East African Scholars Journal of Medical Sciences
Abbreviated Key Title: East African Scholars J Med Sci
ISSN: 2617-4421 (Print) & ISSN: 2617-7188 (Online)
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

Volume-8 | Issue-8 | Aug-2025 | DOI: https://doi.org/10.36349/easms.2025.v08i08.004

Original Research Article

Neurotransmitter and Neurotrophic Effects of Lutein on Scopolamine-Induced Depression in Male Wistar Rats

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Article History
Received: 12.06.2025
Accepted: 16.08.2025
Published: 20.08.2025

Journal homepage:
https://www.easpublisher.com

Quick Response Code

Abstract: This study was carried out to investigate the physiological processes through which lutein affects anxiety and depression behavior in Scopolamine-treated animal models. 30 male Wistar rats (105g-153g) were randomly assigned to six groups. Group 1: Negative Control, Group 2: Scopolamine only treated group, Group 3: Scopolamine + Lutein (20mg/kg), Group 4: Scopolamine + Lutein (40mg/kg), Group 5: Scopolamine + Lutein (60mg/kg), Group 6: Scopolamine + Imipramine (standard drug). scopolamine was administered intraperitoneally and lutein orally. The data was analyzed using one way ANOVA followed by Post hoc Fischer's LSD. Multiple comparison values were considered significant at $P<0.05$ Results. The administration of Scopolamine led to decreased acetylcholinesterase (AChE) concentration in Group 2 ($P < 0.0001$) and Group 3 ($P < 0.01$) when compared to the control group. The combined use of lutein and imipramine preserved Acetylcholinesterase levels between Groups 4, 5 and 6 after brain treatment. Brain-derived neurotrophic factor (BDNF-α) experienced a significant decrease in Groups 2 and 3 ($P < 0.0001$) together with Group 4 ($P < 0.001$) whereas the levels in Groups 5 and 6 remained comparable to the control. Nitric oxide (NO) levels significantly increased in Group 2 ($P < 0.01$) but remained unchanged in Groups 3-6 compared to the control. The findings established that while scopolamine negatively affected AChE and BDNF-α levels, lutein and imipramine mitigated these effects. Scopolamine caused a decrease in neurotrophic factors (BDNF-α, AChE) and also increased the level of oxidative marker (NO) and depressive like behaviors. Lutein treatment, at moderate (40 mg/kg) and high (60 mg/kg) doses effectively normalized these biomarkers and augmented neurotrophic signaling.

Keywords: Anti-Depressant, Lutein, Scopolamine-Induced, Depression, Anxiety, Brain-Derived Neurotrophic Factor.

INTRODUCTION

Researchers have chosen the xanthophyll carotenoid known as lutein for neuropsychiatric investigation because of its strong antioxidant and anti-inflammatory capabilities that occur primarily in green leafy vegetables and yellow-orange fruits (Bhat & Mamatha, 2021). Research shows that lutein traditionally functions as an agent for eye health protection (O'Brien, 2018) but experts now recognize its capability for neurological defense. Navinder Singh (2024) describes depression as a complex neuropsychiatric disorder which creates disturbances in neurotransmitter events and impairs synaptic plasticity (Pap *et al.,* 2021). Researchers have started investigating natural substances including lutein because this compound shows the ability to modify neurotransmitter regulation while supporting neuronal development. Research laboratories frequently use the muscarinic acetylcholine receptor antagonist scopolamine as a preclinical tool to simulate depression and cognitive impairment because it creates scientific findings comparable to depressive behavioural and biochemical alterations (Hasselmann, 2014).

The multiple components of depression pathophysiology include oxidative stress combined with neuroinflammation together with deficits in cholinergic neurotransmission (Correia *et al*., 2023). Experimental scopolamine models enable research of these mechanisms because scopolamine blocks acetylcholine which plays a fundamental role in cognitive and mood processes (Barak & Weiner, 2006; Suryanarayanan,

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2014). Experimental design using these models allows researchers to replicate brain deficits which also produces depressive behavior symptoms while diminishing brain-derived neurotrophic factor (BDNF) quantities that support synaptic plasticity and neuronal maintenance. Scientists deem scopolamine useful for evaluating potential antidepressant drugs due to its properties (Drevets *et al*., 2020; Jaffe *et al*., 2013).

