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Effect of *Woodfordia fruticosa* on dexamethasone induced insulin resistance in mice

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Abstract: Diabetes is a group of syndrome characterized by hyperglycemia, altered metabolism of lipids, carbohydrates and proteins, resulting in an increased risk of complications from vascular disease. The flowers of *Woodfordia fruticosa* (L.) Kurz, Lythraceae, have been used traditionally in the treatment of diabetes, dysentery, diarrhea, other bowel complaints, internal haemorrhages, in leucorrhoea and menorrhagia. Externally powdered flower is sprinkled over foul ulcers and wounds for diminishing their discharge and promoting granulations. In Konkan leaves are used in bilious sickness. W. fruticosa is also reported to have DNA topoisomerase inhibitor, antibacterial, antifertility, antipeptic ulcer, free radical scavenging, and hepatoprotective activity. *W. fruticosa* is a medicinal plant used to treat a wide range of disorder including diabetes. The present work investigates the effects of the WF in dexamethsone induced insulin resistance in mice. The results of animal study revealed that the extract at dose 100, 200 and 400 mg/kg was found to be significant (p<0.01) after 22 days of treatment. Further isolation studies afforded an anthraquinone glycoside, chrysophanol-8-*O*-β-D-glucopyranoside. Moreover further experiments will be required to identify their exact mechanism of action.

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Introduction

Diabetes is a group of syndrome characterized by hyperglycemia, altered metabolism of lipids, carbohydrates and proteins, and an increased risk of complications from vascular disease (Goodman & Gilman, 2001). It may occasionally arise secondarily from any disease causing extensive destruction of pancreatic islets, including pancreatitis tumors, certain drugs iron overload (hemochromatosis), certain acquired or genetic endocrinopathies, and surgical excision. However the most common and important forms of diabetes mellitus arise from primary disorders of the islet cell-insulin signaling system (Ramzi et al., 2000). Most patients can be classified clinically as having either type 1 diabetes mellitus (insulin dependent diabetes mellitus) or type 2 diabetes mellitus (Non insulin dependent diabetes mellitus). The number of people with diabetes is increasing due to population growth, aging, urbanization, and increasing prevalence of obesity and physical inactivity. In developing countries, the majority of people with diabetes are in the age range of 45 to 64 years. In contrast, the majority of people with diabetes in developed countries are >64 years of age. By 2030, it is estimated that the number of people with diabetes >64 years of age will be >82 million in developing countries and >48 million in developed countries (Wild et al., 2004). There are lots of chemical agents available to control and treat diabetic patients, but total recovery from diabetes has not been reported up to date. Alternatives to these synthetic agents, plants provide a potential source of antihyperglycemic drugs and are widely used in several traditional systems of medicine to prevent diabetes with less side effect and compatible with the physiological system. There is a constant attempt by scientists to understand the active principle present in medicinal plants with antidiabetic properties. Metformin, a less toxic biguanide and potent glucose lowering agent was developed from *Galega officinalis* and used to treat diabetes. Out of dozens of oral medications for diabetes, only one medication (metformin) is approved for use in children and it has been originated from herbs (Jerald et al., 2008). Thus there is a dire need to search for more phytoconstituents for the treatment of diabetes.

The flowers of *Woodfordia fruticosa* (L.) Kurz, Lythraceae, have been used traditionally in the treatment of diabetes, dysentery, diarrhea, other bowel complaints, internal haemorrhages, in leucorrhoea and menorrhagia. Externally powdered flower is sprinkled over foul ulcers and wounds for diminishing their discharge and promoting granulations. In Konkan leaves are used in

bilious sickness (Nadkarni., 2009, Kunwar et al., 2009). The present communication reports the antidiabetic activity of the ethanolic extract of the leaves of *W. fruticosa* (WF) by employing dexamethasone induced insulin resistance in mice, along with the estimation of the lipidemic parameters. In addition to this, isolation of the compound that may be responsible for the said activity was also carried out.

Material and Methods

Plant collection

The leaves of *Woodfordia fruticosa* (L.) Kurz, Lythraceae, were collected from hilly areas in Aurangabad (India), and authenticated by Botanical Survey of India, Pune with a voucher specimen no. BSI/WRC/Tech./2010/567. A herbarium was also deposited for future reference.

Reagents

Dexamethasone was procured from Cadila Pharmaceuticals Ltd. Pioglitazone, a standard antidiabetic agent was purchased from Sun Pharma Sikkim, India. Glucose, triacylglyceride and cholesterol kits were procured from Biolab Pvt. Ltd, India. All the chemicals and reagents were of analytical grade.

