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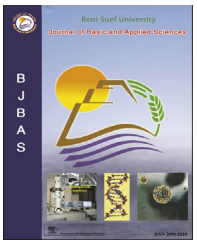

Full Length Article

Antidiabetic effect of *Chloroxylon swietenia* bark extracts on streptozotocin induced diabetic rats

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ABSTRACT

Diabetes has been increasing at an alarming rate around the world, and experts have relied on remedies from the utilization of ancient drugs that are essentially derived from plants. The present study aimed to evaluate the antidiabetic potential of *Chloroxylon swietenia* bark extracts on streptozotocin induced diabetic rats. Diabetes was induced in male albino Wistar rats by single intraperitoneal injection of streptozotocin (STZ) (50 mg/kg b.w.). The diabetic rats were administered orally with *C. swietenia* bark (CSB) methanolic (CSBMEt) and aqueous (CSBAEt) (250 mg/kg b.w.) extracts and glibenclamide (600 µg/kg b.w.) by intragastric intubation for 45 days. The result showed a heavy loss in weight, increase in blood glucose and glycosylated hemoglobin level, and decline in plasma insulin and total hemoglobin content. Furthermore, glucose-6-phosphatase and fructose-1,6-bis phosphatase were found to be increased whereas hexokinase and glycogen contents were decreased in STZ induced diabetic rats. CSBAEt, CSBMEt and glibenclamide treated diabetic rats showed moderate reduction in blood glucose and glycosylated hemoglobin levels; in addition, plasma insulin and hemoglobin levels were elevated. The altered activities of carbohydrate metabolizing enzymes and liver glycogen were improved remarkably. CSBMEt results were comparable to the standard drug glibenclamide. The present findings support the usage of the plant extracts for the traditional treatment of diabetes.

1. Introduction

Diabetes mellitus (DM) could be a bunch of metabolic disorders portrayed via hyperglycemia following imperfections in insulin emission, insulin activity, or both. The chronic hyperglycemia is identified with stretched pathology and damage, which distress multiple organs. It additionally incorporates a bigger chance of getting dyslipidemia, high blood pressure, and obesity (American Diabetes Association, 2011). The pathogenesis of insulin-dependent DM includes ecological reasons that may initiate immune system mechanisms on hereditarily defenseless people, prompting dynamic loss of pancreatic islet β-cell resulting in insulin deficiency (Harrison and Honeyman,

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1999; Gurudeeban et al., 2012). Non-insulin-dependent DM is related to impaired insulin secretion, obesity, insulin resistance, and hereditary disposition in individuals over 40 years of age (Zimmet et al., 1990). Streptozotocin could be an intense poison to the islets of Langerhans and causes extreme diabetes (Brenna et al., 2003). This condition is characterized by a noteworthy increment in serum glucose levels and a huge lessening in insulin discharge (Nabila, 2010). The surplus glucose within blood responds to hemoglobin (Hb) and frame glycosylated hemoglobin (HbA1c) (Koenig et al., 1976). Prior reports have demonstrated that insulin boosts property of glibenclamide and has thereby been accepted as qualified antidiabetic medication (Andrade Cetto et al., 2000).

The therapeutic management of polygenic disorder without feature impacts remains disputed. In light of the developing enthusiasm on assessing homegrown cures, these are seen to be less harmful and to possess insignificant feature impacts (Gupta et al., 2012). East Indian satinwood grows in dry deciduous forest, and is indigenous to Asian countries. It has been employed as a part of chimerical medicine (Venkataswam et al., 2010). This plant is conventionally used for cuts, burns, wounds, rheumatism, optical infection, snakebites, etc. (Kiran et al., 2008). Various extracts of *Chloroxylon swietenia* have been reported to possess antimicrobial activity (Vinatha and Estari, 2013), antibacterial and antihelminthic (Ranjith et al., 2011) hepatoprotective, antioxidant (Palani et al., 2010), larvicidal (Kiran et al., 2006), anti-inflammatory (Kumar et al., 2006), and analgesic (Senthilraja and Ramkumar, 2003) properties, and root bark consumed along with milk have been reported to treat impotence (Parrotta, 2001). Investigation of lipid profile and ocular oxidative stress of *C. swietenia* on Streptozotocin-nicotinamide-induced diabetic rats (Patchimatla et al., 2014) was studied in aerial parts. However STZ induced animal model on *C. swietenia* bark (CSB) extracts was not yet studied. The current study was intended to explore the antidiabetic activity of aqueous (CSBAEt) and methanolic extracts (CSBMEt) of *C. swietenia* bark (CSB) on STZ induced diabetic rats.

