

## STUDY OF SUPEROXIDE DISMUTASE ACTIVITY IN PLASMA OF DIABETIC PATIENTS HAVING NEPHROPATHY – A Review

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### ABSTRACT

Superoxide excess plays a central role in tissue damage that results from diabetes, but the mechanisms of superoxide overproduction in diabetic nephropathy (DT) are incompletely understood. In the present study, we investigated the enzyme superoxide dismutase (SOD), a major defender against superoxide, in the kidneys with nephropathy.

**Key words:** *Diabetic nephropathy, DT, Enzyme superoxide dismutase, Diabetes mellitus.*

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## INTRODUCTION

Diabetes mellitus is a group of metabolic disorders mainly characterized by hyperglycemia which occurs due to body's inability to synthesize insulin or utilize insulin to its full potential. It is a lifelong progressive metabolic disease affecting more than 230 million people worldwide and this number is expected to reach 350 million by year 2025. It is the fourth leading cause of death by disease globally and has become one of the most challenging health problem of 21st century. In a study by Bertram et al. on the prevalence of type 2 diabetes in 2009, they reported a potential increase from 5.5% to 9% in people aged 30 or older since the previous estimates of year 2000 representing approximately 2 million cases of diabetes in South Africa. Their secondary studies also revealed that around 55% of cases are undiagnosed for South Africa meaning around 1 million people with type 2 diabetes are unaware of their disease. They also modelled 8000 new cases of blindness and 2000 new amputations annually caused by diabetes.

Diabetic retinopathy, one of the complications of diabetes mellitus is the fifth leading cause of blindness in the world, affecting 1.8 billion people and responsible

for 4.8% of blindness. In South Africa, after cataract and glaucoma, retinopathy is the third leading reason of blindness. Diabetic retinopathy is characterized by gradual and progressive alterations in vascular system of retina due to chronic hyperglycemia. Diabetic retinopathy is classified into background or non-proliferative diabetic retinopathy (BDR/NPDR) and Proliferative diabetic retinopathy (PDR). The pathogenesis of diabetic retinopathy is very complicated and many factors contribute to it which includes persistent hyperglycemia in retinal vasculature leading to build-up of advanced glycation end-products (AGEs), inflammation, neuronal dysfunction, changes in redox homeostasis and oxidative stress. In the present study we are study the effect of super oxide dismutase in diabetic patients with nephropathy.

## Discussion

Hyperglycemia in diabetes activates various biochemical pathways leading to increased production of superoxide and hydroxyl radical which could lead to decrease in SOD enzyme activity. But conflicting results have been reported in different studies. Most researches have reported a decrease in SOD

enzyme activity in diabetic patients compared to healthy control group, whereas some did not report any changes and few researchers reported high serum ECSOD enzyme activity in diabetic patients. Mizobuchi et al., Turk et al., Kimura et al., Soliman and Bandeira et al. all reported an increase in EC- SOD enzyme activity in diabetic group as compared to healthy control group. They also reported that this increase was seen more in patients with microangiopathy as compared to patients without complications, which is similar to the present study finding.

The reason for increase in the total SOD enzyme activity in the patients with retinopathy in this study could be due to, increased expression of the enzyme as a

compensatory mechanism in response to increased oxidative stress. It may also be due to a reduced tissue binding of SOD as a result of glycation of SOD, which reduces the affinity of this enzyme to heparin without affecting its enzyme activity. In diabetes, the proportion of glycated SOD is higher than healthy control. Also another reason could be decrease in tissue binding of SOD due to reduced heparin sulphate leading to an increase in plasma SOD levels. This reason is more evidence based as there have been reports that in diabetic nephropathy, heparan sulphate is reduced in glomerular basement membrane, proportional to degree of proteinuria and damage to glomerulus can also reduce the ability of membrane glycolyx to bind SOD

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