Extraction and isolation

The leaves of *W. fruticos*a (WF) were washed with water, shade dried and then powdered to an optimum size for extraction. The powdered drug (120 g) was extracted using Soxhlet apparatus with 95% ethanol for about 36 h. The extract was filtered and concentrated in vacuum under reduced pressure using rotary flash evaporator (Dan & Dan, 1984). A dark green colored semisolid mass weighing 18.45 g (15.38% w/w) was obtained. The extract (1 g) was taken into the minimum amount of methanol and was stirred until it gets well dissolved into the methanol and was filtered. Cold ether was slowly added into the filtrate and an anthraquinone glycoside (0.35 g) with a yield of 35% was obtained as yellowish brown powder which was then separated by filtration and dried under vacuum and subjected to spectral analysis (Bhujbal et al., 2010).

Preliminary phytochemical screening

The ethanol extract was subjected to qualitative chemical investigation for the identification of the phytoconstituents like sterols, triterpenoids, glycosides, alkaloids, carbohydrate and flavonoids (Khandelwal, 2004). The preliminary phytochemical analysis was also carried out using thin layer chromatography (TLC). The TLC analysis was performed on precoated silica gel plates F_{254}, developed with a mixture of $CHCl_3$:glacial CH_3CO_2H:MeOH:H_2O (64:32:12:8) for saponins (Wagner & Bladt, 2007), benzene:EtOAc (7:3) for triterpenoids (Klimek & Tokar, 1998) and EtOAc:HCO_2H:glacial CH_3CO_2H:H_2O (100:11:11:26) for flavonoids. Spots were revealed by the anisaldehyde sulphuric acid reagent for saponins and triterpenoids and UV 365 nm for flavonoids.

Animals

Albino mice weighing 25-30 g were used for the study and were kept in animal house at 26±2 °C with relative humidity 44-56% along with light and dark cycles of 12 h. Institutional Animal Ethics Committee has approved the experimental protocol (DYPIPSR/IAEC/10-11/P-03). Animals were provided with standard diet and water *ad libitum.* The food was withdrawn 18-24 h before the start of the experiment.

Experimental design

Acute toxicity studies (OECD 425)

Healthy adult female Swiss albino mice weighing between 20 and 25 g body weights were selected for the acute toxicity study with the ethanolic extract of leaves of *W. fruticos*a (WF). The animals were fasted overnight prior to the experiment and maintained under standard conditions. The dose of 2000 mg/kg body weight was administered to the animals and were observed for any behavioral changes.

Dexamethasone-induced insulin resistance in mice

All the mice were weighed before treatment, group I (Normal Control) received equivalent amount of 0.9% w/v saline (1 mL/kg, *p.o*.), and thirty mice were rendered hyperglycemic by daily administration of a prestandardised dose of dexamethasone (1 mg/kg, *i.m.*) for consecutive seven days and then divided into five groups of six each (Ghaisas et al., 2009). Group II (Dexa-control) continued to receive only dexamethasone for next fifteen days, group III received pioglitazone (2 mg/kg, *p.o*.) along with dexamethasone respectively for fifteen days. Groups IV, V, VI were treated with dexamethasone along with three different doses of WF 100, 200 and 400 mg/kg, *p.o*. respectively for fifteen days. Simultaneously four other groups (groups VII, VIII, IX, and X), each with six normoglycemic animals, were administered equivalent amount of pioglitazone and three different doses of WF

100, 200 and 400 mg/kg, *p.o*., respectively. On the 0th, 8th, 15th and 22nd day blood samples were collected and treated with anticoagulant ethylene diamine tetra acetic acid (EDTA) and centrifuged to obtain plasma and this plasma was used for the estimation of plasma glucose (GOD/POD Method). Serum triacylglyceride (GPO/POD Method), serum cholesterol, and serum HDL levels were estimated at 22nd day. On the 0th, 8th, 15th and 22nd day body weight of the animals were also recorded. (Ghaisas et al., 2009; Gholap & Kar, 2005).

