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Mitigation of atrazine-induced metabolic and oxidative dysregulation by *Ocimum sanctum*: An integrated experimental study

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ABSTRACT

Environmental exposure to herbicides such as atrazine has emerged as a significant toxicological concern due to its persistence and systemic effects in mammals. The present investigation provides an integrated evaluation of atrazine-induced biochemical, oxidative, and histological alterations and examines the modulatory role of *Ocimum sanctum*. Experimental animals were subjected to sub-chronic atrazine exposure, with parallel intervention using Tulsi extract at two graded doses. Atrazine administration resulted in pronounced metabolic imbalance, oxidative stress, and tissue degeneration, reflected in altered glucose, lipid, protein, and enzymatic profiles. Supplementation with *Ocimum sanctum* significantly attenuated these alterations, restoring physiological homeostasis in a dose-responsive manner. The findings highlight the central role of oxidative stress in atrazine toxicity and establish *O. sanctum* as a potent natural modulator of toxicant-induced dysfunction.

Key Words - *Ocimum sanctum*, Atrazine, physiological homeostasis,triazine

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INTRODUCTION

Atrazine is one of the most widely used triazine herbicides globally, known for its persistence in soil and water and its potential to affect non-target organisms (Ruchi & Yadav, 2023; Sharma *et al*., 2019). Its extensive agricultural application has led to continuous environmental exposure, raising concerns regarding its toxicological effects in mammals.

At the cellular level, atrazine induces the generation of reactive oxygen species (ROS), leading to oxidative stress and subsequent damage to lipids, proteins, and nucleic acids (Lim, 2005). These alterations are often reflected in metabolic disturbances such as changes in glucose, lipid, and protein metabolism.

Medicinal plants have gained attention as protective agents against chemical toxicity. *Ocimum sanctum* (Tulsi) is widely recognized for its antioxidant, anti-inflammatory, and hepato protective properties (Gupta *et al.*, 2002; Pattanayak *et al*., 2010). Its phytochemical constituents, particularly flavonoids and eugenol, contribute to its ability to neutralize free radicals and enhance endogenous antioxidant defenses.

The present study evaluates atrazine-induced toxicity and investigates the protective potential of *O. sanctum* through biochemical, oxidative, and histopathological assessments.

MATERIALS & METHODS

Experimental Animals

Healthy adult albino mice (*Mus musculus*) were used in the present study. The animals were acclimatized for one week prior to experimentation and maintained under standard laboratory conditions, including a temperature of 22 ± 2°C, relative humidity of 50–60%, and a 12 h light–dark cycle. Animals were provided with standard pellet diet and water *ad libitum*.

Experimental Design

Animals were randomly divided into four groups (n = 6 per group):

- Group I (Control): Normal diet and water
- Group II (Atrazine): Atrazine only
- Group III (Atrazine + *O. sanctum* 100 mg/kg)
- Group IV (Atrazine + *O. sanctum* 200 mg/kg)

Preparation of *Ocimum sanctum* Extract

Fresh leaves of *O. sanctum* were collected, washed, air-dried, and finely chopped. Aqueous extraction was carried out by boiling in distilled water, followed by filtration using Whatman No. 1 filter paper. The extract was freshly prepared for administration.

Chemical Treatment

Atrazine (analytical grade) was dissolved in distilled water and administered orally at a predetermined dose based on earlier studies to induce sub-chronic toxicity.

Sample Collection

At the end of the experimental period, animals were fasted overnight and sacrificed under ethical conditions. Blood samples were collected via cardiac puncture and centrifuged at 3000 rpm for 15 minutes to obtain serum. Liver and kidney tissues were excised and processed for biochemical and histopathological analyses.

Biochemical and Antioxidant Assays

Serum biochemical parameters including glucose, cholesterol, total protein, and acid phosphatase (ACP) were estimated using standard methods. Oxidative stress markers such as lipid peroxidation (MDA), superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH), and glutathione peroxidase (GPx) were also analyzed following established protocols.

Statistical Analysis

Data were expressed as mean ± standard error (SE). Statistical analysis was performed using one-way ANOVA followed by appropriate post hoc tests. A p value < 0.05 was considered statistically significant.

RESULTS

Physiological and Growth Responses

Atrazine exposure resulted in reduced locomotor activity, poor grooming, and decreased food intake. Body weight gain was significantly reduced, whereas *O. sanctum* treatment improved growth in a dose-dependent manner (Table 1).

Table 1. Effect of Atrazine and *O. sanctum* on Body Weight Gain (%)

Group	Treatment	Body Weight Gain (%)
Control	—	20.3
Atrazine	—	3
Atrazine + 100 mg/kg	—	12.1
Atrazine + 200 mg/kg	—	16.8

Metabolic Alterations

Atrazine exposure caused hyperglycemia, hypercholesterolemia, and reduced protein levels along with increased ACP activity. These alterations indicate metabolic imbalance and hepatic dysfunction. *O. sanctum* treatment restored these parameters in a dose-dependent manner.

Table 2. Effect of Atrazine and *O. sanctum* on Serum Biochemical Indicators

Parameter	Control	Atrazine	Atrazine + 100 mg/kg	Atrazine + 200 mg/kg
Serum Glucose mg/dL	90.6 ± 2.5	140.2 ± 4.0	114.1 ± 3.6	99.2 ± 3.0
Total Cholesterol mg/dL	122.9 ± 3.6	178.4 ± 5.3	148.2 ± 4.5	135.1 ± 4.1
Total Protein g/dL	7.28 ± 0.17	5.78 ± 0.15	6.61 ± 0.18	7.02 ± 0.16
ACP IU/L	11.6 ± 0.4	22.1 ± 0.7	16.5 ± 0.5	13.9 ± 0.5

Oxidative Stress and Antioxidant Defense

Atrazine significantly increased lipid peroxidation and reduced antioxidant enzyme activity. