantly ($p < 0.05$) decrease the blood glucose level w.r.t diabetic rats on glucose in experimental animals are depicted in Figures 1 and 2, indicating that the test and standard drugs treated rats increase liver glycogen levels significantly ($p < 0.05$). Lipid profile improves in test and standard drug-treated groups, comparable to the disease control group (Table 5). (Table 6) indicates liver function parameters, it was significantly improved with test and standard drugs in treated animals in comparison with untreated. Table 7 indicates serum creatinine and urea levels, which represent the kidney function. There is a significant decrease in serum urea and creatinine levels in groups treated with the test and standard drug. Table 8 represents

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Effect of standard and test drugs on blood glucose level

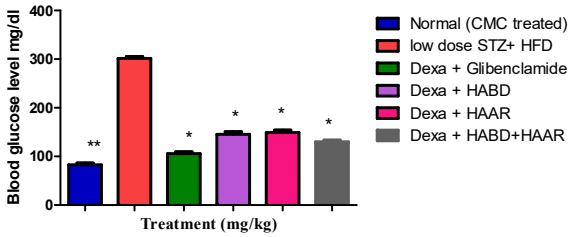

Fig. 1: Stated as Mean ± SEM (*n* = 5); Values with * indicate SD (*P* < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (*P* < 0.05) concerning Normal control (NC) and Negative control Dexamethasone induced group

Effect of standard and test drugs on liver glycogen level

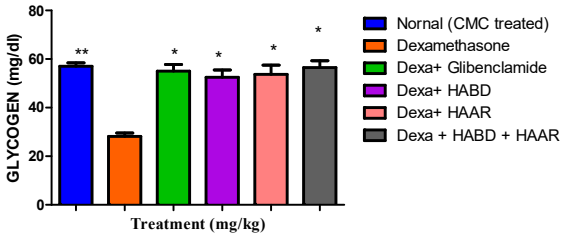

Fig. 2: Stated as Mean ± SEM (*n* = 5); Values with * indicate SD (*P* < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (*P* < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Effect of standard and test drugs on Lipid Profile

Table 5: Values are expressed as Mean ± SEM (*n* = 5); Values with * indicate SD (*P* < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (*P* < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Group	Triglyceride	LDL	VLDL	HDL	TC
Normal (CMC treated)	53.2 ± 3.14**	61.4 ± 3.14**	10.64 ±0.68**	14.34 ± 1.17**	86.38 ± 4.19**
NC (Dexamethasone)	103.2 ± 4.28	120.4± 5.93	20.64 ± .86	7.72 ± 0.73	148.76 ± 5.55
Dexa + Glib	57.8 ± 3.26*	77. 8 ±1.98*	11. 57 ±0.66*	12.8± 1.15*	102.16 ± 2.64*
Dexa + HABD	76. 4 ± 1.36*	85.8 ± 5.67*	20.12±0.20*	9.2±0.78*	118. 68 ± 6.48*
Dexa + HAAR	77.2± 2.13*	87.8 ± 6.30*	16.24±0.21*	9.26± 0.71*	124.9±0.66*
Dexa + HABD+ HAAR	67.2 ± 2.62*	80.2± 4.82*	13.44±0.52*	11.46 ± 1.41*	104.3 ± 5.34*

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Effect of standard and test drugs on Liver function test

Table 6: Values are expressed as Mean ± SEM (n = 5); Values with * indicate SD (P < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (P < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Group	SGOT	SGPT	ALP	BIL
Normal (CMC treated)	28.8±0.92**	29.4±1.03**	166.8±3.25**	0.354±0.076**
Dexa	51.2±5.91	55.4±1.91	397.2±3.99	1.41±0.138
Dexa + Glibenclamide	31.4±1.17*	34.8±1.2*	186.4±4.98*	0.56±0.137*
Dexa+ HABD	41.2±0.97*	36.2±0.97*	188.4±2.89*	0.66±0.104*
Dexa+ HAAR	38.8±2.51*	38.4±0.87*	183.8±2.97*	0.71±0.075*
Dexa + HABD + HAAR	34.8±0.97*	35.4±5.4*	214.8±3.18*	0.302±0.135*

Effect on Serum creatinine and Urea level

Table 7: Values are expressed as Mean ± SEM (n = 5); Values with * indicate SD (P < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (P < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Group	serum creatinine (mg/dl	Serum Urea (mg/dl)
Normal (CMC treated)	0.8 ± 0.06	85.5± 5.75
NC (Dexamethasone)	3.3 ± 0.23	325.6± 8.36
Dexa + Glib	1.13 ± 0.08	92.8± 6.5
Dexa + HABD	1.5±0.35	102.5±7.53
Dexa + HAAR	1.4±0.05	101.3±6.9
Dexa + HAAR+ HABD	1.2±0.09	95.6± 5.8

Effect on Serum Antioxidant enzyme level

Table 8: Values are expressed as Mean ± SEM (n = 5); Values with * indicate SD (P < 0.05) between negative control and test, standard drug-treated group. ** indicate SD (P < 0.05) for Normal control (NC) and Negative control Dexamethasone induced group