Biochemical estimations

Plasma concentration of glucose and serum concentration of cholesterol, triacylglyceride and HDL were estimated by using standard diagnostic kits from Biolabs India Ltd., India. LDL, VLDL and atherogenic index were calculated using the following formula:
LDL = Total cholesterol-triacylglyceride/5-HDL
VLDL = Triacylglyceride/5
Atherogenic index = Total cholesterol/HDL cholesterol (Adebayo et al., 2011)

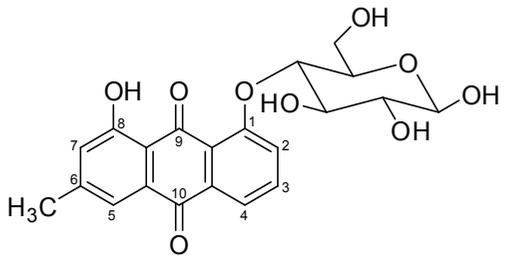
Statistical analysis

The results were expressed as mean±S.E.M. and statistically analyzed by using INSTAT software by ANOVA followed by Dunnett test. $p<0.05$ was considered statistically significant.

Result and Discussion

Ethanolic extract gave positive reaction for anthraquinone, cardiac and saponin glycosides, flavonoids, steroids, triterpenoids, carbohydrates and tannins. The TLC results showed the presence of two spots at R_f 0.43 and 0.46 as green spot and violet spot respectively for saponins, three spots at R_f 0.28, 0.84 and 0.94 as pink, violet and violet spot respectively for triterpenoids and a single spot at R_f 0.52 as yellow fluorescence for flavonoids.

The isolated yellowish brown powder obtained, had a yield of 35%, melting point 220-222 0C. The spectral data showed $UV^{MeO}H$ λ_{max} nm: 272, FT-IR (cm^{-1}) (KBr): 3209,3321, 2939, 1037, 1442, 1512, 1562, 1724, 1H NMR in DMSO-d_6 (300MHz) (δ ppm): 6.79 (1H, d, H-2), 6.4 (1H, t, H-3), 6.86 (1H, d, H-4), 7.16 (1H, s, H-5), 6.25 (1H, s, H-7), 8.76 (phenolic proton), 3.447-4.905 (sugar protons) DART- MS: spectra showed a molecular ion peak at *m/z* 416.17 [M^+] indicating molecular weight of 416. It also showed peaks at 415.19 [M-H+], 253.04 (aglycone), 165.05 (glucose). From the above spectral studies the compound was thus interpreted to be chrysophanol-8-*O*-β-D-glucopyranoside, an anthraquinone glycoside (**1**).



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The acute toxicity study showed that the ethanolic extract of the leaves of *W. fruticosa* (WF) was found to be safe up to dose of 2000 mg/kg. *p.o.* On the basis of this, doses 100, 200 and 400 mg/kg were selected for the study.

The results of the animal studies revealed that in Dexa control group there was significant increase in plasma glucose level ($p<0.01$), serum triacylglyceride level ($p<0.01$), serum total cholesterol ($p<0.01$), serum VLDL ($p<0.01$), serum LDL ($p<0.01$), atherogenic index ($p<0.01$) and significant decrease in serum HDL ($p<0.01$) and body weight ($p<0.01$) when compared to the normal control, this reveals the induction of hyperglycaemia and hyperlipidaemia. The mice treated with Dexa and pioglitazone showed significant decrease in plasma glucose level ($p<0.01$), serum triacylglyceride ($p<0.01$), total cholesterol ($p<0.01$), serum VLDL ($p<0.01$), serum LDL ($p<0.01$), atherogenic index ($p<0.01$) and significant increase in serum HDL ($p<0.01$) and body weight ($p<0.01$). All mice treated with Dexa and WF also showed significant decrease in plasma glucose level ($p<0.01$), serum triacylglyceride ($p<0.01$), total cholesterol ($p<0.01$), serum VLDL ($p<0.01$), serum LDL ($p<0.01$), atherogenic index ($p<0.01$) (Table 1 and 2) and significant increase in serum HDL ($p<0.01$) and body weight ($p<0.01$) (Table 3), thus reveals the ability of *W. fruticosa* to reduce hyperglycaemia and hyperlipidaemia. The administration of WF at the dose of 400 mg/kg showed marginal hypoglycemia ($p<0.05$) when compared to the normal control.

Excess of either endogenous or exogenous glucocorticoids has been shown to increase gluconeogenesis and decrease tissue glucose uptake thus resulting in hyperglycemia, potentially inducing diabetes. Different mechanism for corticosteroid induced diabetes mellitus have been postulated from time to time. One of those is the insulin resistance, caused by the alteration in binding of insulin to its receptor (receptor defect) or by the impairment of the intracellular response to insulin (post receptor defect) (Gholap & Kar, 2005).

Tyrosine phosphatase 1 B has been implicated in insulin dependent pathways and in the insulin insensitivity, that is the most common pathology of type 2 diabetes and obesity. Tyrosin phosphatase 1 B acts to reverse tyrosine kinase action and is a key phosphatase for

insulin receptor and insulin receptor substrate-1, major mediators of glucose transport pathways. Chrysophanol-8-*O*-β-D-glucopyranoside (**1**) is reported to inhibit tyrosine phosphatase 1 B with IC50 values of 18.34±0.29 μM. Thus this reduction activity of tyrosine phosphatase 1 B could influence glucose transport activity through consistent phosphorylation on its specific substrate, the insulin receptor. Thus chrysophanol-8-*O*-β-D-glucopyranoside can be said to enhance insulin stimulated glucose transportation by insulin receptor activation. It has also been reported to prevent hyperglycemia associated diabetes through its mammalian intestinal α-glucosidase inhibitory activity (Lee & Sohn, 2008). The effect of ethanolic extract of *Woodfordia fruticosa* (WF) on blood glucose may be due to chemical constituent such as tannins, terpenoids, saponins and flavonoids reported to have antihyperglycemic effect (Matsudha et al., 2002; Kambouche et al., 2009; Sharma et al., 2008). Various compounds such as hecogenin, lupeol, oleanolic acid, ursolic acid, quercetin have also been isolated from *W. fruticosa* which enhances the importance of this plant.

As expected, in the present study, dexamethasone group showed reduction in body weight, while WF and pioglitazone treatment inhibited dexamethasone induced reduction in body weight and showed increase in body weight. The effect of WF on the body weight may be attributed to the increase in the sensitivity to insulin and the subsequent increase in the glucose uptake. Hyperlipidemia caused in diabetes is due to excess mobilization of fat from the adipose tissue is due to

Table 1. Effect of leaves of *Woodfordia fruticosa* on plasma glucose level in dexamethasone induced insulin resistance in mice.

Groups	Plasma Glucose Level (mg/dL)			
	Day 0	Day 8	Day 15	Day 22
NC	53.71 ± 0.15	53.43 ± 0.28	53.67 ± 0.71	53.56 ± 0.35
Dexa C	54.03 ± 0.15	86.52 ± 0.24##	87.41 ± 0.23##	89.53 ± 0.34##
Dexa+PIO	53.86 ± 0.16	85.98 ± 0.20	69.35 ± 0.19**	56.42 ± 0.15**
Dexa+WF 100	53.74 ± 0.23	87.47 ± 0.13	87.24 ± 0.06	82.58 ± 0.62**
Dexa+WF 200	54.05 ± 0.17	87.18 ± 0.36	85.95 ± 0.35*	75.04 ± 0.99**
Dexa+WF 400	54.02 ± 0.21	87.45 ± 0.30	83.23 ± 0.23**	72.06 ± 1.10**
PIO	53.59 ± 0.11	53.59 ± 0.09	53.53 ± 0.09	53.43 ± 0.08
WF 100	53.38 ± 0.10	53.59 ± 0.15	53.50 ± 0.14	53.25 ± 0.11
WF 200	54.17 ± 0.11	53.77 ± 0.13	53.65 ± 0.14	53.18 ± 0.15
WF 400	54.07 ± 0.25	53.76 ± 0.23	53.51 ± 0.12	52.70 ± 0.14#

Results are presented as mean±SEM. (n=6), ANOVA followed by Dunnett's test; ## (p<0.01) when compared with normal control group; *p<0.05; **p<0.01 when compared with dexamethasone control group. NC: Normal control group, received 0.9% w/v saline (1 mL/kg, *p.o.*/day); Dexa C: Dexamethasone control group, received dexamethasone (1 mg/kg, *i.m.*/day), PIO: Pioglitazone (2 mg/kg, *p.o.*); WF: Ethanolic extract of leaves of *Woodfordia fruticosa* 100, 200, 400 mg/kg *p.o.*

Table 2. Effect of leaves of *Woodfordia fruticosa* on total cholesterol, serum triacylglyceride, serum HDL, serum LDL, serum VLDL, and atherogenic index in dexamethasone induced insulin resistance in mice.

