AJND Reports
African Journal of Nutrition and Dietetics

https://doi.org/10.58460/ajnd.v5i01.185

RESEARCH ARTICLE

Evaluation of Spectral Transmission in Lens Tissue of Monosodium Glutamate-Induced Hyperglycemic Male Wistar Rats

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Submitted: 8th October 2025 | Accepted: 08th November 2025 | Published Online: 18th June 2026

ABSTRACT

Monosodium glutamate (MSG), a widely used flavor enhancer, is linked to metabolic and ocular complications, potentially increasing cataract risk. This study evaluated spectral transmission in lens tissue of MSG-induced hyperglycemic male Wistar rats to assess optical changes. Ten male Wistar rats were randomly assigned to control (distilled water) or experimental (750 mg/kg MSG daily for 28 days) groups (n = 5 each) using a random number table. Body weight and blood glucose were monitored. Lenses were extracted on day 36, homogenized, and analyzed for spectral transmission (250–700 nm) using UV-Visible spectrophotometry. Data were analyzed with ANOVA test $p < 0.05$) in GraphPad Prism. MSG-treated rats showed significant weight gain ($p < 0.05$) and hyperglycemia ($p < 0.05$) compared to controls. Lens transmittance was reduced ($p = 0.053$), and absorbance was increased ($p = 0.145$), suggesting protein aggregation. Chronic MSG exposure induces metabolic stress and may impair lens clarity in rats, potentially linked to cataract risk. Further studies on oxidative biomarkers are needed to confirm MSG's ocular toxicity, specifically by quantifying lens-specific oxidative stress markers (GSH/GSSG ratio, malondialdehyde, protein carbonyl content, 8-OHdG, and activities of SOD, CAT, GPx).

Keywords: *Monosodium glutamate, lens homogenate, spectral transmission, hyperglycemia, cataract, oxidative stress, Wistar rats, ocular toxicity*

How to Cite this Article: IGE, A., OJO, M. O., DEMEHIN, K. T., & NKOLLO, I. M. (2026). Evaluation of Spectral Transmission in Lens Tissue of Monosodium Glutamate-Induced Diabetic Rats. *African Journal of Nutrition and Dietetics*, 5(01). https://doi.org/10.58460/ajnd.v5i01.185

INTRODUCTION

Monosodium glutamate (MSG), the sodium salt of glutamic acid, is widely used in processed foods as a flavor enhancer, with daily intakes of 0.3–1.0 g in Western countries (Walker & Lupien, 2000). Studies have shown associations between MSG intake and metabolic disruptions in various populations (He et al., 2008, 2011). Research demonstrates that MSG induces metabolic disruptions, including obesity, hyperglycemia, and oxidative stress in rodents through mechanisms involving hypothalamic lesions, hyperlipidemia, and increased oxidative stress (Kazmi et al., 2017; Zanfirescu et al., 2019). MSG consumption has been linked to increased oxidative stress markers and inflammatory responses in multiple organ systems (El-Bakry et al., 2025; Kesherwani et al., 2024).

The ocular lens is critical for vision but susceptible to oxidative and metabolic stress. Cataracts remain a leading cause of global visual impairment (Bourne et al., 2013). Lens transparency requires both the orderly arrangement of lens cells and the high density and close packing of crystallin proteins (Shiels & Hejtmancik, 2019). In hyperglycemia, elevated blood glucose promotes glycation and aggregation of lens crystallins, reducing transparency and increasing cataract risk (Kumar & Cherian, 1996; Lyons et al., 1991; Swamy et al., 1993). Biochemical changes, particularly protein aggregation and post-translational modifications, impair lens function and contribute to cataract formation (Hains & Truscott, 2010; Truscott & Friedrich, 2019). MSG's induction of oxidative stress raises concerns about potential lens damage. However, data on MSG's direct effects on lens transparency in hyperglycemic models remain limited.

