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# ANALYTICAL STUDY ON OXIDATIVE AND ANTIOXIDATIVE STATUS IN PATIENTS WITH DIABETIC CATARACTS

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**Abstract**

The goal of this study is to evaluate the oxidative and antioxidative state of diabetic and non-diabetic individuals with cataracts, as well as healthy controls. In all, 149 people participated in this analysis. Blood samples were collected from people who had diabetic cataracts, people who had age-related cataracts, and those who were healthy enough to serve as a control group. In this study, we evaluated total oxidant status (TOS), total antioxidant status (TAS), total thiol levels (TTLs), and paraoxonase 1 (PON1) in the blood. TTLs were significantly lower in diabetes cataract patients compared to both the nondiabetic cataract patients and the control group. Patients with diabetic cataract also had significantly reduced plasma PON1 levels compared to the control group. Both TAS and TOS levels were similar between groups, without any statistical significance. This research shows that individuals with diabetes cataracts had significantly lower serum TTLs and serum PON1 activity than controls. These results point to problems with the body's antioxidant defenses, which may be linked to cataract formation.

**Key words:** Oxidative status, antioxidative status, diabetic cataract

## INTRODUCTION

Cataractogenesis is a complicated and multi-factoral process that may be triggered by a variety of environmental and genetic variables (1–3). Cataracts are a common complication of diabetes, which has been linked to a number of other complications (4,5). Despite the results of several clinical, epidemiological, and preclinical studies addressing the link between diabetes and cataractogenesis, the precise mechanism remains unclear. Many potential pathogenic mechanisms have been proposed, including abnormal glycosylation of lens proteins, advanced glycation end product formation, and increased tissue sorbitol concentration.

Cataracts are only one complication of diabetes that may arise from oxidative stress brought on by an excess of reactive oxygen species (ROS). When more reactive oxygen species (ROS) are produced than the body's antioxidant defences can remove, oxidative stress is present. Besides damaging DNA, cellular proteins, and lipids and creating catastrophic clinical effects, high amounts of reactive oxygen species (ROS) have long been suspected of having a critical role in cataractogenesis.

Although many other criteria have been used to measure oxidative stress state, total oxidant status (TOS) and total antioxidant status (TAS) are often used and safe markers for the evaluation of oxidative stress levels.

Antioxidants are chemicals that prevent oxidative damage to cells caused by free radicals. The body has various antioxidant defense mechanisms that neutralize the ROS. There has been a lot of research on how low antioxidant levels contribute to the pathogenic processes of many illnesses. It has been shown that diabetic cataract patients, as compared to both nondiabetic cataract patients and healthy controls, have a significantly reduced concentration of antioxidant molecules in their blood and lenses.

A set of metabolic illnesses, diabetes is characterized by persistently elevated blood sugar levels (hyperglycemia). Unconverted glucose has been linked to the progressive damage, malfunction, and failure of several organs, including the eyes, kidneys, nerves, heart, and blood vessels. Patients with diabetes also have a higher chance of acquiring malignancies such pancreatic and ovarian tumours.

Although it is believed that the diabetic condition, including its associated problems, is genetically determined, the risk of acquiring complications differs from patient to patient. However, the metabolic hypothesis suggests that long-term hyperglycemia leads to problems such as cellular and vascular damage. Diabetes-related complications might be delayed or reduced if blood sugar levels are kept consistently low, according to the Diabetes Control and Complications Research, a major randomised clinical trial. The purpose of this study was to compare the levels of TOS, TAS, TTL, and PON in diabetic patients with diabetic cataracts and nondiabetic cataract patients to a control group.