Groups	Total cholesterol (mg/dL)	Serum triacylglyceride (mg/dL)	Serum HDL (mg/dL)	Serum LDL (mg/dL)	Serum VLDL (mg/dL)	Atherogenic index (A.I)
NC	78.10±0.44	80.68±0.15	34.54±1.60	27.41±1.57	16.13±0.03	2.27±0.09
Dexa C	165.45±0.80##	150.71±0.46##	22.56±1.07##	112.74±0.89##	30.13±0.09##	7.40±0.30##
Dexa+PIO	113.13±2.88**	88.14±0.32**	31.06±1.02**	65.90±4.10**	17.62±0.06**	3.90±0.30**
Dexa+WF 100	162.61± 2.22	148.71±0.56	25.85±0.73	107.01±2.42	29.74±0.11	6.31±0.20**
Dexa+WF 200	141.02±1.15**	130.27±0.43**	27.86±1.21*	87.10±1.38**	26.05±0.08**	5.10±0.22**
Dexa+WF 400	121.33±3.05**	109.00±0.94**	32.24±1.01**	67.28±3.04**	21.79±0.18**	3.77±0.12**
PIO	76.93±0.65	80.46±0.66	33.11±1.04	27.72±1.06	16.08±0.13	2.32±0.07
WF 100	77.15±0.05	80.19±0.34	32.73±0.90	28.37±0.94	16.03±0.06	2.36±0.06
WF 200	77.13±1.06	80.07±0.53	34.17±0.36	26.94±0.82	16.01±0.10	2.25±0.02
WF 400	77.10±1.62	81.41±0.31	35.58±0.82	25.23±1.86	16.28±0.06	2.16±0.07

Results are presented as mean±SEM. (n=6); ANOVA followed by Dunnett's test, ##p<0.01 when compared with normal control group; *p<0.05, **p<0.01 when compared with dexamethasone control group. NC: Normal control group, received 0.9% w/v saline (1 mL/kg, *p.o.*/day); Dexa C: Dexamethasone control group, received dexamethasone (1 mg/kg, *i.m.*/day); PIO: Pioglitazone (2 mg/kg, *p.o.*); WF: Ethanolic extract of leaves of *Woodfordia fruticosa* 100, 200, 400 mg/kg *p.o.*

under utilization of glucose. Untreated diabetes causes hypertriglyceridemia and hypercholesteromia with the increase of LDL and VLDL which may lead to various health problems. Treatment with ethanolic extract of *W. fruticosa* (WF) decreased cholesterol, triacylglycerides, LDL, VLDL and increased HDL level, showed the effectiveness of WF to treat hyperlipidemia (Sen et al., 2010).

In conclusion, the ethanolic extract of the leaves of *W. fruticosa* at the dose of 100, 200 and 400 mg/kg was found to be effective in dexamethasone induced insulin resistance in mice and plant can be a future effective medicine for the treatment of diabetes, thus supports the traditional claim of the plant in treatment of diabetes. Further studies are infact underway to confirm the exact mechanism of action and isolation of other phytoconstitutents responsible for such activity.

Table 3. Effect of leaves of *Woodfordia fruticosa* on % change in body weight in dexamethasone induced insulin resistance in mice.

Groups	% Change in body weight		
	At day 8	At day 15	At day 22
NC	0.72±0.72	1.44±0.91	1.44±0.91
Dexa C	-9.62±0.93	-17.13±0.63$^{\#\#}$	-23.18±1.42$^{\#\#}$
Dexa+PIO	-8.76±0.11	-2.17±0.97**	0.75±0.75**
Dexa+WF 100	-5.61±2.97	-5.61±2.97**	-1.99±2.83**
Dexa+WF 200	-8.77± 0.17	-6.62±1.04**	-2.23±1.00**
Dexa+WF 400	-11.11±2.93	-6.85±3.00**	-2.59±3.07**
PIO	1.41±0.89	0.72±0.72	0.72±0.72
WF 100	2.20±0.98	2.20±0.98	2.20±0.98
WF 200	2.23±1.00	2.23±1.00	1.51±1.47
WF 400	1.38±0.87	0.72±1.25	-1.30±1.70

Results are presented as mean±SEM; n=6; ANOVA followed by Dunnett's test, $^{\#\#}p<0.01$ when compared with normal control group; $*p<0.05$; $**p<0.01$ when compared with dexamethasone control group. NC: Normal control group received 0.9% w/v saline (1 mL/kg, *p.o.*/day); Dexa C: Dexamethasone control group, received dexamethasone (1 mg/kg, *i.m.*/day); PIO: Pioglitazone (2 mg/kg, *p.o.*); WF: Ethanolic extract of leaves of *Woodfordia fruticosa* 100, 200, 400 mg/kg *p.o.*

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