We hypothesized that chronic MSG exposure at 750 mg/kg reduces lens spectral transmission in male Wistar rats through hyperglycemia-induced protein aggregation and oxidative damage, potentially increasing cataract risk. This study evaluates spectral transmission in lens homogenates of MSG-induced hyperglycemic rats using UV-Visible spectrophotometry to assess optical changes

METHODS

Experimental Design

Rats were randomly assigned to two groups (n = 5 each) using a computer-generated random number table: Control group received distilled water orally while experimental group received MSG (Aji-no-moto, 99% purity) at 750 mg/kg body weight daily via oral gavage for 28 days. The MSG dose was selected based on studies showing metabolic effects in rodents, equivalent to high human dietary exposure (Sharma et al., 2013; Rahimi Anbarkeh et al., 2019). Body weight was measured weekly during acclimatization and on alternate days during treatment using a 500 g mechanical scale. Blood glucose was monitored using a glucometer and test strips.

Animals and Housing

Ten healthy male Wistar rats (80–100 g) were sourced from a certified animal farm in Ogbomosho, Oyo State, Nigeria, and housed in the Physiology Laboratory at the University of Ilorin under standard conditions (22 ± 2°C, 12-hour light/dark cycle). Rats had ad libitum access to grower mash and distilled water during a 7-day acclimatization period. Procedures were approved by the University of Ilorin Ethics Committee (approval no. UIL/ERC/2023/012) and adhered to the National Research Council's Guide for the Care and Use of Laboratory Animals (2011) and the 3Rs principle (Replacement, Reduction, Refinement).

MSG Preparation and Administration

MSG (15 g) was dissolved in 19.8 ml distilled water daily to prepare a fresh stock solution (He et al., 2008). The experimental group received 750 mg/kg MSG via oral gavage, while controls received an equivalent volume of distilled water.

Euthanasia and Lens Extraction

On day 36, after overnight fasting, rats were anaesthetized with intraperitoneal ketamine (50 mg/kg) and euthanized by cervical dislocation by trained personnel (National Research Council, 2011). Eyes were enucleated, and lenses were dissected via a posterior approach, rinsed in cold phosphate-buffered saline (PBS, 0.01 M, pH 7.4), and fixed in 10% phosphate-buffered formalin (Li et al., 2014).

Lens Homogenate Preparation

Lenses were homogenized in 180 µL cold PBS at 4°C using a dounce homogenizer. Homogenates were centrifuged at 10,000 × g for 10 minutes at 4°C, and supernatants were stored at −20°C until analysis (Donaldson et al., 2001).

UV-Visible Spectrophotometry

Lens homogenates were analyzed using a double-beam UV-Visible spectrophotometer (Analytik Jena) scanning from 250 to 700 nm. Parameters included scan speed (10.0 nm/s), integration time (0.1 s), measurement interval (1.00 nm), and lamp change wavelength (320 nm). A PBS-filled cuvette served as the blank, and the spectrophotometer was calibrated before each run. Three replicates per sample were analyzed, and absorbance/transmittance spectra were recorded to assess lens clarity (Hejtmancik & Shiels, 2015).

Ethical Considerations

All experimental procedures involving Wistar rats were conducted in strict accordance with the principles of the National Code of Health Research Ethics of Nigeria and the ARRIVE (Animal Research: Reporting of In Vivo Experiments) guidelines. Efforts were made to minimize animal suffering, including appropriate housing, handling, and humane endpoints.

Statistical Analysis

Data normality was confirmed using the Shapiro-Wilk test. Unpaired t-tests compared body weight, blood glucose, and lens transmittance between groups, with significance set at $p < 0.05$. Analysis of variance (ANOVA) assessed effect sizes (eta-squared, η^2). Analyses were performed using GraphPad Prism (version 9.0). Results were visualized using tables and graphs. A power analysis justified the sample size (n = 5 per group) to detect a 20% difference in transmittance with 80% power ($\alpha = 0.05$).

RESULTS

Body Weight and Blood Glucose

Administration of monosodium glutamate (MSG) induced significant metabolic alterations in the treated Wistar rats. As shown in Figure 1, the MSG-treated group exhibited a progressive and statistically significant increase in body weight compared to the control group throughout the experimental period ($p < 0.05$). The difference became more pronounced over time, consistent with the development of MSG-induced obesity in this model. Similarly, Figure 2 shows that fasting blood glucose levels were markedly elevated in the MSG group compared with the controls ($p < 0.05$). This hyperglycemia was evident at the end of the treatment period, suggesting impaired glucose homeostasis and potential early signs of insulin resistance or glucose intolerance associated with chronic MSG exposure.

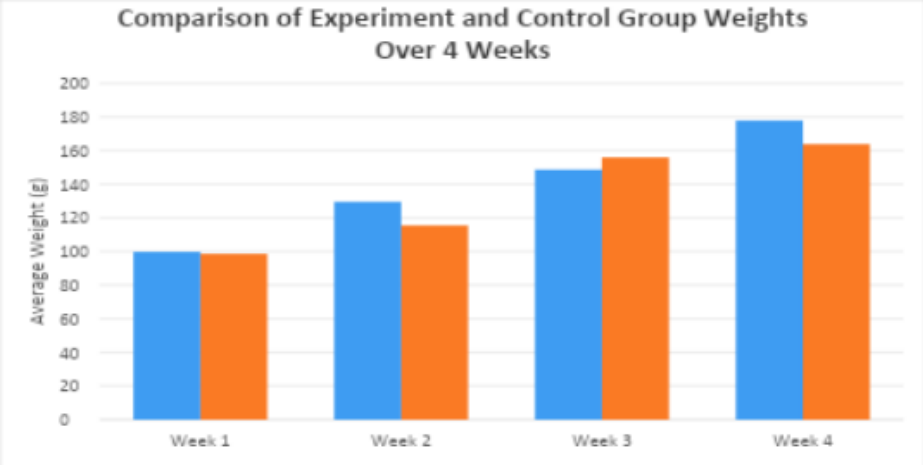

Figure 1: *Body weight gain in MSG-treated vs. control rats. The MSG group showed a significant weight increase ($p < 0.05$).*

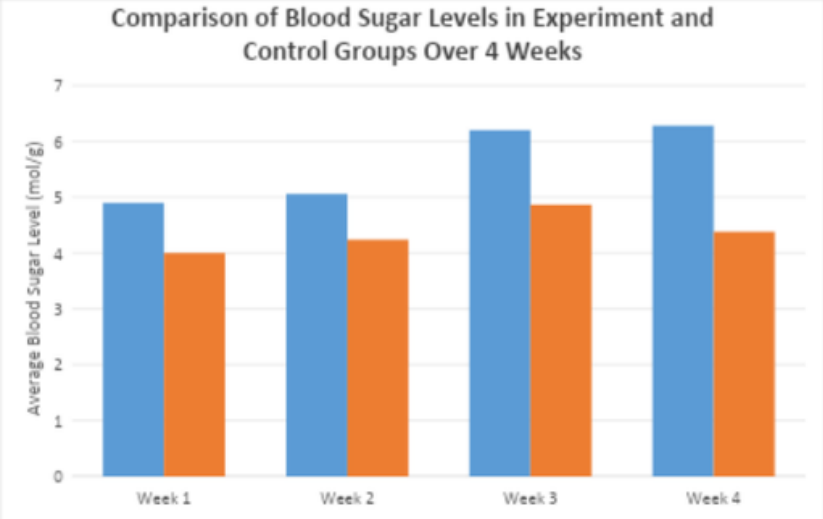

Figure 2: *Blood glucose levels in MSG-treated vs. control rats. The MSG group exhibited significant hyperglycaemia ($p < 0.05$).*

Lens Spectral Transmission

Effect of MSG on lens transmittance in Wistar rats

Figure 3 illustrates the impact of MSG treatment on the optical properties of the ocular lens. Lens transmittance in the MSG-treated group was significantly reduced ($p < 0.05$) across the entire measured spectrum from 250 to 700 nm when compared to the control group. This reduction indicates compromised lens transparency, which may impair light passage and contribute to visual dysfunction.