Studies show that lutein protects the nervous system through its various impact mechanisms (Zhang *et al*., 2015). Lutein acts as a powerful antioxidant to destroy free radicals that cause neuronal damage as well as neurodegenerative disorders (Pap *et al*., 2021). The anti-inflammatory mechanism of lutein uses its ability to control pro-inflammatory cytokines thus decreasing neuroinflammation which represents a crucial characteristic of depressive disorders according to Prathyusha *et al*., 2025; Pap *et al*., 2021. The antioxidative together with anti-inflammation mechanisms enable the protection of both neuronal structure and operational capacity (Ahn & Kim, 2021). The ability of lutein to control cholinergic neurotransmitter networks makes it a significant therapeutic opportunity for reversing scopolamine-caused neurochemical deterioration. Acetylcholine quality and function improvement through lutein consumption counteracts the cholinergic deficiencies caused by scopolamine treatment according to research by Li *et al*., 2024.

Research demonstrates that lutein stimulates neurotrophic support according to Yalcin Gunal *et al*., (2021). The neurotrophin BDNF which is crucial for synaptic plasticity and hippocampal neurogenesis becomes depleted when people have depressive symptoms or are treated with scopolamine (Miranda *et al*., 2019). Research shows that lutein strengthens BDNF expression which suggests its effectiveness in improving neuronal plasticity for treatment of depressive symptoms (Parekh *et al*., 2024). Neurotoxicity along with stress threatens the hippocampus specifically because this brain region serves as the foundation for memory and emotion regulation (Lieblein-Boff *et al*., 2015). Lutein improves scopolamine-related mood deterioration and cognitive impairment by elevating BDNF levels while enhancing hippocampal functionality according to Patel *et al*., (2021).

Wistar rats serve as a popular research model in preclinical studies because scientists understand their biological processes and behavioral reactions which enables studies about how lutein affects scopolamine-induced depression (Patel *et al*., 2024). The combination of laboratory evaluations and biochemical examinations of neurotransmitters together with oxidative stress parameter analysis and BDNF assessment gives researchers detailed knowledge about how lutein functions as an antidepressant (Scapagnini *et al*., 2012).

Studies in development have demonstrated that lutein effectively reduces the neurochemical and behavioral effects caused by scopolamine administration. Research conducted by Patel *et al*., (2021) established that supplementing the diet with lutein elevated thinking abilities while minimizing oxidative damage thus recognizing its capability to serve as a cognitive improvement agent. Research by Liu *et al*., (2022) established that lutein increased BDNF production in hippocampal regions while improving depressive symptoms within rodent subjects. Laboratory research proves that lutein acts as an important factor which alters critical biological mechanisms associated with depression and cognitive impairment.

The investigation uses Wistar rats to dissect how lutein regulates neurotransmitters and neurotrophic effects during scopolamine depression. The study investigates biochemical and behavioral markers to explain how lutein interacts with scopolamine-induced pathophysiology as a neuroprotective agent. The research findings will expand the current body of evidence regarding lutein as a therapeutic treatment for neuropsychiatric disorders. The research maintains compatibility with the fundamental mission of locating secure natural substances possessing complex effects for serving either independently or as backup to classic antidepressant medicines.

The scientific understanding of how lutein works has been enhanced through this research which provides the foundation for its possible utilization in treating depression and associated disorders. Additional research about applying this translational knowledge is needed to achieve full therapeutic uses of lutein for neuropsychiatric treatment.

MATERIALS AND METHODS

Experimental Animals

Experimental rats were purchased from the animal house of the Faculty of Basic medical sciences, Abuja campus, University of Port Harcourt. The animals were housed in steel cages and kept at room temperature. The rats had no history of drug consumption, that is; they had not been used for any investigation. The rats were put on standard rat pellet (feed) and pure drinking water and allowed to get acclimatized for 21 days before the start of the experiment. The study was done in accordance with the guidelines for animal use of the Faculty of Basic Medical Sciences, University of Port Harcourt.