2. Materials and methods

2.1. Chemicals

STZ and glibenclamide was obtained from Sigma-Aldrich Company (Bangalore, India). The other experimental chemicals used were of analytical grade and were purchased from HiMedia (Mumbai, India).

2.2. Plant material collection, processing, and preparation of extracts

CSB placed in rue family was gathered amid December from Kalvarayan Hills Kallakurichi, Tamil Nadu, India. Dr. V. Chelladhurai (Research Officer – Botany Central Council for Research in Ayurveda and Siddha, Govt. of India) has given the taxonomic distinguishing proof. It was shade dried for a month, granulated in mechanical grinder and stuffed within airproof pack. CSB (300 g) was extracted with methanol (1 L) by soxhlet apparatus (72 h), and the mixture was dried (45 °C) in a rotary evaporator (Heidolph, Germany). The pounded bark (300 g) was soaked (3 days) in distilled water (1 L) in ambient temperature, subjected to periodic agitation and set aside using cotton attachment. Afterward, the bark was taken out with the use of Whatman filter paper (no. 1) and dehydrated at ambient temperature. The dried extracts were stored at 4 °C until further use.

2.3. Experimental animals

Grown-up adult male albino Wistar rats with body weight (b.w.) above 180 g at 8 to 10 weeks from conception were acquired from Madhavaram Veterinary Medical College, Chennai, Tamil Nadu, India. They have been housed at poly propylene confines and kept up in standard environment [12 h light and 12 h dark cycle, (25 ± 3)°C]. The rats were fed with standard rat pellet diet (Pranav Agro Industry Ltd, Maharashtra) and given water ad libitum and maintained at Central Animal House, RMMCH. All studies were conducted as per the National Institute of Health's Guide for the Care and Use of Laboratory Animals, and the study was endorsed by the Institutional Animal Ethical Committee of Rajah Muthiah Medical College and Hospital (Proposal No. 998, Reg. No. 160/1999/CPCSEA), Annamalai University, Tamil Nadu, India. Animals were adapted for 3 days in the research laboratory before start of the experiments.

2.4. Experimental induction of diabetes

Diabetes was prompted through single intraperitoneal injection of freshly prepared streptozotocin (STZ) (50 mg/ kg b.w.) in 0.1 M citrate buffer (pH = 4.5) to overnight starved rats (Gupta and Gupta, 2009). Diabetic rats were permitted to drink 20% glucose solution overnight to overcome the initial drug induced hypoglycemic death. The blood glucose level was measured after three days, and rats with glucose levels >250 mg/dL were considered as diabetic. At the time of induction, control rats were injected with 0.2 mL of vehicle (0.1 M citrate buffer, pH 4.5) alone.

2.5. Experimental design

In this experiment 30 rats (6 normal and 24 STZ diabetic existing rats) were used. They were separated into five groups of 6 rats each. The CSBMEt were dissolved in 2% CMC (Carboxyl methyl cellulose) in distilled water (Kumar et al., 2011), CSBAEt and glibenclamide 0.5 mL of 0.9% saline and administered orally (45 days).

Group I. Control rats (were given 0.5 mL of 0.9% saline orally for 45 days).
Group II. Diabetic group (STZ 50 mg/kg b.w.).
Group III. Diabetic rats were given CSBAEt (250 mg/kg b.w. dissolved in 0.5 mL of 0.9% saline) orally for 45 days.
Group IV. Diabetic rats were given CSBMEt (250 mg/kg b.w. dissolved in 0.5 mL of CMC) for 45 days.
Group V. Diabetic rats were given Glibenclamide (600 µg/kg b.w. dissolved in 0.5 mL of 0.9% of saline) for 45 days (Subash et al., 2007).

Toward the study's end (45 days), the animals were euthanized by ketamine (24 mg/kg/body) intramuscular injection and

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