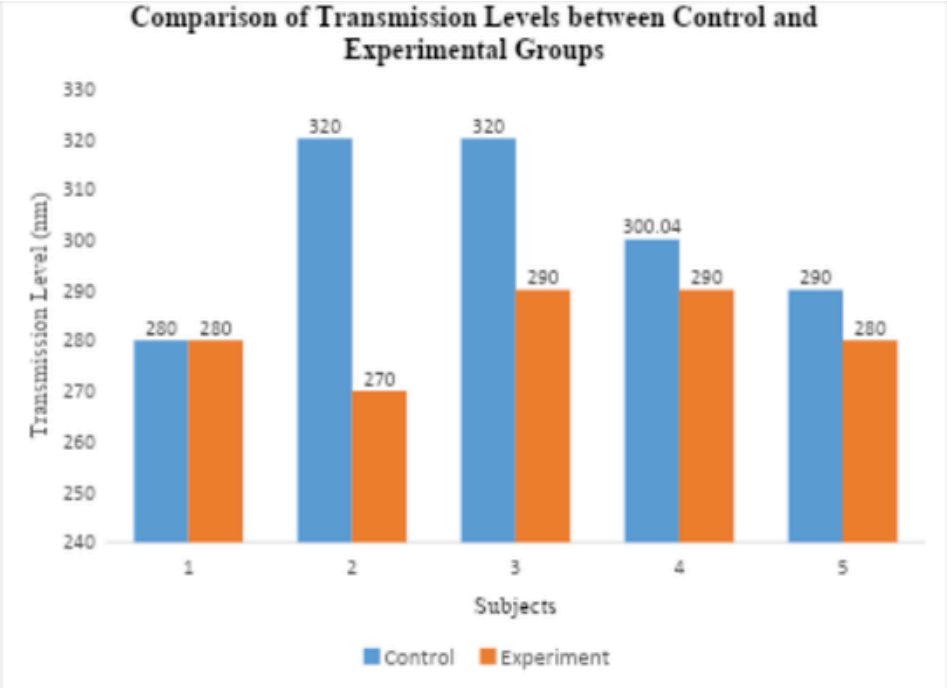

Figure 3: *Lens transmittance in the MSG group was significantly reduced ($p < 0.05$) across the 250–700 nm spectrum compared to controls.*

Effect of MSG on lens absorption in Wistar rats

As presented in Figure 4, lens absorption was significantly increased ($p < 0.05$) in the MSG-treated rats across the 250–700 nm wavelength range relative to the control animals. The heightened absorption suggests biochemical or structural changes in the lens tissue (such as protein modifications or oxidative damage) that could underlie the observed decrease in transmittance.

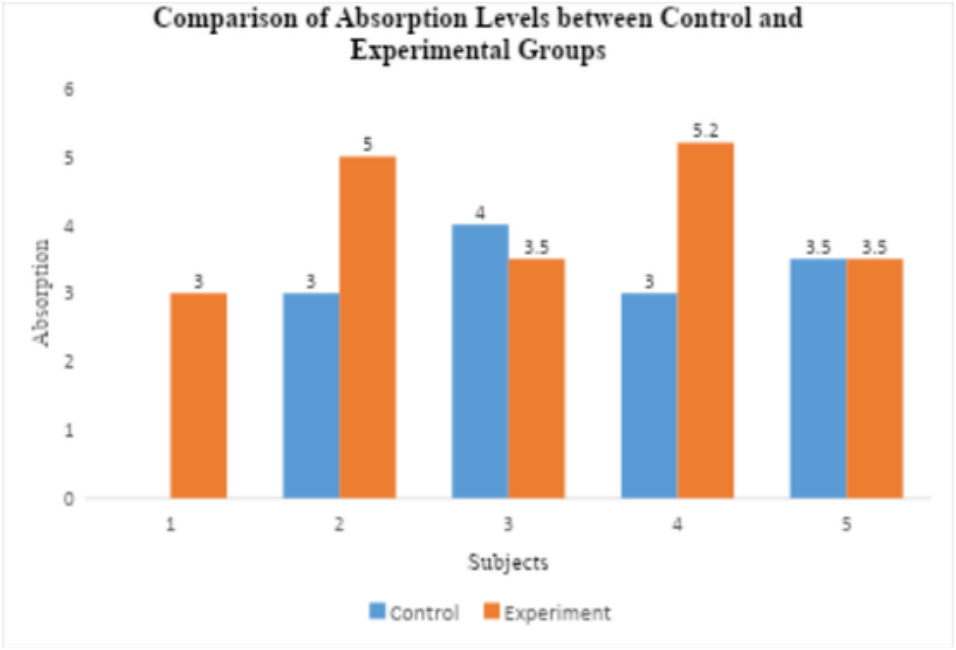

Figure 4: *Lens absorption in the MSG group was significantly increased ($p < 0.05$) across the 250–700 nm spectrum compared to controls.*