Ethical Approval

Ethical approval was obtained from the faculty of basic medical science, Abuja campus, University of Port Harcourt. Rat handling and treatment conform to the guideline of the National Research Council (2011) for care and use of laboratory animals.

Chemicals and Reagents

The chemicals and reagents used for this study were purchased from GGI Intl' Nigeria Ltd. located at GGI Place, Plot 8 GGI Crescent, (Opp. Mikab Filling Station), Port Harcourt, Rivers State, Nigeria. The chemicals and reagents used are:

a. Scopolamine hydrobromide: Used to induce depression-like behavior.
b. Lutein: Administered as the test compound.
c. Imipramine hydrochloride (10 mg/kg) (standard antidepressant drug)
d. Acetylcholine esterase assay kit: For cholinergic function assessment.
e. Brain-derived neurotrophic factor (BDNF-α) ELISA kit: For neurotrophic signaling evaluation.
f. Nitric oxide assay kit: Used for oxidative stress assessment.

Experimental Design

Thirty (30) male wistar rats were weighed and allocated into six different groups, each containing five wistar rats. The impact of lutein supplementation on scopolamine-induced anxiety and depression like behavior in rats, and the physiological mechanism by which lutein may modulate scopolamine-induced anxiety and depression like behavior in rats was checked, thirty male wistar rats were weighed and allocated into five groups of five rats each. The groups were designated as groups 1, 2, 3, 4, 5 and 6.

The experimental groups were administered different doses of scopolamine and lutein as follows;

- Group 1 (negative control group) was given feed and 250mg/kg of distilled water only
- Group 2 (positive control group) was administered 0.06ml of scopolamine
- Group 3 (low dose group) was administered with 0.056ml of scopolamine and was treated with 0.026ml of lutein.
- Group 4 (medium dose group) and was administered with 0.055ml of scopolamine and treated with 0.07ml of lutein.
- Group 5 (high dose group) and was administered with 0.054ml of scopolamine and was treated with 0.15ml of lutein.
- Group 6 (standard drug group) and was given 0.096ml scopolamine 30 mins before being administered with 0.035ml of imipramine.

Scopolamine was induced intraperitoneally into the rats and dissolved lutein capsule was used to treat the effect of brain toxicity. Group 6 were given scopolamine 30 mins before imipramine was administered intraperitoneally; this was done to check for the prophylactic attributes of scopolamine against imipramine.

Lutein Administration: Lutein was given orally to the designated groups for a specified period daily. Doses were chosen based on previous research and human dietary intake levels.

Scopolamine-Induced Anxiety and Depression: All experimental groups (except the negative control) received scopolamine to induce anxiety and depression following established protocols.

Exposure of Animals to Test Substances

Animal Acclimatization: Upon arrival, the rats were weighed, identified, and housed in wire-mesh cages with sawdust bedding for dryness.

They were allowed to acclimate for one week under standard laboratory conditions:
Temperature: 18-26°C (64-79°F)
Light/Dark Cycle: 12 hours each

After acclimatization and toxicity test, the rats were weighed, and the weight was between 105g-153g. The Wistar rats were allocated into six groups. Administration of lutein was done orally using oral cannula and administration of scopolamine was done Intraperitoneally using a I ml syringe.

Collection of Blood Samples

After 14 days of administration, prior to the termination of the experiment, the rats were weighed and their weight was recorded. Blood was obtained through jugular vein after the rats were anesthetized and put into heparinized bottles and blood samples (Serum) was analyzed for biochemical parameters (Neurotransmitter, Neuropeptide, Protein and Lipid)

Statistical Analysis

Data were analyzed using the Graph Pad prism 8 software, and Results were expressed as mean ± SEM. Comparisons was done by using One-way Analysis of Variance (One-way ANOVA) followed by Newman-Keuls'post hoc multiple comparison test.

