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Evaluation of Diabeat, A Polyherbal Formulation in Streptozotocin-Induced Diabetic Rats: Implication of Hepatic Insulin Signaling

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ABSTRACT

Background and Aim: Type 2 diabetes mellitus (T2DM) is a progressive polygenic disorder requiring a multi-targeted therapeutic approach. In T2DM, hyperglycemia impairs insulin signaling by disrupting glucose and lipid metabolism, resulting in insulin resistance and associated complications. In the recent scenario, the use of polyherbal formulation is increasing for the management and treatment of T2DM. Diabeat, a polyherbal formulation (DPHF) is comprised of four herbs, widely used for their anti-diabetic, anti-oxidant, and hepatoprotective activities. In the present study, *in-vitro* and *in-vivo* experiments were carried out to ascertain the plausible mechanisms underlying the anti-diabetic effects of DPHF.

Methods: DPHF was evaluated for residual toxins as per Ayurvedic Pharmacopeia of India (API) procedures. The effect of DPHF extract on carbohydrate digestive enzymes α-amylase and α-glucosidase was studied *in-vitro*. T2DM was developed in Wistar rats using nicotinamide and streptozotocin (120 and 55 mg/kg, i.p. single dose, respectively). Diabetic rats were treated with DPHF (100, 200, and 300 mg/kg p.o. respectively) for 12 weeks. After 24 hours of the last dose, animals were sacrificed and glycaemic, biochemical, histopathological, ultrastructural changes were measured. The real-time polymerization chain reaction was used to measure mRNA expression of genes involved in hepatic insulin signaling including insulin receptor substrate (IRS), phosphoinositide-3-phosphate kinase (PI3K), Akt, and glucose transporter 2 (GLUT2), inflammation such as TNF-α and IL-6 and AMP-activated protein kinase (AMPK).

Results: Residual toxins present in DPHF were within the API limits. DPHF inhibited the α-amylase and α-glucosidase activity *in-vitro*. Further, diabetic rats demonstrated a significant change in the glycaemic, biochemical, and antioxidant parameters. H&E staining and ultrastructural study of hepatic tissues also indicated significant liver damage. DPHF treatment significantly restored the glycaemic state, antioxidant status, lipid profile, hepatic architecture, and ultrastructural changes in the diabetic rats. Similarly, diabetic rats have shown detrimental effects on AMPK/IRS/PI3K/Akt/GLUT2 which were attenuated by DPHF treatment.

Conclusion: DPHF demonstrated marked anti-diabetic, anti-hyperlipidemic, antioxidant, anti-inflammatory, and hepatoprotective effects in diabetic rats. DPHF ameliorated glucose-lipid homeostasis by improving insulin sensitivity possibly through alleviating AMPK mediated hepatic insulin signaling.

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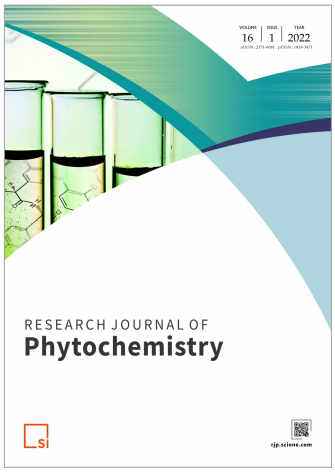

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