Group	Gsh (Mg Of Gsh/G Of Tissue)	Sod (Unit/Mg Of Tissue)	Catalase (Mm Of H_2o_2/G Of Tissue/Min	Lpo (Nm Of Mda/G Of Tissue
Normal (CMC treated)	28.34±2.45**	75.34 ± 3.25**	8.14±0.13	10.35±1.23
NC (Dexamethasone)	14.25± 1.38	29.53± 2.43	4.12±0.34	25.34±1.56
Dexa + Glib	23.25± 2.42*	73.42± 2.56*	7.63±0.56	11.23± 1.45
Dexa + HABD	22.45±1.57	62.24± 2.49*	6.19±0.86	12.52± 1.65
Dexa + HAAR	23.56±1.89*	65.25± 2.45*	6.8± 0.24	12.13± 1.34
Dexa + HAAR+ HABD	24.34±1.54*	68.34± 2.67*	7.1±0.13	11.45± 1.86

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Liver Histopathology

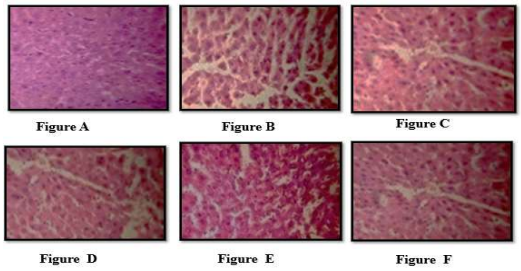

Fig. 3: A, B, C, D, E, F: Effects of HABD, HAAR and Glibenclamide on histopathological changes induced by Dexamethasone induced diabetes in Wistar rats. Figure A: Normal (CMC treated), Figure: B NC (Dexamethasone), Figure: C Dexamethasone + Glibenclamide, Figure D: Dexa + HABD, Figure E: Dexa + HAAR, Figure F: Dexa + HABD+ HAAR

the level of antioxidant enzymes, which is a marker for the determination of diabetic complications. There is a significant increase in the level of GSH, SOD, and Catalase in HABD, HAAR, and glibenclamide-treated groups, also there is a decrease in lipid peroxidation.

The extracts demonstrated significant antidiabetic activity by reducing blood glucose levels.

Discussion

Plant-based drugs are useful in the healing of different diseases and popular among researchers as a probable alternative to traditional pharmaceutical drugs. [21]

This research indicates that HAAR and HABD due to their effects. The chemical substances found in the extracts are responsible for the observed ability to control diabetes.

As anticipated, in this research, the disease decreased in BW, while HAAR, HABD, and standard drug inhibited it and caused an increase in BW. It may also contribute to insulin and glucose uptake.[22]

The effect of HAAR and HABD has been evaluated for its anti-diabetic activity in animals. Dexa administration at a dose of 10 mg/kg/day injected s.c. for 10 days intramuscular resulted in a significant rise in blood glucose (252.2 ± 2.45). Animals with glucose level of more than 150 mg/dl were selected for further studies. The effect of HAAR and HABD due to the glucocorticoid antagonism and the presence of other chemical constituents like terpenoids and tannins, which are reported to have antihyperglycemic action. PC showed a significant increase in insulin-assisted glucose uptake, which indicates that there was an upsurge in sensitivity to insulin.[23]

Based on existing research on HAAR and HABD, an effort has been made to assess their anti-diabetic action. According to the findings, both HAAR and HABD, along with their specific effects, significant drop in FBG in comparison with untreated rats.

In light of available literature on HAAR and HABD, an action was taken to evaluate the Pharmacological action of these drugs and establish a correlation with their positive effect on lipid profile. HAAR and HABD, and their combination ($p<0.05$), significantly decreased FBG levels in comparison with dexa-induced rats.

Dexa is responsible for hyperglycemia by either decreasing or enhancing insulin secretion or resistance. Glucocorticoids are employed as temporary steroid treatment for acute gout, respiratory disorders, and cancer. In the field of organ transplantation, it is commonly used as an immunosuppressant.[24] When it is employed to

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induce diabetes, it may increase glucose formation in the liver, enhancing resistance of insulin resistance, and diminishing insulin secretion.[25]

The roots of HAAR have been proven to improve insulin secretion in both the perfused pancreas and isolated islets.[26] HABD leaf extract. Possessing the ability to lower glucose levels, this compound exhibited good outcomes in rats with STZ.[27]

This research shows an elevation of oxidative effect, evidenced by elevated levels of key antioxidant enzymes SOD, CAT, and LPO in DM and a SD in the level of GSH. It is responsible for different diabetic complications.[28–30] It is responsible for generation of reactive oxygen species overwhelms the body's endogenous antioxidant defenses.

The effects of the HABD, HAAR on liver histological sections were examined. Animals treated with HABD, HAAR showed protective effects. The results indicated that the dexamethasone-treated group exhibited fatty liver, necrosis, hyperplasia, and inflammation, while the liver cells in the HABD and HAAR-treated groups appeared healthy.

Conclusion

The research offers proof of the antidiabetic properties of HAAR and HABD. More research is needed to identify and understand the active components of the plant, as well as to assess their safety and effectiveness in humans. This research demonstrates that the combination of HAAR and HABD has an action in diabetic rats induced by dexamethasone. Thus, the results obtained in the present investigation indicate that HAAR and HABD may prove to be useful in insulin resistance owing to their antioxidant activity and ability to increase glucose uptake. It was also noted that correcting the lipid profile in diabetic rats is of great significance. Histopathological study of the liver also indicates protective actions of HAAR and HABD. More research is required to fully understand how the plant extract works, including studying its different components and how they interact with the body.

Conflict of Interests

There are no conflicts of interest.

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