RESULTS AND DISCUSSION

The experimental groups in this study were categorized as follows:

Group 1: Negative Control
Group 2: Scopolamine only treated group
Group 3: Scopolamine + Lutein (20mg/kg)
Group 4: Scopolamine + Lutein (40mg/kg)
Group 5: Scopolamine + Lutein (60mg/kg)
Group 6: Scopolamine + Imipramine (10mg/kg)

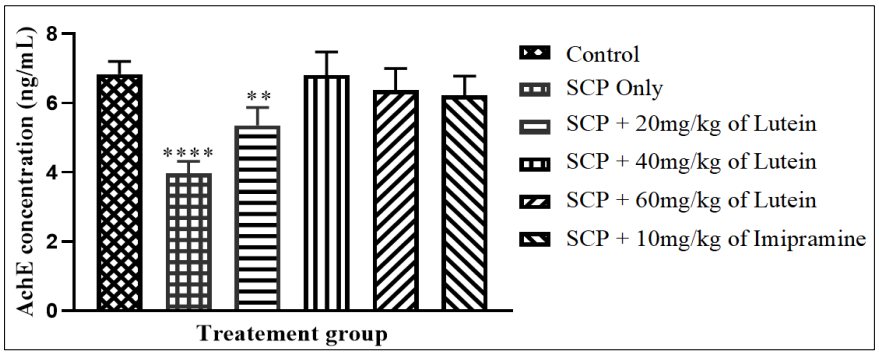

Figure 1: Graphical Representation of the Acetyl Choline Concentration

The columns represent the mean values and error bars indicate the standard deviation (SD) of our independent experiments. CTRL = Control, SCP = Scopolamine, STD = Standard drug. * indicates statistical significance of scopolamine and treatment versus the negative controls. **significant at P<0.01, and **** significant at 0.0001.

There was a highly significant decrease in AchE concentration in group 2 (P<0.0001) and significant difference in group 3 (P < 0.01) when compared with the control group. There was no significant difference in AchE concentration in groups 4, 5, and 6, when compared to the control group. This suggests that while scopolamine administration reduces AchE concentration, luteinand imipramine elev ate the concentration respectively.

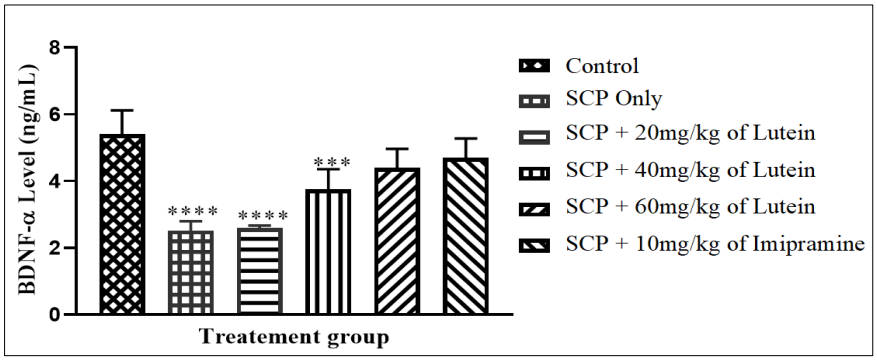

Figure 2: Graphical Representation of the Brain-Derived Neurotrophic Factor Level

The columns represent the mean values and error bars indicate the standard deviation (SD) of our independent experiments. CTRL = Control, SCP = Scopolamine, STD = Standard drug * indicates statistical significance of scopolamine and treatment versus the negative controls. *** significant at P<0.001 and **** significant at 0.0001.

There is a very significant decrease in BDNF-α LEVELS (ng/mL) in group 2 and 3 (P<0.0001) when compared with the control group. There was also a very significant decrease in BDNF-α LEVELS (ng/mL) in group 4 (P<0.001) when compared with the control group. In groups 5 and 6, BDNF-α LEVELS (ng/mL) showed no significant difference when compared with the control group.

