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Research Article

JOURNAL OF OPHTHALMOLOGY AND RESEARCH

ISSN: 2644-0024



Association of Oxidative Stress-Related Gene Polymorphisms with Glaucoma in a South Indian Population

Asawari Kabure[1], Bhushan Kulkarni[2], Shilpa Kodkany[3], Nikita Harale[4], Murigendra Hiremath[1*]

Abstract

Glaucoma is a multifactorial optic neuropathy in which oxidative stress plays a critical pathogenic role by contributing to trabecular meshwork dysfunction and retinal ganglion cell loss. Genetic variations in oxidative stress–related genes may influence susceptibility to Glaucoma, particularly in populations with distinct genetic architectures such as India. This study aimed to evaluate the association between key oxidative stress gene polymorphisms and the risk of developing glaucoma in a Belagavi. A case-control study was conducted involving 24 clinically diagnosed Glaucoma patients and 20 age- and sex-matched healthy controls. Detailed demographic and clinical data were recorded, and all participants underwent IOP measurement, optic nerve evaluation, and visual field testing. Genomic DNA extracted from peripheral blood samples was used for genotyping *GSTM1/GSTT1* deletion variants and *CAT* and *GSTP1* polymorphisms. Genotype and allele distributions were compared between groups, and associations were analyzed. The *GSTM1* null genotype was more prevalent in cases (62.5%) than controls (35.0%), while *GSTT1* deletion was less frequent among cases. Neither *GSTM1* nor *GSTT1* deletions showed significant associations with glaucoma ($p = 0.13$ and $p = 0.76$, respectively). Similarly, no meaningful differences were observed in genotype or allele frequencies for *CAT* rs1001179 ($p = 0.779$) or *GSTP1* rs1695 ($p = 0.50$), indicating no significant relationship between these polymorphisms and glaucoma. Although *GSTM1* deletion appeared more common among Glaucoma cases, none of the oxidative stress–related genetic variants examined demonstrated a statistically significant association with Glaucoma in this cohort. These findings suggest that *GSTM1, GSTT1, CAT* rs1001179, and *GSTP1* rs1695 polymorphisms do not independently contribute to glaucoma susceptibility in the studied population. Larger, multicentric studies are warranted to validate these observations.

Keywords: Glaucoma; Oxidative stress; Genetic polymorphisms; GST; CAT; Indian population

Introduction

Glaucoma is a leading cause of irreversible blindness worldwide, characterized by progressive optic neuropathy with corresponding visual field loss and structural damage to the optic nerve head [1]. Emerging evidence from recent studies indicates that oxidative stress, resulting from a disturbance in the equilibrium between pro-oxidant and antioxidant systems, is a key contributor to POAG pathogenesis [2-5]. Under physiological conditions,

Affiliation:

*[1]Department of Microbiology and Biotechnology, Karnatak University, Dharwad, Karnataka India

[2]Dr. Prabhakar Kore Basic Science Research Center & JNMC Women's and Childs Research Unit, Belagavi, Karnataka, India

[3]Dr. Kodkany's Superspeciality Eye Care Centre, Belagavi, Karnataka, India

[4]D.Y. Patil Education Society, Deemed to be University, Kolhapur, Maharashtra, India

***Corresponding author:**
Murigendra Hiremath, Department of Microbiology and Biotechnology, Karnatak University, Dharwad, Karnataka India

Citation: Asawari Kabure, Bhushan Kulkarni, Shilpa Kodkany, Nikita Harale, Murigendra Hiremath. Association of Oxidative Stress-Related Gene Polymorphisms with Glaucoma in a South Indian Population. Journal of Ophthalmology and Research. 9 (2026): 90-96.

Received: November 11, 2026
Accepted: November 17, 2026
Published: November 24, 2026

Kabure A, et al., J Ophthalmol Res 2026
DOI:10.26502/fjor.2644-002400121

oxygen free radicals and other reactive oxygen species (ROS) are produced as by-products of normal metabolic and biochemical processes [6]. These are counterbalanced by the body's antioxidant defences, including enzymatic systems such as superoxide dismutase (*SOD*), catalase (*CAT*) and glutathione S-transferase (*GST*) amongst others, which collectively neutralize ROS and maintain redox homeostasis [6]. In the eye, when the balance between oxidant and antioxidant systems is disrupted, excessive ROS generation leads to oxidative damage in both trabecular meshwork (TM) and retinal ganglion (RGC) cells. This impairment of TM cells restricts aqueous humour outflow, elevates IOP, and ultimately triggers RGC apoptosis [7,8].

Considering this, genetic variations in oxidative-stress-related enzymes may alter individual susceptibility to POAG by modulating antioxidant capacity. Studies in diverse populations have linked polymorphisms in *SOD, CAT, GPX*, and glutathione S-transferase (*GST*) genes with altered risk of glaucoma and other neurodegenerative conditions. Several genetic association studies have explored oxidative stress related gene variants in glaucoma and ocular hypertension. In a study on Slovenian cohort, variants including *CAT* rs1001179, *GPX1* rs1050450 and *GSTP1* rs1695 were evaluated, although no statistically significant association with POAG risk was observed for the *CAT* and *GSTP1* single nucleotide polymorphism (SNPs) [2]. Abu-Amero *et al.*, investigated *GSTM1* and *GSTT1* gene polymorphisms in Arab glaucoma patients and found no significant association between these deletions and glaucoma risk [9]. These variations implicate on many other diseases like several cancers, asthma and also have been linked to neuronal cell damage in neurodegenerative disorders; Parkinson's, Alzheimer's and motor neuron disease [8]. Polymorphisms of GST widely expressed in ocular tissue also and shown to be associated with glaucoma, cataract, and age-related macular degenerations and optic neuropathies In another study, the *CAT* promoter variant was evaluated in a Saudi cohort with primary angle closure glaucoma, and while no association with disease susceptibility was found, a trend with visual acuity was observed [10].

In the Indian and international cohort several studies have explored oxidative stress–related gene variants, but data from North Karnataka remain scarce. Given the ethnic and genetic heterogeneity of Indian populations, the high burden of glaucoma in India, and environmental exposure to oxidative stressors it is crucial to evaluate the oxidative-stress-gene polymorphisms in Indian patients with POAG. This is the first genetic association study conducted in in Belagavi to evaluate oxidative stress related genes (including *CAT* rs1001179 and *GSTP1* rs1695) in patients with POAG. Restriction fragment length polymorphism (RFLP) analysis was employed to evaluate the potential association between these genetic polymorphisms and susceptibility to POAG in the study population. A better understanding of these genetic contributions may shed local disease susceptibility patterns and may contribute to the development of personalized preventive or therapeutic strategies in the future.

Materials and Methods

Study Population

This single center, prospective, case-control study was conducted at Prabhakar Kore's Basic Science Research Center, Belagavi, India. Ethical approval was obtained from the Institutional Ethics Committee of Prabhakar Kore's Basic Science Research Center, Belagavi (Letter No.: KLESKF/IEC/23/13), and informed consent was collected from all participants.

Sample Collection and Genomic DNA Preparation

Peripheral blood samples (10 mL) were collected in EDTA-containing vacutainer tubes from patients with POAG (cases) and individuals (control) meeting the inclusion and exclusion criteria, and stored at 4°C, following the protocol described by Malinowska *et al*. [11] Genomic DNA was extracted from fresh peripheral blood of glaucoma patients and healthy controls using the QIAamp DNA Blood Mini Kit (Qiagen), according to the manufacturer's instructions. DNA concentration and purity were assessed using a NanoDrop 2000c spectrophotometer (JH Bio), and integrity was confirmed by 0.8% agarose gel electrophoresis. The extracted DNA was resuspended in elution buffer and store at 20°C for subsequent PCR amplification and analysis [12].

Polymerase chain reaction (PCR) amplification

PCR amplification of selected single nucleotide polymorphisms (SNPs) in the *CAT* and *GSTP1* genes was carried out using gene-specific primers (details provided in Table 1). Each 25 µL reaction contained 50–100 ng of genomic DNA, 10 pmol of each primer, 200 µM of each dNTP, 1.5 mM $MgCl_2$, 1X Taq DNA polymerase buffer, and 1 unit of Taq DNA polymerase (Thermo Fisher Scientific). The amplification was performed in a thermal cycler under the following cycling conditions: an initial denaturation at 95°C for 5 minutes, followed by 35 cycles of denaturation at 95°C for 30 seconds, annealing at optimized temperatures specific to each primer set for 30 seconds, and extension at 72°C for 45 seconds. A final extension step was carried out at 72°C for 7 minutes.

Genotype Analysis of *GSTT1* and *GSTM1* Polymorphisms

GSTT1 and *GSTM1* genotypes of the study participants were determined by polymerase chain reaction (PCR) amplification as above (details given in Table 1). Products were resolved on agarose gel and visualized under UV

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DOI:10.26502/fjor.2644-002400121

illumination. The multiplex assay yielded bands of 480 bp (*GSTT1*) and 215 bp (*GSTM1*). All null genotypes were re-verified by separate confirmatory PCRs for *GSTT1* and *GSTM1* using β-globin as an internal control under identical conditions.

RFLP analysis and genotype determination

PCR-amplified products of *CAT & GSTP1* were digested using gene-specific restriction enzymes selected based on the SNPs of interest. Each reaction mixture included 10 µL of PCR product, 1X reaction buffer, and 5–10 units of restriction enzyme, followed by incubation at 37°C for 4–16 hours under optimal conditions. The resulting DNA fragments were separated by electrophoresis on 6% polyacrylamide gels, stained with ethidium bromide, and visualized under ultraviolet illumination using a UV transilluminator. Genotypes were assigned by analyzing the banding patterns. Alleles were scored accordingly, and the number of samples with each genotype was documented for further analysis.

Statistical Analysis

Descriptive statistics were used to summarize demographic and clinical variables, with results expressed as mean ± standard deviation (SD) for continuous variables and number (percentage) for categorical variables. Genotype and allele frequencies were calculated and compared between glaucoma patients and controls using the Chi-square test. A p-value < 0.05 was considered statistically significant.

Results

GSTM1/GSTT1 deletion patterns in cases and controls and association with POAG

The distribution of *GST* gene deletions showed noticeable differences between POAG cases and controls. The *GSTM1* null genotype was more frequently observed in cases (62.5%) than in controls (35.0%), while the *GSTT1* null genotype occurred less often in cases (16.7%) compared with controls (25.0%). Combined genotype analysis further demonstrated that the *GSTM1*-null/*GSTT1*-present genotype was the most common pattern among cases, whereas the double-null genotype remained uncommon in both groups (Figure 1; Table 2).

Table 2: Combined GSTM1–GSTT1 genotype distribution in cases and controls

Combined Genotype (*GSTM1* / *GSTT1*)	Cases (n = 24)	Controls (n = 20)
Both Present	7 (29.2%)	8 (40.0%)
GSTM1 Null / *GSTT1* Present	12 (50.0%)	5 (25.0%)
GSTM1 Present / *GSTT1* Null	4 (16.7%)	5 (25.0%)
Double Null (Both Deleted)	1 (4.2%)	2 (10.0%)

Analysis of GST gene deletions showed no statistically significant associations with POAG. Although the GSTM1 null genotype was more common in cases than controls, this difference did not reach statistical significance ($p = 0.13$). Similarly, the GSTT1 null genotype did not differ significantly between cases and controls ($p = 0.76$). These findings indicate that neither GSTM1 nor GSTT1 gene deletions demonstrate a significant independent association with POAG in this study population (Table 3).

Association of CAT, and GSTP1 gene polymorphisms with POAG

The analysis of the *CAT* rs1001179 polymorphism showed no significant association with POAG. Genotype frequencies (CC, CT, TT) were comparable between cases and controls, and the distribution of C and T alleles also showed no meaningful difference (C: 71.7% vs. 62.5%; T: 28.3% vs. 37.5%). Statistical testing demonstrated no

Table 1: Primer sequences, amplicon size, and restriction enzymes used for genotyping in *CAT, GSTM1, GSTT1* and *GSTP1* genes

Gene	SNP (rsID)	Primer Sequence (5′–3′)	Amplicon Size (bp)	Tm (°C)	Restriction Enzyme	Fragment Sizes (bp)
CAT	rs1001179	F: AGAGCCTCGCCCCGCCGGACCG R: TAAGAGCTGAGAAAGCAT AGC	185	63	SmI	C/C: 155+21+9 C/T: 164+21 T/T: 164+155+21+9
GSTM1	-	F: GAACTCCCTGAAAAGCTAAAGC R: GTTGGGCTCAAATATACGGTGG	215	62	-	-
GSTT1	-	F: GAACTCCCTGAAAAGCTAAAGC R: GTTGGGCTCAAATATACGGTGG	480	59	-	-
GSTP1	rs1695	F: AATACCATCCTGCGTCACCT R: TGAGGGCACAAGAAGCCCCTT	A:308,258 G:258,219,89	59	BsmAI	A:308,258 G:259,219,89

Abbreviations: SNP, single nucleotide polymorphism; rs, reference SNP, A, adenine; T, thymine; C, cytosine; G, guanine; F, forward; R, reverse; bp, base pair; Tm, melting temperature.

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DOI:10.26502/fjor.2644-002400121

significant relationship between *CAT* rs1001179 variants and disease status ($\chi^2 = 0.499$, $p = 0.779$), indicating that this SNP does not contribute independently to POAG susceptibility within this cohort (Figure1; Table 4).

Similarly, the *GSTP1* rs1695 polymorphism did not exhibit a significant association with POAG. The genotype distribution (AA, AG, GG) was comparable across cases and controls, and allele frequencies also showed no substantial differences (A: 29.5% vs. 42.9%; G: 70.5% vs. 57.1%). The chi-square test confirmed the absence of significant association ($\chi^2 = 1.37$, $p = 0.50$) (Figure 1; Table 4).

Table 3: Association of GSTM1-GSTT1 with Glaucoma

Gene	Genotype	Cases (n = 24)	Controls (n = 20)	X^2	*p*-value
GSTM1	Null (0/0)	15 (62.5%)	7 (35.0%)	2.29	0.13
	Present	9 (37.5%)	13 (65.0%)	—	—
GSTT1	Null (0/0)	4 (16.7%)	5 (25.0%)	0.09	0.76
	Present	20 (83.3%)	15 (75.0%)	—	—

Table 4: Genotypic and allelic distribution of CAT & GSTP1 polymorphisms and their association in glaucoma cases and controls

SNP	Genotype Frequency (Cases / Controls)	Allele Frequency (Cases / Controls)	Chi-square	*p*-value	Genotype Association with Glaucoma	Observation
CAT rs1001179	CC=15/11 CT=3/3 TT=5/6	C=71.7% / 62.5% T=28.3% / 37.5%	0.499	0.779	Not significant	Genotype and allele frequencies were comparable between POAG cases and controls.
GSTP1 rs1695	AA=3/5 AG=7/8 GG=12/8	A=29.5% / 42.9% G=70.5% / 57.1%	1.37	0.50	Not significant	No significant association observed between *GSTP1* rs1695 polymorphism and POAG; allele and genotype distributions are similar between groups.

Abbreviations: SNP, single nucleotide polymorphism; rs, reference SNP, A, adenine; T, thymine; C, cytosine; POAG, primary open-angle glaucoma

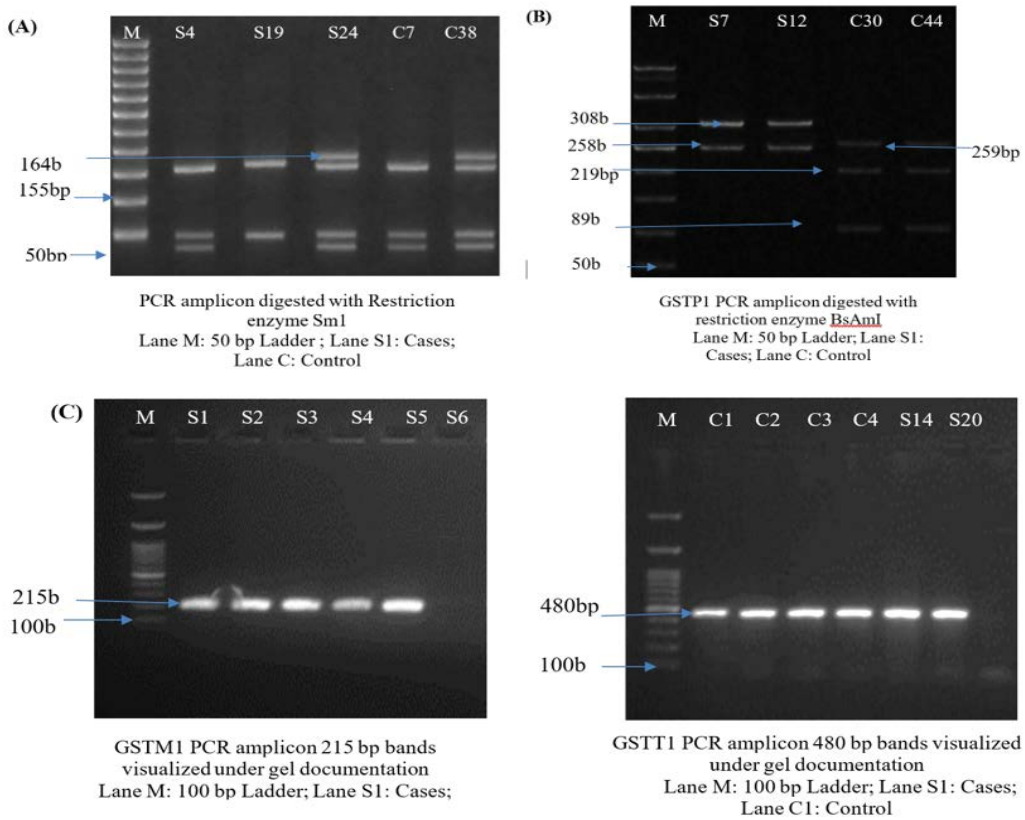

Figure 1: Representative RFLP gel electrophoresis patterns showing genotype distributions for SNPs analysed in the study. DNA was amplified by PCR and digested with specific restriction enzymes followed by separation on 3% agarose gels. **(A)** *CAT* rs1001179 **(B)** *GSTP1* rs1695 **(C)** *GSTM1 & GSTT1 gene deletion*

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Discussion

POAG is increasingly recognized as a neurodegenerative disorder in which oxidative stress plays a central pathogenic role. Retinal ganglion cells and trabecular meshwork tissues are particularly vulnerable to ROS, and impaired detoxification capacity may accelerate optic nerve damage. The GST superfamily constitutes a major antioxidant defense system responsible for conjugation of electrophilic compounds with glutathione. Among these, *GSTM1* and *GSTT1* gene deletions and *GSTM1/T1*-null genotypes have been explored extensively for their contribution to POAG susceptibility. In the present study, although no statistically significant association was identified, the distribution trends, especially the higher prevalence of the *GSTM1* null genotype in cases, support the hypothesis that compromised GST-mediated detoxification may influence oxidative stress burden in POAG. To the best of our knowledge, this is the first study evaluating oxidative stress–related gene polymorphisms in POAG patients from the Belagavi region of Karnataka, India.

The finding of this study show that that the *GSTM1* null genotype was more frequent in POAG cases (62.5%) than controls (35%), though not statistically significant, aligns with multiple reports suggesting its role in increasing oxidative susceptibility. The *GSTM1*-null genotype reduces cellular capacity to neutralize electrophilic and oxidative intermediates, contributing to trabecular meshwork damage and elevated IOP. Studies from Turkey [13], Brazil [14] (Rocha), and China [15] have similarly reported positive associations between *GSTM1*-null genotypes and POAG, often with moderate to strong effect sizes. These findings reinforce the possibility that reduced *GSTM1* activity potentiates ROS-mediated neuronal loss in genetically susceptible individuals. Conversely, several populations, including Caucasian cohorts in European population [16] (Jannson), Greece [17], and Saudi Arabia [6] have reported no such association, supporting the negative or null trend observed in the present cohort. These inconsistencies across populations may be attributed to ethnic differences in deletion frequencies, environmental exposures, lifestyle factors (e.g., diet, smoking), and population stratification. For *GSTT1* deletion, results of this study showed a slightly higher null frequency among controls than cases, consistent with a number of previous studies where *GSTT1*-null genotype did not emerge as a significant risk factor [6]. However, positive associations have been documented in certain East Asian and Latin American groups, suggesting that population-specific gene–environment interactions may modulate *GSTT1*'s effect on POAG risk.

The combined genotype analysis did not reveal significant differences, though the *GSTM1*-null/*GSTT1*-present pattern was more frequent in cases, supporting *GSTM1*'s stronger role in oxidative susceptibility. The double-null genotype, which theoretically confers the weakest detoxification capability, remained infrequent in both groups. These findings mirror global patterns where combined *GST* deletions occasionally amplify risk (as seen in studies from Turkey and Brazil) [13,14] but generally produce inconsistent associations. Considering the fundamental role of *GST* enzymes in counteracting oxidative stress, it remains biologically plausible that *GST* deletions could influence POAG development. Trabecular meshwork cells from *GSTM1*-null individuals show evidence of ROS accumulation, mitochondrial dysfunction, and accelerated senescence, mechanisms directly linked to aqueous outflow resistance and IOP elevation. Therefore, while statistical significance was not reached in the dataset of the present study, the direction of effect is consistent with oxidative-stress-centric models of POAG pathogenesis.

