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Curcumin Protects Diabetic Mice against Isoproterenol-Induced Myocardial Infarction by Modulating CB2 Cannabinoid Receptors

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Abstract: Molecular docking revealed curcumin as a potent CB2 cannabinoid receptor (CB2R) agonist. Since CB2R is involved in cardioprotective functions, we explored its role in ameliorative actions of curcumin against myocardial damage triggered by isoproterenol in diabetic animals. Mice were kept on a high-fat diet (HFD) throughout the experiment (30 days). Following 7 days of HFD feeding, streptozotocin was administered (150 mg/kg, intraperitoneal) to induce diabetes. From day 11 to 30, diabetic mice received either curcumin (100 or 200 mg/kg/day, oral), CB2R antagonist AM630 (1 mg/kg/day, intraperitoneal) or both, with concurrent isoproterenol (150 mg/kg, subcutaneous) administration on day 28 and 29. Diabetic mice with myocardial infarction showed an altered hemodynamic pattern and lipid profile, reduced injury markers, antioxidants with increased lipid peroxidation in the myocardium, and elevated glucose and liver enzymes in the blood. Moreover, an increased pro-inflammatory markers, histological severity, myonecrosis, and edema were observed. Curcumin compensated for hemodynamic fluctuations, restored biochemical markers, preserved antioxidant capacity, decreased cytokines levels, and restored cardiac functionality. However, the AM630 pre-treatment attenuated the effects of curcumin. The data suggest the involvement of CB2R in the actions of curcumin such as in the prevention of myocardial stress and in the improvement of the normal status of the myocardial membrane associated with diabetes.

Keywords: curcumin; CB2 receptor; myocardial infarction; oxidative stress; diabetes

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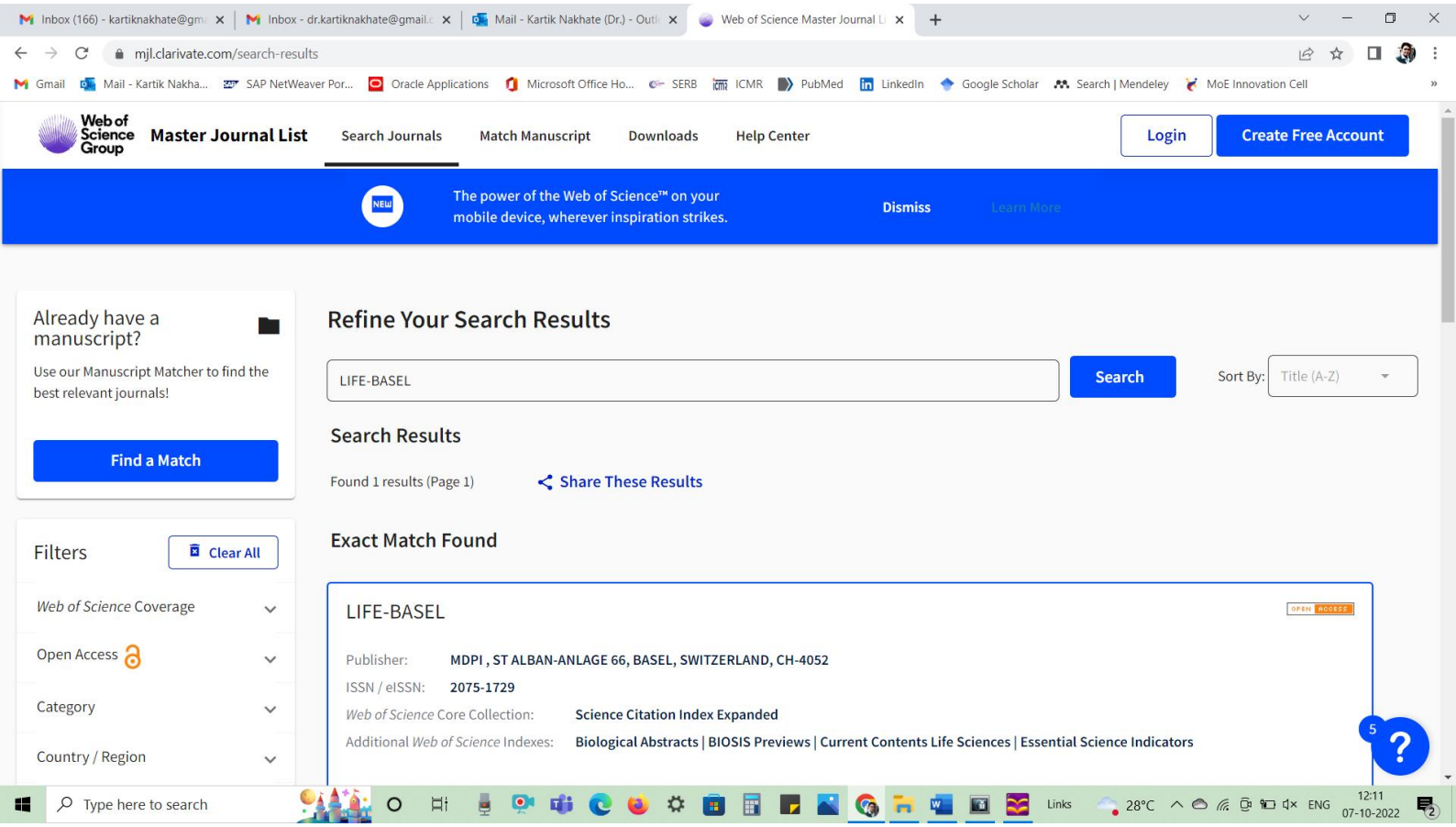
1. Introduction

The risk of cardiovascular morbidity and mortality in diabetic patients is 2- to 4-fold higher than non-diabetic persons. [1]. Indeed, in patients of diabetes mellitus, myocardial infarction (MI) is the foremost cause of mortality. The incidence of MI in diabetic conditions increases depending on the number of comorbidities such as hyperglycemia, dyslipidemia, oxidative stress, and inflammatory conditions [2]. Interestingly, the pathogenesis of diabetes and MI involves the secretion of inflammatory mediators and the excessive formation of reactive oxygen species (ROS) [3,4]. In cardiac cells, oxidative stress is exacerbated by chronic hyperglycemia which is associated with diabetes, and intensifies the depletion of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