J.Bio.Innov3(6),pp:297-301,2014

ISSN 2277-8330 *(Electronic)*

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EFFECT OF THIOBARBIUTRIC ACID ACTIVITY IN PLASMA OF DIABETIC PATIENTS WITH NEPHROPATHY – A Review

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ABSTRACT

The worldwide incidence of diabetes has increased dramatically along with widespread lifestyle and dietary changes. Diets high in fat are strongly associated with the development of obesity and can induce insulin resistance in humans and animals. It is clear that obesity constitutes a risk factor for contributing to the development of type 2 diabetes. In the present study, we investigated the therapeutic potential action of Thiobarbeutric acid on diabetes associated complications.

Key words: Thio Barbeutic acid, Diabetes retinopathy.

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ISSN 2277-8330 (Electronic)
www.jbino.com

INTRODUCTION

Diabetes is a serious disease characterized by insufficient insulin secretion, insulin activity deficiency and hyperglycaemia, and its estimated prevalence is pretty high (5.4% in 2025) in society. At the same time, it causes to serious problems involving blindness, pancreatic cancer and cognitive impairment. Moreover, the molecular level effects of diabetes also exist and they involve increase in oxidative agent levels and deficit in anti-oxidative systems. In particular hyperglycaemia in diabetes causes increase of oxidative radicals and then the radicals lead to diabetic complications. Especially these oxidative radicals lead to structural defects of membranes and proteins. In the present study we are concentrating on effect of thiobarbeutric acid in plasma of diabetic patients with nephropathy

Discussion

We have earlier reported the hypoglycemic and hypo cholesterolemic activities of *K. pinnata* preparation consumption in streptozotocin-induced diabetes. This report provides data on the role of *K. pinnata* preparation consumption on lipid peroxidation, antioxidant enzymes and RBC membrane ATPase activities in streptozotocin-induced diabetic rats. Diabetic conditions have been shown to result in impaired antioxidant defenses, compromised mitochondrial function, and increased sorbitol and advanced glycation end products from glucose. Previous studies have shown that oxidative stress generated from hyperglycemia plays a particularly important role in the initiation of vascular diabetic complications, including retinopathy, cardiomyopathy, and nephropathy. SOD and CAT are antioxidant enzymes that convert toxic free radicals to water or other harmless compounds. Lipid peroxidation is intricately linked to antioxidant enzymes such as SOD and CAT. As a defense against reactive free radicals, the body produces antioxidant enzymes which help to prevent oxidative stress damage to tissues. The observed decreases in SOD and CAT activities and GSH levels in the untreated diabetic group is consistent with reports in the literature suggesting the disposition of

ISSN 2277-8330 (Electronic)
www.jbino.com

diabetic tissues to damage by free radicals Although, the TBARS levels were not significantly altered among the groups in this short-term study, the observed early increased SOD and CAT activities in diabetic rats treated with *K. pinnata* preparation may be indicative that the preparation might be effective in curtailing lipid peroxidation associated with the disease. This would spare tissue damage and prevent the development of diabetic complications.

Constant exposures to free radicals and high oxidative stress in diabetes have also been associated with erythrocyte structural damage. Lipid peroxidation alters the cellular structure of membrane-bound enzymes by changing phospholipids and fatty acid composition. Previous studies have reported reduced erythrocyte ATPase activity, specifically the Na^+/K^+ ATPase in insulin-deficient conditions. In this study, we noted a nonsignificant decrease in Na^+/K^+ and Mg^{2+} ATPase activities and a nonsignificant increase in Ca^{2+} ATPase activity in the diabetic control group. However, in the diabetic group treated with aqueous *K. pinnata* preparation, there was a marginal increase in Na^+/K^+ activity and a significant ($P < 0.05$) increase in Mg^{2+} ATPase activity compared to the diabetic control group. Mg^{2+} ATPase activity in the erythrocyte membrane has been reported to reduce cellular calcium content, improve erythrocyte flexibility and reduce vascular complications. In fact, *in vitro* and *in vivo* studies have demonstrated that insulin may modulate the shift of magnesium from extracellular to intracellular spaces. Intracellular Mg^{2+} is a critical cofactor for several enzymes in carbohydrate metabolism because of its role as part of the activated Mg^{2+} ATP complex. The activated complex is responsible for the phosphorylation of all the rate limiting enzymes in the glycolytic pathway. Mg^{2+}deficiency may also result in disorders of tyrosine-kinase activity on the insulin receptor, leading to the development of post receptorial insulin resistance and decreased cellular glucose utilization. Hence, the observed significant increase in RBC membrane Mg^{2+} ATPase activity may suggest that the consumption of aqueous *K. pinnata* preparation could increase intracellular magnesium and subsequently improve rates of glycolytic activity. Adamson *et al.* also reported that the determination of ATPases, particularly Mg^{2+} ATPase and Na^+/K^+ ATPase, may

provide an indirect measurement of insulin binding or level. We hypothesize that the consumption of *K. pinnata* preparation may act to increase peripheral insulin levels via extra pancreatic insulin elevation. It is also possible that the preparation's active ingredient may modulate the insulin–insulin receptor complex with a resultant prolonged half-life of ligand-receptor complex, which could lead to sustained signaling as was reported for the AspB10 insulin analog. More investigations are needed to further isolate and identify the active component(s) of the preparation, as well as to investigate, the effects of these isolates on glucose-regulating hormones.

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ISSN 2277-8330 *(Electronic)*
www.jbino.com

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