Several studies have specifically examined the *CAT* rs1001179 promoter variant in glaucoma cohorts, with variable results across populations. In a Saudi study of 225 POAG patients and 403 controls, no statistically significant difference in rs1001179 genotype or allele frequencies was observed between the groups, although a trend was noted between the T-allele and clinical indices such as age of onset, intraocular pressure, and disease duration [10]. Similarly, in a Chinese cohort of 416 POAG patients and 997 controls, Gong et al. failed to find an association with rs1001179, although they reported a significant link for another *CAT* SNP (rs769217), suggesting that functional *CAT* variation affecting POAG risk may differ by ethnicity [23]. These cross-population findings underscore the complexity of catalase's role in glaucoma and reinforce that genetic regulation of antioxidant defence is likely modulated by both ethnic background and specific functional variants. Consistent with these international findings, the present study also found no significant differences in *CAT* rs1001179 genotype or allele distributions between POAG patients and controls. The convergence of evidence from Saudi Arabia, China, and the current Indian cohort collectively reinforces that rs1001179 is unlikely to serve as a strong independent predictor of POAG.

Multilayered oxidative stress genetics parallel evaluation of *CAT* rs1001179 and *GSTP1* rs1695 polymorphisms also demonstrated no significant associations, further supporting the multifactorial and polygenic nature of oxidative stress regulation in POAG [18]. *GSTP1* is particularly abundant in ocular tissues and plays a crucial role in protecting the optic nerve head from oxidative injury [18,19]. Despite this biological relevance, reports from India and other Asian populations have often shown mixed or weak associations, similar to the results of this study. Likewise, *CAT* gene variants have produced inconsistent findings globally, suggesting redundancy and compensatory antioxidant mechanisms within ocular tissues.

Despite the lack of significant associations, this study provides valuable baseline genetic data for the Belagavi population. Given the genetic diversity within India, regional studies are essential to understand local disease patterns. The observed higher frequency of *GSTM1* null genotype among cases, although not statistically significant, warrants further exploration in larger cohorts.

These findings pave the way for future investigations into the functional implications of these variants, which may reveal novel molecular mechanisms and therapeutic targets. Moving forward, genome-wide association studies and next-generation sequencing approaches can offer a comprehensive overview of genetic contributors to POAG. Integrating genetic data with clinical parameters and biomarkers may enable personalized risk stratification and early intervention strategies. Such efforts are crucial for developing targeted therapies and improving clinical outcomes in POAG patients, particularly in genetically diverse populations such as India.

Conclusion

The associations between oxidative stress–related antioxidant gene variants, *GSTM1* and *GSTT1* deletions, and the *CAT* rs1001179 and *GSTP1* rs1695 polymorphisms, and susceptibility to primary open-angle glaucoma were evaluated in an Indian cohort, and no statistically significant relationships were identified. Although the *GSTM1* null genotype and certain combined *GSTM1/GSTT1* patterns appeared more frequent among cases, these differences were not significant, and *CAT* and *GSTP1* genotype and allele distributions were comparable between groups. While strong independent genetic effects were not demonstrated, the observed trends support the biological plausibility that weakened antioxidant defence and increased oxidative stress may contribute to POAG pathophysiology. Finding of this study add to the growing body of evidence from South Asia and the Middle East indicating that *GSTM1/GSTT1* deletions and *CAT/GSTP1* polymorphisms are not consistent or robust predictors of POAG. The variability in oxidative-stress–gene associations across populations highlights the need for larger, ethnically diverse studies integrating environmental exposures and broader redox-regulatory pathways.

Limitations of the Study

A key limitation of the current study is the relatively small sample size, which may restrict the statistical power and generalizability of the findings. Larger, multicentric studies are needed to validate these associations. Furthermore, functional assays are essential to determine the biological relevance of identified SNPs. The role of gene-gene and gene-environment interactions also remains an open area for research.

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Citation: Asawari Kabure, Bhushan Kulkarni, Shilpa Kodkany, Nikita Harale, Murigendra Hiremath. Association of Oxidative Stress-Related Gene Polymorphisms with Glaucoma in a South Indian Population. Journal of Ophthalmology and Research. 9 (2026): 90-96.