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Research Article

In Vitro Pharmacological Evaluation of Prunetine for the Management of Cataract

Bhumika Chandrakar [1], Swarnali Das Paul [1], Nivedita Gautam [2], Rajesh Chaudhary [1], Jaya Shree [1]*

[1] Shri Shankaracharya Professional University, Junwani, Bhilai, Chhattisgarh, India

[2] Columbia College of Pharmacy, Raipur, Chhattisgarh, India

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For Correspondence:

Dr. Jaya Shree, Shri Shankaracharya Professional University, Junwani, Bhilai, Chhattisgarh, India

Abstract

Cataracts, characterized by lens opacity and vision impairment, remain a leading cause of blindness globally. Oxidative stress plays a major role in cataractogenesis, with antioxidants offering therapeutic potential. This study investigates the anti-cataract efficacy of prunetine in a glucose-induced in vitro cataract model using goat lenses. Various concentrations of Prunetine were evaluated against standard treatment (ascorbic acid), with assessments on lens opacity, enzymatic antioxidant activity, lipid peroxidation, and protein content. The findings suggest Prunetine significantly inhibits cataract formation, restores biochemical parameters, and may delay cataract progression. Lenses exposed to high glucose showed significant opacification, decreased antioxidant enzymes (CAT, SOD, GSH), elevated MDA levels, and abnormal protein aggregation. Treatment with Prunetine significantly reduced opacity and restored antioxidant enzymatic activity. The 100 µg/ml dose of Prunetine was most effective, restoring lens biochemistry near or above normal levels and preventing protein aggregation more efficiently than ascorbic acid. Prunetine demonstrated potent anti-cataract effects by mitigating oxidative damage and preserving lens protein integrity. The findings underscore its potential as a non-surgical pharmacologic intervention for cataract prevention, warranting further in vivo and clinical evaluation.

Keywords - Oxidative stress, Cataractogenesis, lipid peroxidation, Cataracts, antioxidant

INTRODUCTION

Cataract, defined by the progressive clouding of the ocular lens, remains a principal cause of visual impairment and blindness globally, accounting for approximately 51% of all cases of blindness, particularly in older adults [6]. Though modern cataract surgery is commonly practiced, its affordability and availability remain limited in low-resource settings. Furthermore, surgery does not prevent continued oxidative damage in the contralateral lens, nor address underlying biochemical causes of lens opacity. As a result, increasing attention is being directed toward identifying pharmacological agents capable of delaying or preventing cataractogenesis [10-11].

Pathophysiologically, cataract formation is closely linked with chronic oxidative stress, protein aggregation, and polyol pathway abnormalities, particularly in hyperglycaemic conditions [16,19]. In diabetic patients, high glucose levels are converted to sorbitol via aldose reductase, leading to osmotic stress, production of Reactive Oxygen Species [ROS], and lipid peroxidation. These oxidative insults deplete protective antioxidant defences such as catalase [CAT], superoxide dismutase [SOD], and glutathione [GSH], causing malondialdehyde [MDA] accumulation and structural breakdown of lens proteins, particularly elevated insoluble protein fractions. Therefore, supplementing the lens with exogenous antioxidant compounds may serve as a viable strategy to halt or delay opacification. Natural flavonoids are of particular interest due to their ability to scavenge free radicals, reduce lipid peroxidation, and upregulate endogenous antioxidant enzyme systems [8,14,31]. Among them, Prunetine, a methylated flavonoid derived from leguminous plants and fruits, has demonstrated antioxidant and anti-inflammatory activities in cellular systems, though its application in ocular models remains poorly characterized. Given the structural similarity to established flavonoids like genistein and apigenin both known to exert lens-protective effects Prunetine is a strong candidate for further pharmacological validation.This investigation was designed to evaluate the anti-cataract activity of Prunetine in a glucose-induced lens opacity model using goat lenses cultured ex

vivo.The model simulates diabetic cataractogenesis through osmotic and oxidative stress pathways[12,16,20,25]. The study assessed both morphological (opacity grade) and molecular parameters, including antioxidant enzyme levels [CAT, SOD], oxidative stress marker [MDA], GSH status, and changes in soluble/insoluble protein distribution all of which are central indicators of lens health [1,13,17,36,37,38]. The hypothesis was that Prunetine treatment would mitigate glucose-induced oxidative damage and preserve lens transparency via activation of endogenous antioxidant systems and inhibition of protein aggregation. Results from this study support Prunetine's potential role as a therapeutic candidate for non-surgical cataract management and lay the groundwork for future in vivo and clinical assessments [2,8,15,19,35].

