In the flesh scoparia dulcis has a protective effect on the liver, kidney, and brain.

Hasan Abosbah*

Department of Chemistry, Faculty of Science, Thaksin University, Phatthalung Campus, Pa Phayom, Phatthalung, 93210 Thailand

Abstract

The researchers wanted to see how an aqueous extract of Scoparia dulcis affected the occurrence of oxidative stress in the brains of diabetic rats by evaluating the level of oxidative damage and the antioxidant defence system. The effect of an aqueous extract of the Scoparia dulcis plant on blood glucose, plasma insulin, and the levels of thiobarbituric acid reactive substances (TBARS), hydroperoxides, superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione-S-transferase (GST), and reduced glutathione (GSH) in diabetic rats induced with the standard reference medication was glibenclamide. Plasma insulin, superoxide dismutase, catalase, glutathione peroxidase, glutathione-S-transferase, and reduced glutathione activity all increased significantly in brain on treatment with body weight of Scoparia dulcis plant extract (SPEt) and glibenclamide. Both treated groups had significantly lower levels of TBARS and hydroperoxides in the brain, implying that it may have a role in protecting against lipid peroxidation-induced membrane damage. These findings point to a putative antiperoxidative role for Scoparia dulcis plant extract, as activation of antioxidant enzymes is regarded a trustworthy marker for evaluating the antiperoxidative performance of medicinal plants. Scoparia dulcis, in addition to having an anti-diabetic effect, also has antioxidant properties that could be used for therapeutic purposes.

Keywords: Acetylcholinesterase, brain, erythrocytes, lipid peroxidation, Scoparia dulcis.

Introduction

Diabetes mellitus' neurological effects in the Central Nervous System (CNS) are increasingly garnering more attention. The neurological problems of diabetes are not restricted to peripheral neuropathies, as evidenced by cognitive deficits as well as morphological and neurochemical changes. Hyperglycemia's central problems include the potentiation of neuronal damage seen after hypoxic/ischemic events, as well as stroke. In diabetes, glucose consumption in the brain is reduced, suggesting a possible reason for greater vulnerability to acute pathogenic events [1].

In diabetes, as well as stress in euglycemic animals, oxidative stress, which leads to an increase in the generation of reactive oxygen species (ROS) and lipidperoxidation, is enhanced [2]. Similarly, experimentally induced hyperglycemia increases oxidative damage in the rat brain. Hyperglycemia greatly promotes neuronal changes and glial cell damage produced by transient ischaemia in experimental circumstances [3]. Several lines of evidence suggest that the altered oxidative state generated by chronic hyperglycemia may contribute to neurological tissue damage: free radical species wreak havoc on the central nervous system, damaging neurons and Schwann cells, as well as peripheral nerves [4]. Schwann cells and axons are particularly vulnerable to oxygen free radical damage because to their high polyunsaturated lipid content: Cell membrane stiffness may be increased by lipidperoxidation, which may impede cell function.

In the serum of Type 1 diabetes patients, there was an increase in superoxide generation, which was reduced with better glycemic control. In the brains of Type 1 diabetes rats and Type 2 diabetic mice, lipidperoxidation products are likewise elevated. Increases in oxidative stress and antioxidant activity caused by diabetes and stress may render the brain more sensitive to later pathological occurrences [5].

Conclusion

During diabetes, the brain undergoes various structural and functional changes. Some of these changes may be influenced by oxidative stress, a component implicated in the pathophysiology of diabetic problems. Treatment of diabetic rats with Scoparia dulcis plant extract reduced lipid peroxidation and enhanced antioxidant status considerably. These findings imply that Scoparia dulcis plant extract may have an antioxidant role in addition to its antidiabetic impact,

***Correspondence to:** Mohammad Aburumman, Microbial Chemistry Department, National Research Centre, Tahrir St., Dokki, Cairo, Egypt, E-mail: aburumman.mohammad@edu.eg

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