Mol Cell Biochem
DOI 10.1007/s11010-013-1893-2

Effects of *Trigonella foenum-graecum* (L.) on retinal oxidative stress, and proinflammatory and angiogenic molecular biomarkers in streptozotocin-induced diabetic rats

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Received: 27 August 2013 / Accepted: 5 November 2013

Abstract The aim of the present study was to investigate the protective effects of *Trigonella foenum-graecum* Linn. (fenugreek) in Streptozotocin-induced diabetic rat retina. Fenugreek (100 and 200 mg/kg body weights) treatment was carried out for 24 weeks and evaluated for inflammatory [tumor necrosis factor (TNF)-α and interleukin (IL)-1β] and angiogenic [vascular endothelial growth factor (VEGF) and protein kinase C (PKC)-β] molecular biomarkers. Retinal oxidative stress was evaluated by estimating antioxidant (Glutathione, Superoxide dismutase, and Catalase) parameters. Fluorescein angiography was performed to detect retinal vascular leakage. Electron microscopy was performed to determine basement membrane thickness. In the present study, significant rises in the expressions of retinal inflammatory (TNF-α and IL-1β) and angiogenic (VEGF and PKC-β) molecular biomarkers were observed in diabetic retinae compared with normal retinae. However, fenugreek-treated retinae showed marked inhibition in the expression of inflammatory and angiogenic molecular biomarkers. Moreover, results from the present study showed positive modulatory effects of fenugreek on retinal oxidative stress. Fluorescein angiograms and fundus photographs obtained from diabetic retinae showed retinal vascular leakage. On the other hand, fenugreek-treated retinae did not show vascular leakage. Further, thickened BM was recorded in diabetic retina compared with normal retinae. However, fenugreek-treated retinae showed relatively lesser thickening of capillary BM. In conclusion, it may be postulated that fenugreek has great potential in preventing diabetes-induced retinal degeneration in humans after regular consumption in the specified dosage.

Keywords Fenugreek · Blood retinal barrier · Diabetic retinopathy · Basement membrane · Antiangiogenic · Anti-inflammatory

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Introduction

Diabetes is a disorder of chronic uncontrolled hyperglycemic state. Hyperglycemic state leads to generation of reactive oxygen species (ROS) through various mechanisms, leading to a condition known as oxidative stress. Oxidative stress is known to play important role in pathogenesis of diabetic retinopathy (DR). During DR, there is a state of crisis at cellular level due to decreased levels of antioxidant enzyme stores, resulting in insufficient neutralization of ROS [1–4]. This leads to activation of various inflammatory pathways, leading to increased levels of major inflammatory cytokines [tumor necrosis factor-α (TNF-α) and Interleukin-1β (IL-1β)] in serum and retinae of diabetic humans and animals [5–7]. Cytokines are key mediators in the pathogenesis of DR and activation of various apoptotic pathways in diabetic retinae. TNF-α and IL-1β have important roles in basement membrane (BM)

Published online: 17 November 2013