## LITERATURE REVIEW

Darenskaya, M.A., Kolesnikova, L.I. & Kolesnikov, S.I. (2021), Based on an examination of experimental and clinical data, This article presents the most up-to-date thinking on the role oxidative stress responses play in the genesis of type 1 and type 2 diabetes mellitus and the progression of associated comorbidities. Increased reactive oxygen species (ROS) generation in diabetes is traced back to the fundamental pathways of altered glucose metabolism, oxidative damage to pancreatic -cells, and endothelial dysfunction. Several types of stress, including oxidative stress, carbonyl stress, and inflammation, are covered in depth. The significance of oxidative stress responses from hyperglycemia is examined in light of the "metabolic memory" phenomenon. Our research shows that the parameters of the LPO—antioxidant defense system vary significantly among ethnic groups and over the lifespan in patients with diabetes mellitus, a fact that has to be taken into account in the context of comprehensive treatment for the condition. Antioxidants should be included in the treatment of type 1 and type 2 diabetes mellitus, since they have been shown to be helpful in several trials. Targeting reactive oxygen species (ROS) sources and incorporating novel antioxidant delivery techniques are two components of cutting-edge therapeutic approaches for managing diabetic mellitus.

F. R. Mancini; A. Affret; C. Dow; et al (2018), The idea that oxidative stress contributes to the development of type 2 diabetes is relatively new. The total antioxidant capacity index is a straightforward measure of antioxidant capacity that incorporates all dietary antioxidants. This study's overarching objective was to determine whether or not total antioxidant capacity was associated with an increased risk of acquiring type 2 diabetes. Methods: 1751 out of 64,223 women in the French E3N-European Prospective Investigation into Cancer and Nutrition (EPIC) cohort developed type 2 diabetes throughout the 15-year follow-up period. Total antioxidant capacity was determined using the ferric reducing antioxidant power (FRAP) method, which evaluates an organism's ability to neutralise free radicals by decreasing the concentration of ferric ions. To assess the associations between total antioxidant capacity and risk of type 2 diabetes, adjusted Cox proportional hazards regression models were used, which allowed for the determination of hazard ratios (HRs) and 95% confidence intervals (CIs) after correcting for potential confounders. Having a higher total antioxidant capacity was associated with a lower chance of developing type 2 diabetes in multivariate analyses. When comparing women in the third, fourth, and fifth quintiles for total antioxidant capacity to those in the lowest quartile, we find HRs of 0.74, 0.70, and 0.73, respectively. A linear decrease in risk of type 2 diabetes was shown in conjunction with increasing total antioxidant capacity up to 15 mmol/day, but the protective effect plateaued at higher levels. The evidence we provide shows that total antioxidant capacity may be particularly important in preventing type 2 diabetes in middle-aged women.

Diabetic retinopathy is a neurological disease with an unknown underlying mechanism (Sasaki, M., Ozawa, Y., Kurihara, T., et al., 2011). Here, we examined the role that oxidative stress plays in the retina and the antioxidant lutein plays in mitigating its effects on retinal neurodegeneration. Methods From the commencement of their diabetes, The metabolic effects of feeding C57BL/6 mice either a diet enriched with

lutein or a control diet were measured. Diabetic mice were studied for a month, with electroretinograms measuring visual function and dihydroethidium measuring ROS levels in the retina. Diabetic mice were studied for a month, and immunoblotting was utilised to evaluate synaptophysin, ERK, and BDNF activation in the retinas. After 4 months with diabetes, mice had their retinas sectioned to look for histological changes, cleaved caspase-3, and TUNEL staining. Although lutein did not affect the metabolic status of diabetic mice, it did decrease the generation of reactive oxygen species (ROS) in the retina and the severity of diabetic retinopathy. The antioxidant lutein prevented ERK activation, synaptophysin loss, and BDNF depletion in the diabetic retina. Lutein prevented the inner plexiform and nuclear layer atrophy, ganglion cell loss, and elevation of cleaved caspase-3 and TUNEL-positive cells in the retinas of 4-month-old diabetic mice. According to the findings, a diet rich in lutein protects the diabetic retina against the neurodegenerative effects of local oxidative stress. Antioxidant lutein may provide hope as a treatment option for preventing vision loss due to diabetes.

Aditya Kelkar, Jai Kelkar, Hetal Mehta, and Winfried Amoaku (2018), Patients with diabetes make up a significant percentage of those receiving cataract surgery in India, which is often regarded as the diabetes capital of the world. In light of this, we examined established practices and recommendations for treating cataract in diabetic individuals. Patients with diabetic cataracts need meticulous attention to detail before, during, and after surgery. The visual result of cataract surgery is heavily influenced by the diagnosis and treatment of preexisting diabetic retinopathy or maculopathy. We can learn how to effectively manage diabetic patients and get optimal results after cataract surgery if we have a deeper grasp of the elements involved for positive outcomes.

The authors' names are M. A. Darenskaya, L. I. Kolesnikova, and S. I. Kolesnikov (2020), Based on an examination of experimental and clinical data, This article presents the most up-to-date thinking on the role oxidative stress responses play in the genesis of type 1 and type 2 diabetes mellitus and the progression of associated comorbidities. Atypical glucose metabolism, oxidative damage to pancreatic -cells, and endothelial dysfunction are characteristics of diabetes and contribute to increased reactive oxygen species (ROS) generation in this illness. the complex relationship between oxidative damage, carbonyl stress, and inflammation. Using the concept of "metabolic memory," this article explores the role that oxidative stress responses play in hyperglycemia. In patients with diabetes mellitus, we found that the antioxidant defence system features of LPOs varied significantly by ethnicity and age; this variation has to be taken into consideration when treating such a complex disease. Since antioxidants have been widely examined for their effectiveness, they should be included into the treatment of both type 1 and type 2 diabetes. Targeting reactive oxygen species (ROS) sources and incorporating novel antioxidant delivery techniques are two components of cutting-edge therapeutic approaches for managing diabetic mellitus.

**Oxidative Stress-Induced Cellular Damage**

Every main class of biomolecules is susceptible to damage by ROS, and their namesake mechanisms are as follows:

**a. Proteins**

Evidence from in vitro studies suggests that reactive oxygen species (ROS) may react with several amino acid residues, leading to enzyme changes, protein denaturation, and protein malfunction. Peptide chain fragmentation and cross-linking reaction product aggregates lead to a change in electrical charge and an increase in proteolysis. 42 Different peptide amino acids and activated oxygen forms have different levels of responsiveness to attacks. The relative susceptibility of individual amino acids may be affected by the protein's primary, secondary, or tertiary structure. The blood total protein content of diabetic rats has been shown to drop for two reasons in laboratory studies: 1) reduced amino acids uptake,55 and 2) a drastic drop in the concentration of a number of important amino acids,56 respectively. protein synthesis is lowered because there is less mRNA available, and glycogenic amino acids are converted to carbon dioxide and water at a faster rate.

In vitro studies have demonstrated that neutrophil and monocyte-derived myeloperoxidase catalyses a specific process, leading to the conversion of L-tyrosine to 3-3-dityrosine.

Additionally, oxidatively formed dityrosine in proteins may connect neighboring polypeptide chains of the same or distinct proteins. Dityrosine is very uncommon since it lacks a peptide bond and instead has a covalent bond that is stable against protolytic destruction and acid hydrolysis. Dityrosine is a useful marker for oxidation because of its characteristic. It was postulated that the cross-linking of proteins caused by this altered amino acid as a signal for rapid and selective in vivo breakdown by intracellular protease in response to oxidative stress. However, it was previously thought that oxidatively damaged proteins become more resistant to breakdown with age, which would explain why they accumulate with age.

**b. Lipids**

The formation of reactive oxygen species (ROS) during glycation, glucose oxidation, and lipid oxidation has been suggested to contribute to the imbalance between oxidative stress measurements and antioxidant levels in diabetes, whose function has been under greater investigation in recent years. Lipid hydroperoxides (LHP) are produced from a broad variety of long-chain polyunsaturated fatty acid substrates through intermediate radical reactions, and their production requires oxygen and metal cations (iron and copper). These processes as a whole generate highly reactive and damaging lipid radicals, which, because to their proximity to other lipids in biomembranes, create more LHP. In addition, DM produces changes in lipid profiles, including an increased vulnerability to lipid peroxidation, which is responsible for an increased risk of atherosclerosis. When it comes to reactive oxygen species (ROS), lipid peroxidation has been studied the most since it is such a useful indicator of free radical-mediated oxidative stress. This marker is also a vital pathological sign in a wide range of illnesses. As a result, Studies of diabetogenesis are increasingly focusing on the roles that lipid hydroperoxide production and physiologically active metabolites play in modifying cellular structure and function.

Experimental investigations show that polyunsaturated fatty acids (PUFA) in the plasma membrane are particularly vulnerable to assault by free radicals because of the numerous double bonds they contain.

69 Lipid peroxidation, characterized by the degradation of membrane proteins due to subsequent free radical assaults, is caused when hydrogen is removed from a carbon atom in a polyunsaturated fatty acid (PUFA) or a lipoprotein. The oxidized lipoprotein (oxLDL) hypothesis is a popular explanation for this phenomenon. The plasma's principal cholesterol carrier is low-density lipoprotein (LDL); high levels of circulating LDL are associated with an increased risk of atherosclerosis; and particularly, high levels of oxidised low-density lipoprotein (oxLDL) have been associated with hypertension in men. LDL oxidation is greater in diabetic patients than in controls, according to a number of studies.

The accumulation of oxidized lipids in the cell membrane may disrupt cell function by allowing plasmalemma leakage and blocking the action of membrane-bound receptors. Furthermore, Byproducts of lipid peroxidation, such as unsaturated aldehydes and other metabolites, have cytotoxic and mutagenic effects, and oxLDL plays a special role in the pathogenesis of atherosclerosis. The oxidised low-density lipoprotein (oxLDL) is a major contributor to atherosclerosis because it promotes endothelial dysfunction, such as the inhibition of endothelial cell vasodilator function, and increases the release of cytokines and growth factors. There is evidence that lipid peroxidation may lead to membrane anomalies, one of which is a decrease in the reactivity of thiol groups in membrane proteins. PUFA may thus be used to repair oxidative membrane damage and restore normal membrane fluidity and function. Antioxidants that make up for a lack of PUFA may be helpful for transmembrane enzymes, receptors embedded in membranes, and membrane transport systems.

**Role of Antioxidant Defense System and Protection Mechanism**

Antioxidants were explored by chemists in the late 19th and early 20th centuries. This nebulous category of substances is characterized by its capacity to substitute for other compounds in an oxidation reaction. Antioxidants were originally used for a wide variety of purposes, from preserving food to vulcanizing rubber. It wasn't until the publishing of vitamins and flavanoids in the 1960s that scientists realized their significance in health. 39 Cameron and Pauling discovered in the 1970s that vitamin C (ascorbic acid) may help prevent cancer in humans. 76 Research into the processes, molecular targets, and molecular interactions of antioxidants has increased significantly over the last several decades, thanks in large part to the efforts of a number of well regarded scientists who are studying them as potential protective agents. 78 Recent years have seen an uptick in the study of antioxidants, their link to various pathophysiologic

processes, and their potential therapeutic implications, as seen by the proliferation of conferences and reviews devoted to the topic.

The antioxidant defence mechanisms in the body are assisted by both enzymatic and non-enzymatic pathways. Common non-enzymatic antioxidants include vitamins A, C, and E; glutathione; -lipoic acid; mixed carotenoids; coenzyme Q10; various bioflavonoids; antioxidant minerals; and cofactors such folic acid, uric acid, albumin, vitamins B1, B2, B6, and B12. These enzymes include superoxide dismutase, catalase, glutathione peroxidase, and glutathione reductase. Normal physiological pathways allow antioxidants to affect signal transduction, as well as the regulation of proliferation and the immunological response. Diets rich in fruits and vegetables have been linked to reduced cancer risks, which is encouraging evidence for the potential of antioxidants as a therapy for preventing and treating cancer, diabetes complications, and cardiovascular disease. Evidence from experimental, epidemiological, and clinical studies suggests that antioxidants may be helpful in managing diabetes and its related problems.

## MATERIALS AND METHODS

### Subjects and sample collection

Our sample included 42 people with diabetic cataracts, 60 people without diabetic cataracts, and 47 healthy people who served as controls. Research was performed in the Ankara Atatürk Research and Training Hospital's Ophthalmology Department. The study was given approval by the Ethics Committee of Atatürk Research and Training Hospital, and it was conducted in accordance with the Declaration of Helsinki and Good Clinical Practice. Informed permission was gained after participants were briefed on the study's purpose. There was a minimum age requirement of 55 for all participants. Nuclear cataracts were seen in the eyes of both diabetics and those without the disease. Participants were not included in the research if they had any of the following conditions: cardiovascular disease, kidney failure, liver disease, underactive or overactive thyroid, cataracts caused by trauma or toxins. The participants did not include any smokers or anyone who took vitamin or antioxidant supplements.