EXPERIMENTAL WORK

Prunetin was purchased from Alpha Chemicals and the chemicals from institution's central store with 95.2% purity and catalog no.552-590. Fresh, unopacified goat lenses were obtained from a local abattoir in Durg, Chhattisgarh [India]. Immediately post-enucleation, the eyeballs were transported to the laboratory under cold chain conditions [0–4°C] using an insulated ice box to preserve tissue integrity. Lenses were carefully excised using the extra-capsular extraction method, ensuring no mechanical damage, and were rinsed in cold phosphate-buffered saline [PBS, pH 7.8]. The lenses were incubated in artificial aqueous humor containing essential electrolytes [5 mM KCl, 2 mM $MgCl_2$, 0.5 mM $NaHCO_3$, 0.5 mM NaH_2PO_4, 140 mM NaCl, 0.4 mM $CaCl_2$] with glucose concentrations regulated depending on group assignments: 5.5 mM for the normal control and 55 mM for the cataractogenically challenged groups at under controlled incubation condition. The pH was adjusted to 7.8. To prevent microbial contamination during the 72-hour incubation period, penicillin [32 µg/ml] and streptomycin [250 µg/ml] were supplemented in the media [16-20].

To induce cataractogenesis, lenses in all experimental groups (except the normal control) were exposed to high glucose [55 mM], known to initiate osmotic and oxidative stress via the polyol pathway. Specifically, aldose reductase metabolizes excess glucose into sorbitol, causing intracellular accumulation, osmotic imbalance, and oxidative damage, ultimately leading to lens opacification. Thirty-six isolated goat lenses were randomly allocated into six groups (n = 6 per group) into six experimental groups: Normal Control, Negative Control Glucose 55 mM only], Standard [Ascorbic acid 40 µg/ml], and three Prunetine-treated groups [20, 60, and 100 µg/ml, respectively] [39,40].

Lens Homogenate Preparation

Post-incubation, lenses were washed in PBS, weighed, and homogenized in cold PBS buffer [0.23 M; pH 7.8] containing 0.25 mM EDTA to a final concentration of 10% [w/v]. The homogenate was centrifuged at 10,000 rpm for 1 hour at 4°C. The clear supernatant was collected for subsequent biochemical estimations including oxidative stress markers and total protein content [24-27].

Estimation of Oxidative Stress Parameters

All assays were performed spectrophotometrically using a UV-Visible spectrophotometer [UV-1900i, Shimadzu Scientific Instruments, USA].

Catalase [CAT] Activity: The reaction mixture contained 2.5 ml phosphate buffer [0.1 M, pH 7.0], 0.5 ml of 30% H_2O_2, and 0.1 ml lens supernatant. After incubation at 25°C for 10 minutes, 1 ml each of potassium dichromate [0.1 M] and sulfuric acid [1 M] was added to terminate the reaction. Residual H_2O_2 was measured spectrophotometrically at 240 nm [1].

Superoxide Dismutase [SOD] Activity: SOD activity was determined according to the method of Nishikimi et al. [36]. The assay mixture consisted of phosphate buffer [0.1 M, pH 7.8], NBT, PMS, NADH, and lens homogenate. The inhibition of NBT reduction was measured spectrophotometrically at 560 nm. [36]

Reduced Glutathione [GSH] Assay: For this, 1 ml phosphate buffer [0.1 M, pH 7.4], 0.1 ml DTNB [10 mM], and 0.1 ml lens supernatant were mixed. Additionally, 0.01 ml glutathione reductase [1 U/ml] and 0.01 ml NADPH [1 mM] were added, followed by 5-minute incubation at 25°C. Absorbance was read at 412 nm [38].

Glutathione Peroxidase [GPx] Activity : GPx activity was determined according to the method of Paglia and Valentine. The assay mixture consisted of phosphate buffer [pH 7.0], reduced glutathione, glutathione reductase, NADPH, and hydrogen peroxide. The oxidation of NADPH was monitored spectrophotometrically at 340 nm, and GPx activity was expressed as U/mg protein [37].

Malondialdehyde [MDA] Assay: To 1 ml of lens homogenate, 1 ml of 10% TCA and 1 ml of 0.67% TBA were added, along with 0.1 ml of 1 M HCl. The mixture was incubated at 95°C in a water bath for 30 minutes and then cooled. The absorbance of the resulting pink chromogen was measured at 532 nm [17].

Total Protein Estimation [Lowry's Method] : Soluble and insoluble proteins were analyzed using the Lowry method. The supernatant was used to determine soluble protein content, while the sediment was solubilized in 1N NaOH for insoluble protein estimation. To each 0.1 ml sample, 4 ml of alkaline copper reagent was added, allowed to stand briefly, followed by the addition of 0.4 ml Folin–Ciocalteu's phenol reagent. After vortexing and incubation at room temperature (30 minutes), the absorbance was recorded at 610 nm. Bovine serum albumin [BSA] was used as a standard for calibration [13].

RESULT AND DISCUSSION

Visual examination of the lenses after 72 hours of incubation revealed significant differences in opacity between treatment groups [Table 2, Figure 01]. The negative control group [Group II], which was exposed to 55 mM glucose without any treatment, exhibited maximal lens opacification with an opacity score of 3, reflecting severe cataractogenesis. In contrast, the normal control [Group I], maintained in physiological glucose [5.5 mM], showed complete transparency

[opacity score 0]. Notably, the standard group [Group III] treated with 40 µg/ml of ascorbic acid demonstrated reduced lens opacity [score 1], confirming its known antioxidative and lens-preserving properties. Prunetine-treated lenses displayed a dose-dependent reduction in opacity. Group IV [20 µg/ml] showed moderate opacity [score 2], while Groups V [60 µg/ml] and VI [100 µg/ml] showed opacity scores of 1 and 0, respectively, suggesting that higher concentrations of Prunetine conferred substantial protection and were comparable, or superior, to the ascorbic acid standard. Catalase is a vital antioxidant enzyme responsible for the degradation of hydrogen peroxide [H_2O_2], a major reactive oxygen species [ROS] in the lens. As shown in Table 3 and Figure 02, a significant reduction in CAT activity was observed in the glucose-induced cataract group [Group II: 23.74 ± 0.26^c moles H_2O_2/min/mg], indicating oxidative stress and enzyme suppression. Treatment with ascorbic acid [Group III] effectively restored CAT activity to near-normal levels [70.82 ± 0.15 moles H_2O_2/min/mg]. Prunetine also demonstrated a concentration-dependent enhancement, with Group VI [100 µg/ml] exhibiting the highest improvement [73.07 ± 0.34^f moles H_2O_2/min/mg], even surpassing the normal control. These results suggest that Prunetine may stimulate or preserve catalase activity in oxidative environments, thereby offering protection against lens opacification.Superoxide Dismutase [SOD] Activity SOD is another critical antioxidant defense enzyme that catalyzes the dismutation of superoxide radicals to hydrogen peroxide. In the negative control group [Group II], SOD activity was substantially depressed [5.40 ± 0.38 U/mg] [Table 4, Figure 03], indicative of impaired antioxidant capacity. Treatment with ascorbic acid [Group III] significantly improved SOD levels [17.02 ± 0.43 U/mg]. Among Prunetine groups, Group VI again showed the most profound restoration [13.55 ± 0.26^f U/mg], exceedingly even the normal baseline activity. This suggests that Prunetine may augment endogenous enzymatic defences and effectively neutralize oxidative free radicals implicated in cataractogenesis.Reduced glutathione [GSH], a key intracellular antioxidant, plays an indispensable role in maintaining lens transparency and detoxifying peroxides. As shown in Table 5 and Figure 04, the lens GSH levels were drastically depleted in the negative control group [3.16 ± 0.31 nmol/mg], supporting the role of oxidative stress in glucose-induced cataract formation. Standard treatment with ascorbic acid restored GSH to near-normal levels [12.53 ± 0.49], while Prunetine treatments yielded a dose-dependent improvement. Notably, Group VI [Prunetine 100 µg/ml] achieved the most significant increase in GSH content [12.68 ± 0.27^f nmol/mg], even higher than the normal control, reflecting potent antioxidant replenishing capacity.Malondialdehyde [MDA] is a well-established biomarker of lipid peroxidation and oxidative tissue damage. The negative control group exhibited markedly elevated MDA levels [15.32 ± 0.16 nmol/g], indicating substantial membrane lipid damage [Table 6, Figure 05]. Ascorbic acid treatment successfully reduced MDA to 5.57 ± 0.25, while Prunetine at 100 µg/ml lowered MDA even further to 3.30 ± 0.24 nmol/g. Lower doses [20 and 60 µg/ml] also attenuated MDA formation but less pronouncedly. These results confirm that Prunetine mitigates lipid peroxidation, a crucial contributor to cataract progression.Protein aggregation and insolubilization are definitive biochemical events in cataract formation. In the negative control group, soluble protein levels were drastically reduced [162.7 ± 0.16 µg/mg], and insoluble protein content was significantly elevated [392.5 ± 33.1 µg/mg] [Table 7, Figures 06 & 07]. In contrast, the standard group [ascorbic acid] preserved soluble protein content [425.0 ± 0.84 µg/mg] and suppressed protein insolubilization. Prunetine-treated groups demonstrated similar trends: Group VI presented the highest soluble protein content [491.4 ± 1.98 µg/mg] and the lowest insoluble protein accumulation [112.2 ± 8.1 µg/mg], indicating a protective effect against protein degradation and aggregation. These findings suggest that Prunetine maintains protein structure and function in the lens, likely by reducing oxidative burden and preventing cross-linking precipitated by ROS.

Table 1: Treatment groups for Anti-cataract activity

Group No	Name	Treatment	Drug Dose
I	Normal Control	Aqueous Humour + 5.5mM Glucose	-
II	Negative Control	Aqueous Humour + 55mM Glucose	-
III	Standard	Aqueous Humour + 55mM Glucose + Ascorbic Acid	40µg/ml
IV	Test 1	Aqueous Humour + 55mM Glucose + Prunetine	20µg/ml
V	Test 2	Aqueous Humour + 55mM Glucose + Prunetine	60µg/ml
VI	Test 3	Aqueous Humour + 55mM Glucose + Prunetine	100µg/ml

[All incubations performed at 37°C for 24 hours.]

Table 2: Assessment of Lens Opacity [Lens opacity was scored visually after 24 hours]

Group No	Name	Degree of Opacity
I	Normal Control	0
II	Negative Control	3
III	Standard	1
IV	Test 1 [20µg/ml]	2
V	Test 2 [60µg/ml]	1
VI	Test 3 [100µg/ml]	0

Opacity scoring: 0 = clear, 1 = mild opacity, 2 = moderate opacity, 3 = dense opacity.

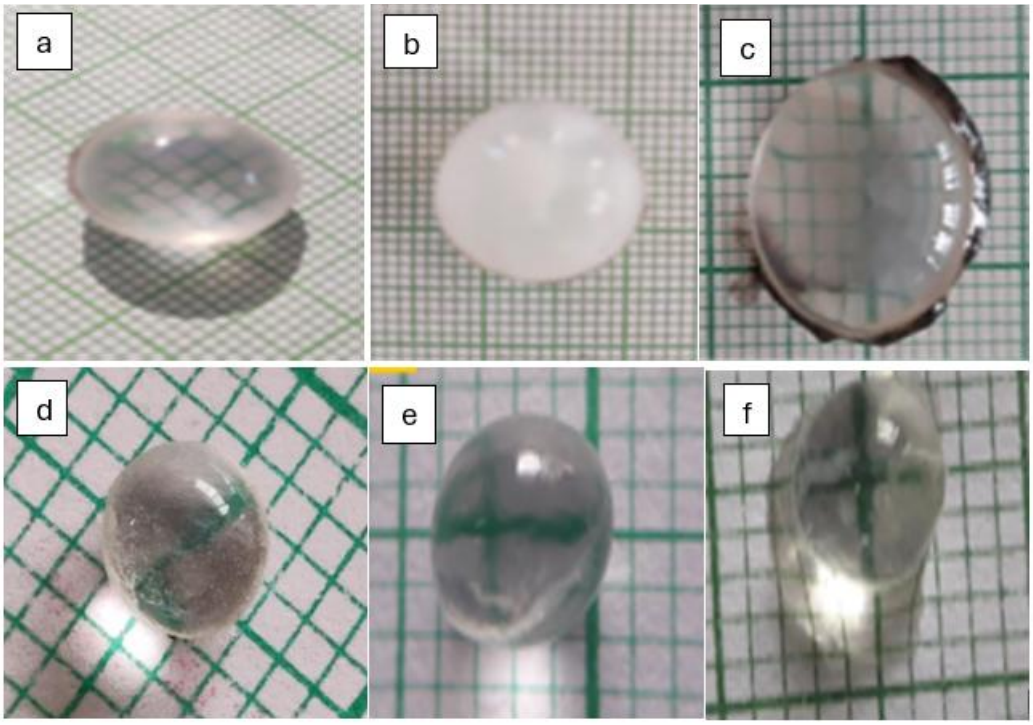

Figure 1: Anti-cataract activity. a: Normal group b: Negative Control c: Standard d: Test 1 Prunetine [20µg/ml] e: Test 2 Prunetine 2 60 f: Test 3 Prunetine 100

Table 3: Catalase [CAT] Activity [Measured as moles of H_2O_2 decomposed per minute per mg protein]

S.No	Group	Catalase [µmoles]
1	I	74.74 ± 0.27
2	II	23.74 ± 0.26^{c}
3	III	70.82 ± 0.15
4	IV	50.78 ± 0.20^{f}
5	V	58.90 ± 0.24^{f}
6	VI	73.07 ± 0.34^{f}

Table 3: Effect of prunetine on catalase: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test,a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

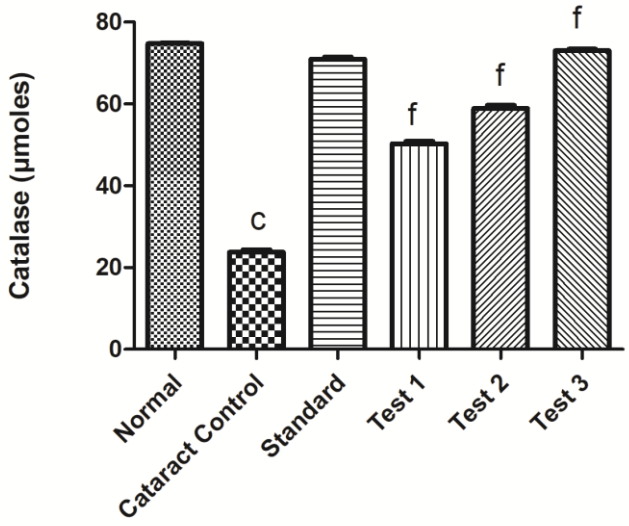

Figure 2: Effect of prunetine on catalase: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001]. **Catalase [CAT] Activity was measured as moles of H_2O_2 decomposed per minute per mg protein.**

Table 4: Superoxide Dismutase [SOD] Activity Measured as U/mg protein

S.No	Group	SOD [U/mg]
1	I	14.02 ± 0.47
2	II	4.10 ± 0.38[c]
3	III	13.08 ± 0.43
4	IV	7.25 ± 0.37[f]
5	V	9.03 ± 0.23[f]
6	VI	13.55 ± 0.26[f]

Table 4: Effect of prunetine on superoxide dismutase: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

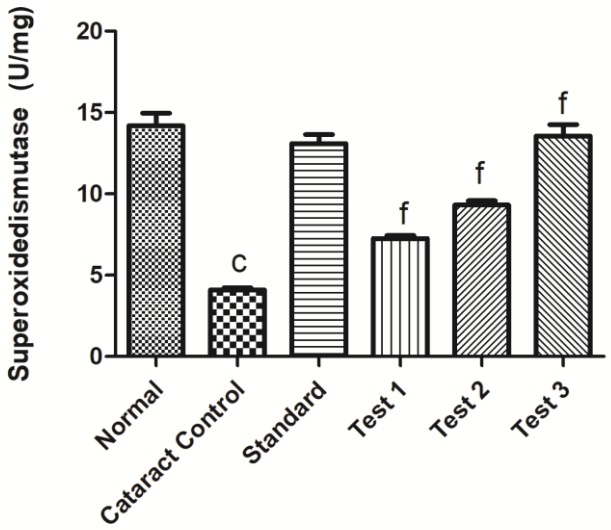

Figure 3: Effect of prunetine on superoxide dismutase: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001]..**Superoxide Dismutase [SOD] Activity Measured as U/mg protein**

Table 5: Effect of Prunetin on reduced glutathione (GSH) levels

S.No	Group	GSH [nmol/mg]
1	I	13.23 ± 0.31
2	II	3.36 ± 0.31[c]
3	III	11.81 ± 0.49
4	IV	8.28 ± 0.19[f]
5	V	10.75 ± 0.22[f]
6	VI	12.68 ± 0.27[f]

Table 5: Effect of prunetine on glutathione: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, where a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

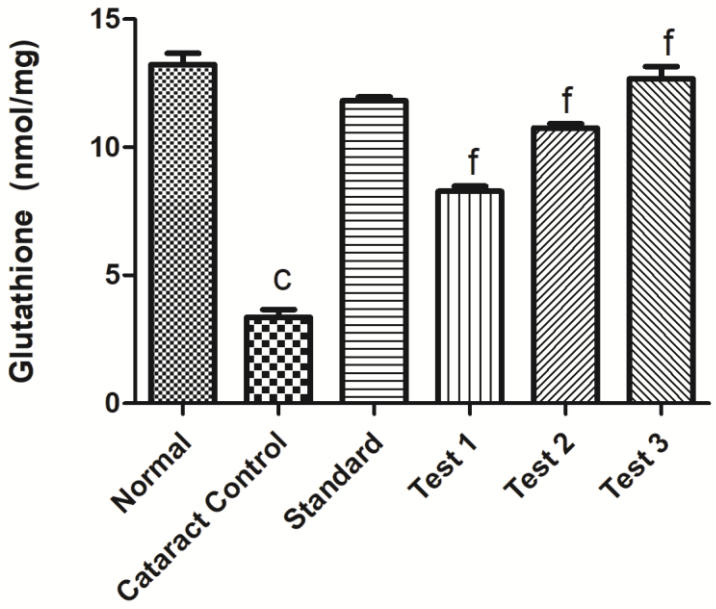

Figure 4: Glutathione [GSH] Enzyme Assay: Effect of prunetine on glutathione: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

Table 6: Effect of Prunetin on malondialdehyde (MDA) levels

S.No	Group	MDA [nmol/g]
1	I	7.40 ± 0.16
2	II	15.32 ± 0.16[c]
3	III	5.57 ± 0.25
4	IV	9.21 ± 0.18[f]
5	V	7.05 ± 0.23[f]
6	VI	3.30 ± 0.24[f]

Table 6: Effect of Prunetine on Malondialdehye [MDA] enzyme assay: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, where [a]*P<0.05*, [b]*P<0.01*,[c]*P<0.001* vs normal and [d]*P<0.05*, [e]*P<0.01*, [f]*P<0.001* vs diabetic control.

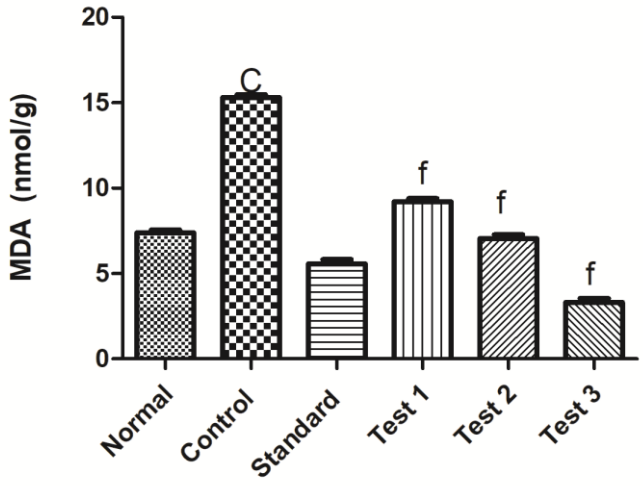

Figure 5: Effect of Prunetine on Malondialdehyde [MDA] Enzyme Assay: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

Table 7: Effect of Prunetin on lens protein content

S.No	Group	Soluble Protein [µg/mg]	Insoluble Protein [µg/mg]
1	I	467.2 ± 0.45	70.5 ± 4.3
2	II	162.7 ± 0.16^{c}	392.5 ± 33.1^{c}
3	III	425.0 ± 0.84	122.2 ± 9.3
4	IV	326.3 ± 0.28^{f}	128.4 ± 7.5^{f}
5	V	406.1 ± 0.27^{f}	142.4 ± 7.4^{f}
6	VI	411.4 ± 1.98^{f}	112.2 ± 8.1^{f}

Figure 7: Effect of prunetine on lens protein content: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