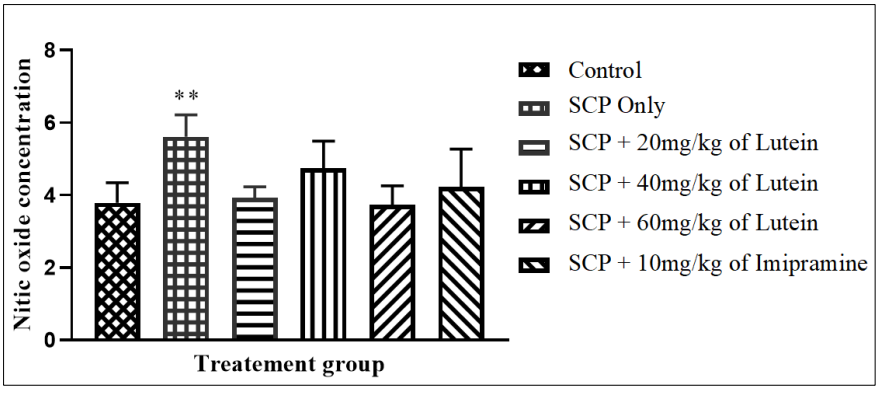

Figure 3: Graphical Representation of the Nitric Oxide Concentration

The columns represent the mean values and error bars indicate the standard deviation (SD) of our independent experiments. CTRL = Control, SCP = Scopolamine, STD = Standard drug * indicates statistical significance of scopolamine and treatment versus the negative controls. **significant at P<0.01.

There is a significant rise in NO levels in group 2 ($P < 0.01$) when compared to the control group. Also, there was no significant difference in NO concentration in groups 3, 4, 5 and 6 in comparison with the control group.

DISCUSSION

Figure 1: Acetylcholine Esterase (AChE) Concentration

The group treated with scopolamine only also had a reduced AChE level, which is indicative of cholinergic dysfunction ($P < 0.0001$). In fact, the elevated dose of lutein restored the AChE level to nearly control value, thus underlining its role in the cholinergic neurotransmission process. This is in agreement with (Tonin *et al*., 2019) who suggested that Lutein has cholinergic enhancing properties.

Figure 2: Brain-Derived Neurotrophic Factor (BDNF-α) Levels

There was a significant decrease in the BDNF-α signaling in the scopolamine-only group ($P < 0.0001$). Lutein at medium and high dosage returned BDNF-α to control levels, hence the neurorestorative effect of Lutein. This is in accordance with Nataraj *et al*., (2015) who pointed out that Lutein increases neurotrophic factor expression.

Figure 3: Nitric Oxide (NO) Concentration

The NO levels of the scopolamine-only group were significantly higher than the baseline ($P < 0.01$) indicating increased oxidative stress. All doses of Lutein restored NO levels to normal, which supports the drug's antioxidative effect.

CONCLUSION

The findings of the present study support the neuroprotective-and antidepressant-like role of Lutein for the amelioration of scopolamine-induced depression like behavior in Wistar rats. The behavioral and biochemical changes induced by scopolamine, which is well known for its effects on oxidative stress, cholinergic dysfunction and neurotrophic factors were significant. Lutein reduced these effects in a dose-dependent manner by elevating antioxidant enzyme activity (GSH, SOD, and catalase), increasing levels of AChE and normalizing BDNF-α expression.

The forced swimming test (FST), tail suspension test (TST), and open field test (OFT) also supported the ability of Lutein in reducing depression-like behavior. In the tests, Lutein was shown to be highly potent with high doses giving results that were almost as good as imipramine, a drug used in the treatment of depression. This means that the antioxidative, anti-inflammatory and neurotrophic-enhancing effects of Lutein play a major role in its therapeutic value.

The present study demonstrates that Lutein has the potential to enhance cognitive and behavioral functions, and reduce scopolamine-induced neurotoxic effects, which suggest that Lutein could be a safe and natural adjunct or an add-on therapy in the management of depressive disorders and neurodegenerative diseases. The impact on oxidative stress and neurotrophic signaling pathways indicates that it may have potential uses in other ailments that are characterized by impaired cognition and neuronal damage.

These outcomes are in consonance with the findings of Nataraj *et al*., (2015), Ahn and Kim (2021) and Tonin *et al*., (2019) who have shown that Lutein has potential to restore mitochondrial dysfunction, oxidative stress and cholinergic deficits. Due to the fact that depression and related conditions involve multiple pathways, Lutein can be a good candidate due to its activity on several pathways.

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Cite This Article: Austin A. Ajah, Ruth Obomanu-Tamunotonjo, Chike CPR (2025). Neurotransmitter and Neurotrophic Effects of Lutein on Scopolamine-Induced Depression in Male Wistar Rats. *East African Scholars J Med Sci, 8*(8), 329-334.