### Samples

The antecubital veins of both healthy controls and patients were used to collect blood samples in standard vacutainers. After 30 minutes of clotting, the blood was centrifuged at 4000 rpm for 10 minutes to separate the serum. The serum was then transferred to Eppendorf tubes and frozen at 80 °C until analysis.

### Measurement of biochemical parameters

Blood samples were analysed for concentrations of haemoglobin, haemoglobin A1c, apolipoprotein A, and fibrinogen in the serum. Standard chemistry laboratory equipment was used for the biochemical studies.

### Measurement of paraoxonase activities

In order to quantify paraoxonase activity, a paraoxon substrate assay was used. Using an autoanalyzer, we tracked the rise in absorbance at 412 nm at 37 °C to determine the paraoxon hydrolysis rate. At a pH of 8, the molar absorption coefficient was 17,000 M-1 cm-1, which allowed us to quantify the quantity of p-nitrophenol produced. Expression of paraoxonase activity was given in units per millilitre.

### Measurement of total oxidant status

Using a cutting-edge automated measuring approach, the TOS concentrations in the sera were calculated (17). When oxidants are present, the ferrous o-dianisidine complexes in the sample are reduced to ferric ions. There were a lot of glycerol molecules in the reaction medium, and these helped speed up the oxidation process. When added to the acidic medium of xylenol orange, ferric ions produce a colorful complex. Thus, the amount of oxidant molecules in a sample was correlated with its colour intensity as measured by spectrophotometry. The results were given in micromolar hydrogen peroxide equivalents per litre, with hydrogen peroxide serving as the benchmark.

**Measurement of total antioxidant status**

Erel's colorimetric automated TAS measurement technique was used to analyze the serum for TAS (18). The Fenton reaction generates the hydroxyl radical, bright yellow-brown dianisyl radical is formed when the colourless substrate O-dianisidine reacts with the most potent biological radical. Effective measurement of TAS is achieved by including a plasma sample in the reaction, since the antioxidant components of the plasma counteract the oxidative processes initiated by the hydroxyl radicals present in the reaction. The concentrations determined by the test were reported in units of mmol Trolox equivalents per liter.

**Measurement of total thiol status**

Elmman's and Hu's techniques for measuring sulfhydryl groups (-SH) in the blood of test subjects with thalassemia major (31). An anion with a strong colour peak at 412 nm is formed when thiols react with 5,5'-dithiobis-(2-nitrobenzoic acid). For the first time, this technique has been developed for use with a fully automated biochemistry analyzer.

Results were shown as means with 95% confidence intervals or as means with standard deviations. After controlling for age, we used analysis of covariance (ANCOVA) to determine whether there were statistically significant mean differences between groups. In the analysis of covariance (ANCOVA), clinical parameters were the dependent variables, while study groups and age were the fixed and covariate factors, respectively. A Bonferroni-adjusted multiple comparison test was employed to evaluate whether there were statistically significant differences between the groups. P-values under 0.05 were considered significant.

**RESULTS**

Table 1 displays the biochemical parameters that were measured in the patients who participated in the study. Patients with diabetic cataracts had considerably lower levels of fibrinogen and ApoA than the control group as a whole. The diabetic cataract group had considerably higher HbA1c values than the non-cataract group and the control group. The Hb values showed the same trends.

Table 2 displays the various TTL, PON, TAS, and TOS values. Patients with diabetes who also had cataracts had substantially shorter TTLs (P 0.001) than those without diabetes who also had cataracts and the control group. Thiol levels in individuals with cataracts who did not have diabetes were significantly different from those in the control group (P 0.05). (Figure 1).

In contrast to the control group, plasma PON1 activity was significantly lower in diabetic cataract patients. There was no noticeable difference in clinical outcomes between diabetes and nondiabetic cataract patients, despite the fact that their PON1 activity were lower in the former group. Cataract patients who did not have diabetes fared equally to the placebo group (Figure 2). There was no statistically significant difference in TAS or TOS values across groups.

**Table 1: Biochemical parameters of study groups (mean ± SD).**

| | Controls (n = 47) | Nondiabetic cataracts (n = 60) | Diabetic cataracts (n = 42) | P-value |
|---|---|---|---|---|
| Fibrinogen (mg/dL) | 295.8 (255.8–335.8) | 355.8 (322.5–389.2) | 407.8 (370.1–445.5) | <0.001 |
| ApoA (mg/dL) | 147.4 (137.1–157.6) | 136.3 (128.0–144.6) | 123.6 (114.1–133.1) | 0.005 |
| HbA1c (%) | 5.67 (5.33–6.04) | 5.85 (5.56–6.14) | 7.33 (7.01–7.66) | <0.001 |
| Folic acid (ng/mL) | 9.6 (8.5–10.6) | 7.8 (6.9–8.7) | 9.4 (8.4–10.4) | 0.018 |
| Hb (g/dL) | 13.9 (13.4–14.4) | 13.8 (13.4–14.2) | 12.8 (12.4–13.3) | <0.001 |

ApoA: apolipoprotein A, HbA1c: hemoglobin A1c, HB: hemoglobin

**Table 2: Enzyme levels of study groups (mean ± SD).**

| Variables | Controls (n = 47) | Nondiabetic cataracts (n = 60) | Diabetic cataracts (n = 42) | P-value |
|---|---|---|---|---|
| TTL (µmol/L) | 569.9 (544.6–595.3) | 525.1 (504.3–545.8) | 444.8 (421.0–468.6) | <0.001 |
| PON (U/L) | 241.9 (209.8–274.1) | 202.5 (176.2–228.8) | 178.3 (148.1–208.5) | 0.024 |
| TAS (mmol Trolox equiv/L) | 2.81 (2.70–2.92) | 2.69 (2.61–2.78) | 2.82 (2.72–2.93) | 0.126 |
| TOS (µmol H2O2 equiv/L) | 14.6 (12.9–16.4) | 15.1 (13.7–16.6) | 13.3 (11.7–15.0) | 0.254 |

TTL: total serum thiol levels, PON: serum paraoxonase activity, TOS: total serum oxidant status, TAS: total serum antioxidant status.

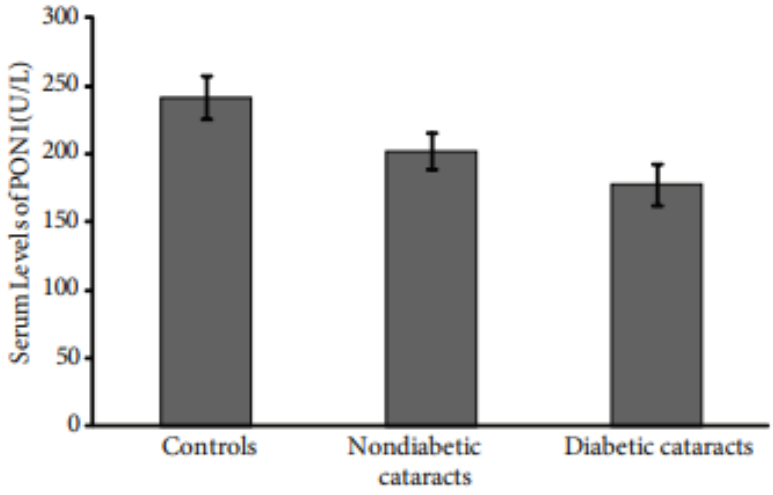


Figure 1: TTLs of study groups.

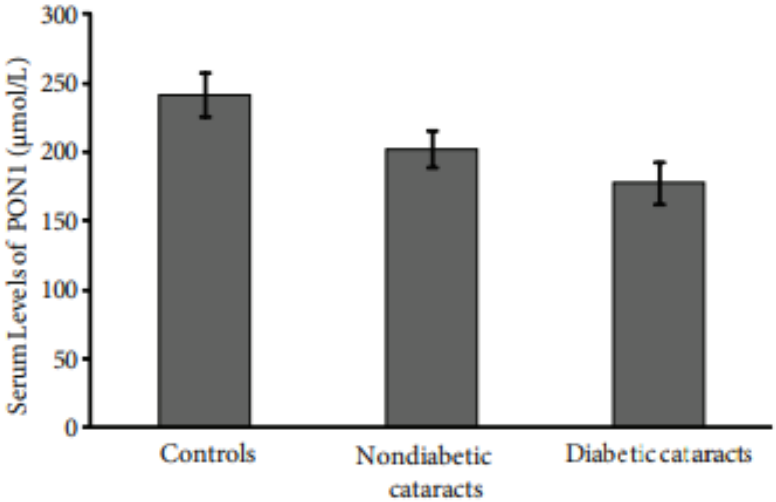


**Figure 2: PON1 activities of study groups.**

## DISCUSSION

Patients with diabetes cataracts were found to have considerably decreased serum TTL and PON1 levels compared to the control group. Diabetic cataract patients had significantly lower TTLs than non-diabetic cataract patients, a statistically significant finding in its own right. The TAS and TOS levels weren't much different across the board.

Total thiol-carrying molecule counts in plasma (TTLs) provide an estimate of the total number of such molecules in the plasma. Derivatives with sulfhydryl groups (-SH) like thiols are essential to the functioning of all forms of life. They are an important part of the body's antioxidant defence system and have several roles, including in protein structure and function, regulating enzymatic and transcriptional activity. Thiols' capacity to neutralize ROS is only one of several aspects that contribute to their potent antioxidant capabilities.

Although reactive oxygen species (ROS) serve a crucial function in regulating cellular activity, their overproduction may lead to oxidative stress and the subsequent destruction of proteins, lipids, and nucleic acids, which can manifest as a number of different illnesses in humans. Cataractogenesis may be aided by biochemical changes in lens tissue brought on by excessive exposure to reactive oxygen species (ROS), such as the degradation, crosslinking, and aggregation of lens proteins. Although the exact cause of diabetic hyperglycemia-induced increases in lens tissue reactive oxygen species (ROS) remains unknown, it is well accepted that this phenomenon occurs. Diabetic hyperglycemia enhances ROS production and directly triggers oxidative damage. The development of diabetic problems has been linked to the increased generation of reactive oxygen species (ROS) seen in diabetes (33). Abnormally high levels of reactive oxygen species (ROS) in the lens tissue cause lipid peroxidation, which in turn triggers metabolic events that damage lens fibers and scatter light via glycation of proteins.

To keep the levels at a healthy level, the body tightly controls the rates at which ROS are produced and removed. Several antioxidant systems in the body, both enzymatic and nonenzymatic, work together to neutralise the body's production of reactive oxygen species (ROS) and maintain this precarious balance. The deactivation mechanism and protection against ROS-induced oxidative damage are also supported by many antioxidant compounds consumed through the diet.

Thiol levels have been shown to be low in several illnesses and disorders that are considered to be pathological. Wet-type ARMD patients also had lower thiol levels than the control group. As far as we can tell, no prior research has analyzed plasma TTL levels in cataract patients.

## CONCLUSION

In conclusion, diabetic cataract patients had considerably lower levels of two key antioxidant molecules compared to controls. This discovery is indicative of a weakening of the body's antioxidant safeguards. The reduction in antioxidant status may indicate that the elevated ROS concentration generated oxidative stress, even though there were no variations in TOS and TAS levels. However, the specific processes through which oxidative stress may hasten the onset of problems in diabetes are only partially understood. Experimental, clinical, and epidemiological research have all shown that antioxidants may have a protective impact, suggesting that they may be useful in managing diabetes and its consequences. However, most prospective randomized controlled clinical trials of antioxidant supplementation (vitamins C, E, and -carotene) have failed to show a significant benefit, either in primary patients without clinical evidence of CVD or in secondary patients with clinical evidence of CVD, in the prevention of cardiovascular events.

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