Short communication

iMedPub Journals
www.imedpub.com

Biochemistry & Molecular Biology Journal
ISSN 2471-8084

2021
Vol.7 No.7:33

Antioxidant Supplementation and Kidney Function Status of Wistar Rats after Type 2 Diabetes Induced by High Fat Diet-Streptozotocin (HFT-STZ)

Alekhya Thirunahari*

Department of Biotechnology, Osmania University, Hyderabad, Telangana, India

***Corresponding author:**
Alekhya Thirunahari

thirunaharialekhya151315@gmail.com

Department of Biotechnology, Osmania University, Telangana, India.

Tel: 551832291000

Citation: Thirunahari A (2021) Antioxidant Supplementation and Kidney Function Status of Wistar Rats after Type 2 Diabetes Induced by High Fat Diet-Streptozotocin (HFT-STZ). Biochem Mol Biol Vol.7 No.7:33

Received: July 05, 2021; **Accepted:** July 15, 2021; **Published:** July 20, 2021

Short Communication

In vitro, the kidney function of albino rats administered antioxidant supplementation were examined in high fat diet-streptozotocin (HFD-STZ) induced NIDDM. Minerals, vitamins, -lipoic acid, phytochemicals, and a D-ribose-Lcysteine conjugate, in appropriate and suggested dietary permitted quantities, were combined in maize oil and kept at 4°C for use. Standard procedures, kits, and equipment were used to measure kidney function indices.

SPSS version 20.0 was used to analyse the data, and the significance threshold was set at p=0.05. There were five different study groups, each with ten rats. Treatment began immediately after induction of diabetes with the HFD-STZ combination, and lasted for a total of 12 weeks, with serum analysis performed at the 4^{th}, 8^{th}, and 12^{th} weeks of the study. The data was analysed using SPSS version 20.0, with a significance level of p=0.05. There were ten rats in each of the five study groups. Treatment commenced as soon as the HFD-STZ combination was used to induce diabetes and lasted for a total of 12 weeks, with serum analysis taking place at the 4^{th}, 8^{th}, and 12^{th} weeks of the trial [1].

When compared to the diabetic control group, serum creatinine, biocarbonate, and potassium levels did not differ statistically (p=0.05) when compared to the diabetic control group, which increased steadily for creatinine and biocarbonate but inconsistently for potassium level over the treatment duration. However, when the treated and normal control groups were compared to the diabetic control group, there was a substantial rise (p=-0.05) in serum sodium and chloride levels [2].

The observed increase was proportional to the duration of treatment. As a result, the antioxidant supplement appears to improve optimal electrolyte balance and management for easy ion passage across cell membranes, as well as boosting kidney function. Diabetic kidney disease is caused by diabetes mellitus, a metabolic condition characterised by a relative or absolute lack of insulin secretion (DKD). Diabetic kidney disease development is linked to complications such as retinopathy, neuropathy, hepatopathy, nephropathy, and cardiomyopathy, and is one of the most serious sequelae of diabetes mellitus. They are the leading cause of morbidity and mortality in the globe. Hyperglycemia-induced oxidative stress has been linked to diabetic problems such as endothelial dysfunction, insulin resistance, and changes in the proportion and functions of pancreatic -cells, which eventually leads to diabetic microvascular and macrovascular issues [3].

As a result of free radical formation, glucose oxidation, protein glycosylation, and lipid peroxidation occur, resulting in an increase in reactive oxygen species (ROS). ROS is involved in cell signalling in several cell types, including renal cells, and is implicated in proliferation, differentiation, apoptosis, and immune response under normal settings. Overproduction of ROS in the kidney under pathological settings, on the other hand, has been linked to renal inflammation, which can alter renal structure and function, eventually leading to end-stage renal disease (ESRD). Chronic renal failure caused by diabetes is becoming more widely recognised as a major health hazard.

Reduced antioxidant activity, on the other hand, can be linked to oxidative stress caused by free radical production and reactive oxygen species. Both endogenous and exogenous antioxidants interact with these oxidants in physiological settings to protect cells from oxidative damage. Superoxide dismutase (SOD), manganese SOD, and copper/zinc SOD; glutathione system: glutathione peroxidase and glutathione reductase; catalase; and coenzyme Q are some of the antioxidant defence systems. Antioxidant enzymes primarily convert reactive oxygen species (ROS) into nonreactive oxygen molecules, resulting in the formation of water. NADPH, which is primarily produced by

glucose-6-phosphate dehydrogenase, is used as a chemical reductant throughout the antioxidant redox system. It is critical to study and apply crucial and important antioxidants such as glutathione, vitamin A, C, and E in order to identify modalities for better treatment of diabetic kidney disease and other related issues. The composition, synthesis, and role of glutathione in the understanding of diabetes-induced oxidative stress and its complications via various signalling pathways, as well as ROS formation attributed to the activation of various downstream signalling cascades affecting structural and functional changes in the kidney, is crucial to the management and treatment of diabetic kidney disease. It is vital to do research into the administration of antioxidant agents and their potential to restore the antioxidant defence system, therefore reducing ROS-mediated damage.

References

1 Adeshara KA, Diwan AG, Tupe RS (2016) Diabetes and complications: cellular signaling pathways, current understanding and targeted therapies. Curr Drug Targets 17: 1309-1328.

2 Arévalo-Lorido JC, Carretero-Gómez J, García-Sánchez F, Maciá-Botejara E, Ramiro-Lozano JM, et al. (2016) Secondary hyperparathyroidism prevalence and profile, between diabetic and non-diabetic patients with stage 3 to 4 chronic kidney disease attended in internal medicine wards MiPTH study. Diabetes Metab Syndr 10: 16-21.

3 Jagdale AD, Bavkar LN, More TA, Joglekar MM, Arvindekar AU (2016) Strong inhibition of the polyol pathway diverts glucose flux to protein glycation leading to rapid establishment of secondary complications in diabetes mellitus. J Diabetes Complications 30: 398-405.