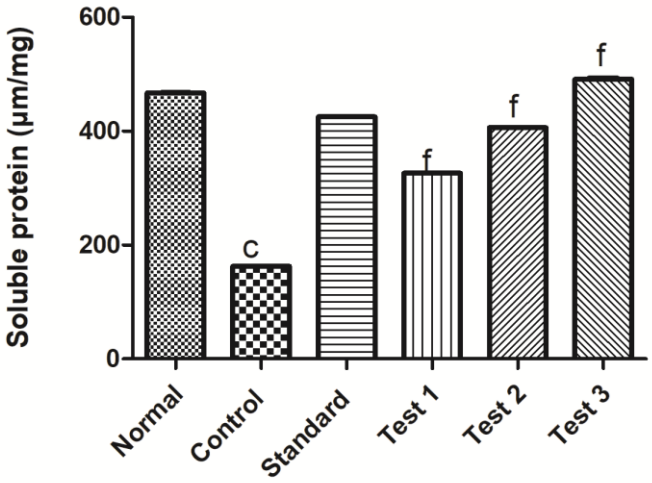

Figure 6: Effect of Protein on Lens Protein Content [Soluble protein]: Values are expressed as mean ± SEM [n=6]. Statistical analysis was accomplished using the one-way ANOVA followed by Newman-Keuls post hoc test, a vs normal [p<0.05]; b vs normal [p<0.01]; c vs normal [p<0.001]; d vs diabetic control [p<0.05]; e vs diabetic control [p<0.01]; f vs diabetic control [p<0.001].

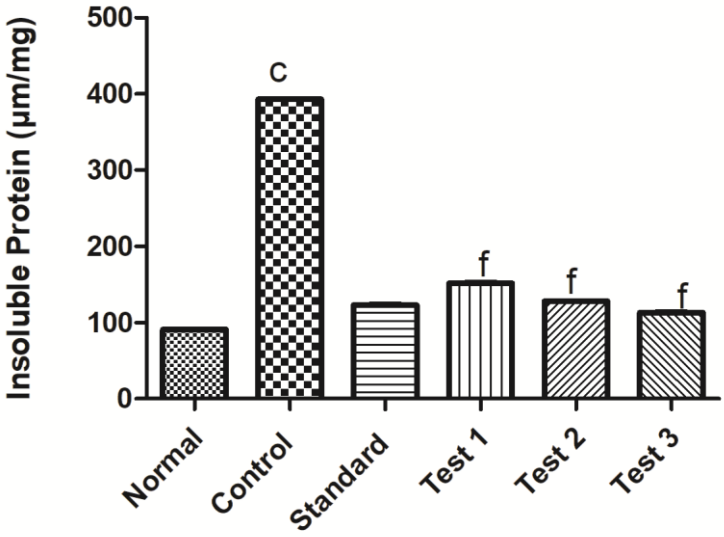

Figure 7: Lens Protein Content [Insoluble protein]

CONCLUSION

Prunetine effectively inhibits glucose-induced cataract formation in vitro, primarily by enhancing endogenous antioxidant levels, reducing lipid peroxidation, and preserving protein solubility. These findings underscore its potential as a candidate for non-surgical cataract therapy or prevention.The pharmacological evaluation of Prunetine reveals significant, dose-dependent anti-cataract activity across all assessed biochemical and visual lens parameters. Its efficacy was either comparable to or surpassed that of the standard antioxidant, ascorbic acid. Prunetine restored antioxidant enzyme activities [CAT, SOD, GSH], decreased lipid peroxidation [MDA], reduced opacity grading, and preserved lens protein solubility. These protective effects can be attributed to its potent ROS-scavenging and membrane-stabilizing properties. Moreover, Prunetine at 100 µg/ml demonstrated superior results across several parameters, thus proposing it as an effective candidate for further in vivo investigation and potential therapeutic development in cataract prevention.

Disclosure: "No Artificial intelligence or AI-assisted tools were used in the writing , data analysis, or creation of figures for the manuscript."

Author contribution statement: Bhumika Chandrakar has done Conceptualization, methodology, investigation, data curation, formal analysis, writing-original draft preparation. Swarnali Das Paul has performed experimental work, validation, data analysis, visualization, writing-review and editing. Rajesh Choudhary has performed methodology, supervision, project administration, writing-review and editing. Jaya Shree has done supervision, manuscript review and final approval.

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