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Evaluation of pancreatic regeneration activity of *Tephrosia purpurea* leaves in rats with streptozotocin-induced diabetes

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ABSTRACT

Background and aim: Flavonoid rich plant *Tephrosia purpurea* (*T. purpurea*), commonly known as Sarpunkha has been used in traditional systems of medicine to treat diabetes mellitus. However, its effectiveness in promoting regeneration of pancreas in diabetes has not been investigated. Therefore, the present study was undertaken to evaluate pancreatic β-cells regeneration, antioxidant and antihyperlipidemic potentials of *T. purpurea* leaves extract, its fractions and main constituent Rutin in diabetic rats.

Experimental procedure: The leaves extract and its fractions were first screened for acute and sub-chronic antidiabetic activity in a dose range of 250–500 mg/kg orally. Further, fractions with potent antidiabetic activity were screened for pancreatic β-cells regeneration activity using histopathological studies and morphometric analysis, which was followed by estimation of biochemical parameters.

Results and conclusion: The most significant antidiabetic, pancreatic regeneration and antihyperlipidemic activity was exhibited by *n*-butanol soluble fraction of ethanol extract at the dose level of 500 mg/kg. Histopathology revealed that treatment with this fraction improved the β-cell granulation of islets and prevented the β-cells damage which was further confirmed by morphometric analysis. Thus, the present study validated the traditional use of *T. purpurea* plant in the treatment of diabetes, which might be attributed to pancreatic β-cells regeneration potential of its active constituent Rutin.

Taxonomy (classification by EVISE): Traditional Medicine; Metabolic Disorder; Experimental Design; Cell Regeneration and Histopathology.

1. Introduction

Diabetes mellitus (DM) is a multifactorial chronic disorder of carbohydrate, protein and lipid metabolisms. It is characterized by persistent hyperglycemia, hypercholesterolemia and hypertriglyceridemia. Chronic hyperglycemia results in micro-vascular complications to the organs like eyes and kidneys, lower-limb amputations, and increased risk of cardiovascular diseases such as hypertension and atherosclerosis, which contribute to diabetes-associated morbidity and mortality.[1–3] Defects in insulin gene expression in the islets of β cell and consequent decrease in insulin secretion are the major causes of glucose toxicity. Decreased levels of insulin gene transcription stimulatory proteins such as pancreas homeobox protein 1 (PDX-1) and musculoaponeurotic fibrosarcoma oncogene homolog A (MafA) are responsible for declined insulin gene expression.[4] The hyperglycemia is controlled by multiple injections of insulin in type I diabetic patients, while type II diabetes is controlled by administration of oral hypoglycemic agents. Currently available treatments are expensive and have serious adverse effects.[5] Likewise, commonly prescribed

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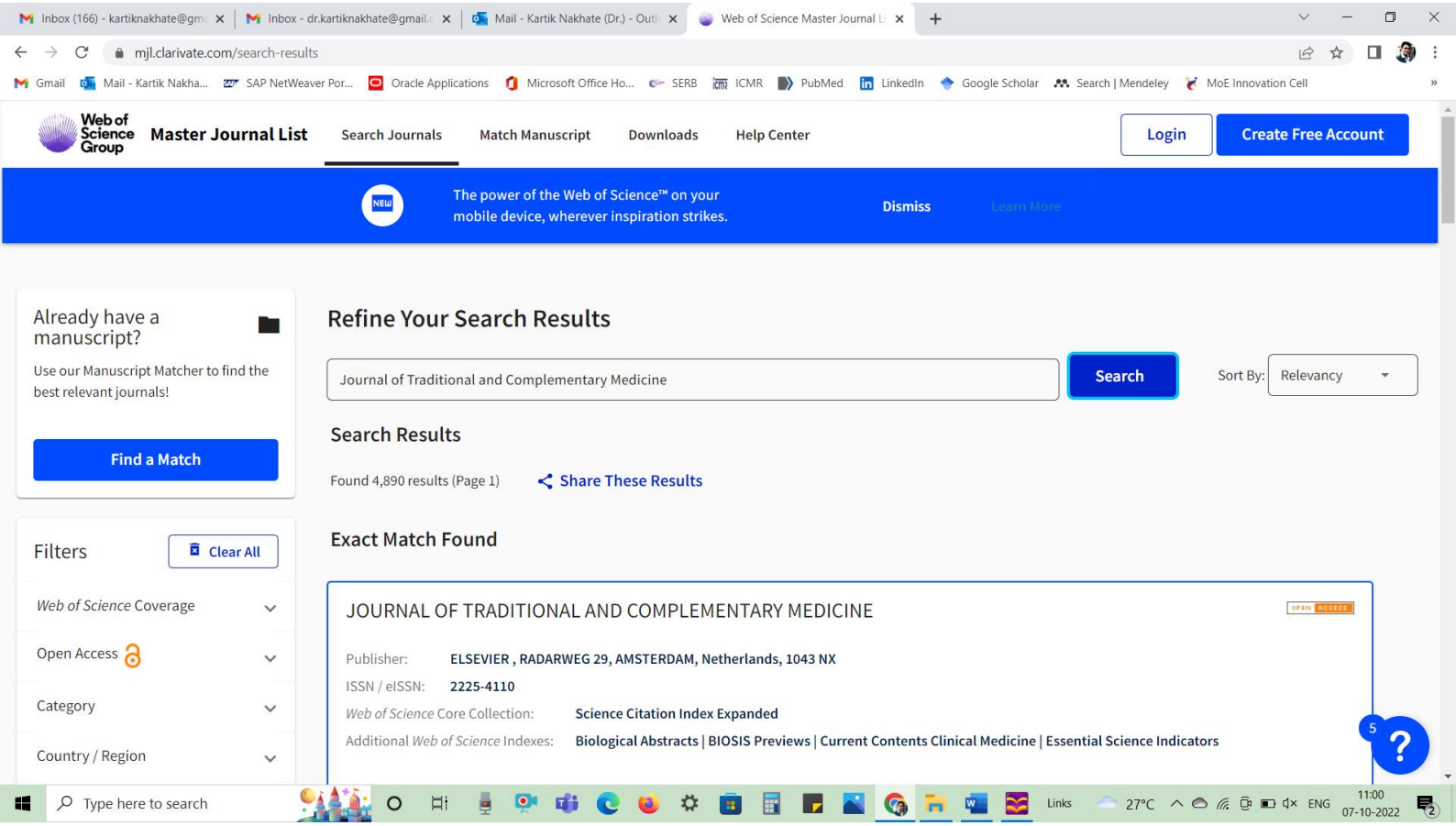
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