DISCUSSION

Chronic monosodium glutamate (MSG) administration at 750 mg/kg for 28 days induced significant metabolic changes in male Wistar rats, including weight gain and hyperglycemia ($p < 0.05$), consistent with prior reports demonstrating that MSG consumption is associated with metabolic dysregulation in both animal models and human populations (He et al., 2008, 2011; Kazmi et al., 2017). The observed reduction in lens transmittance ($p = 0.053$) and increased absorbance ($p = 0.145$) suggest protein aggregation, likely due to hyperglycemia-induced glycation and oxidative stress, processes known to impair lens clarity through accumulation of covalent modifications including deamidation, oxidation, and glycation that destabilize crystallin proteins (Hains & Truscott, 2010; Kumar & Cherian, 1996; Truscott & Friedrich, 2019). MSG administration has been demonstrated to elevate oxidative stress markers, increase lipid peroxidation, and induce inflammatory responses in multiple organ systems (El-Bakry et al., 2025; Kesherwani et al., 2024), which may contribute to reactive oxygen species (ROS) generation that oxidizes lens crystallins and reduces transparency.

The lens transparency depends on maintaining native tertiary structures and high solubility of crystallin proteins, and protein aggregation is the primary cause of cataract, the leading cause of blindness worldwide (Shiels & Hejtmancik, 2019). In diabetic and hyperglycemic conditions, elevated glucose promotes non-enzymatic glycation of lens crystallins, leading to the formation of advanced glycation end products (AGEs) that induce protein cross-linking, aggregation, and loss of transparency (Kumar & Cherian, 1996; Lyons et al., 1991; Swamy et al., 1993). Our findings of reduced transmittance align with established mechanisms of cataract formation wherein oxidative damage, glycation, and other post-translational modifications lead to partially unfolded, aggregation-prone crystallin intermediates that form insoluble, light-scattering aggregates (Hains & Truscott, 2010; Truscott & Friedrich, 2019).

These findings suggest a potential link between MSG exposure and lens opacity in rats, but human implications require cautious interpretation. MSG has been shown to induce metabolic and oxidative stress in animal models (El-Bakry et al., 2025; Kesherwani et al., 2024), and given cataracts' substantial global burden as a leading cause of vision loss (Bourne et al., 2013), dietary MSG may pose a risk for individuals predisposed to hyperglycemia. However, the relationship between MSG consumption and metabolic outcomes in human populations remains controversial, with some studies finding associations with overweight (He et al., 2008, 2011) while comprehensive reviews note the need for careful evaluation of safety evidence (Kazmi et al., 2017; Zanfirescu et al., 2019). Furthermore, causality cannot be inferred from this study alone, as the rodent model, dose administered (750 mg/kg), and duration of exposure may not directly translate to human dietary consumption patterns.

In summary, chronic MSG exposure induces metabolic stress and may impair lens clarity in rats through mechanisms involving hyperglycemia, oxidative stress, and protein aggregation, suggesting a potential association with cataract risk. The progression from initial glycation events to the formation of high-molecular-weight crystallin aggregates, as documented in diabetic cataract models (Lyons et al., 1991; Swamy et al., 1993), provides a mechanistic framework for understanding how MSG-induced hyperglycemia could contribute to lens damage. These findings highlight the need for further research on MSG's ocular safety in humans, particularly longitudinal studies examining lens health in populations with varying levels of MSG consumption and in individuals with pre-existing metabolic conditions that may increase vulnerability to lens damage.

Limitations

The small sample size (n=5 per group) may limit statistical power, though a power analysis supported detecting a 20% transmittance difference. The 28-day exposure may not reflect chronic human consumption. The absence of oxidative stress biomarkers (e.g., malondialdehyde) restricts mechanistic insights. Future studies should use larger samples, both sexes, longer exposures, and measure biomarkers like superoxide dismutase or crystalline gene expression to elucidate MSG's ocular toxicity.

Acknowledgements

We thank the Department of Optometry and Vision Science, University of Ilorin, Nigeria, for providing laboratory facilities, the Physiology Laboratory staff for technical assistance, and the certified animal farm in Ogbomosho, Oyo State, for supplying Wistar rats. No external funding was received.

Conflict of Interest

The authors declare no conflict of interest.

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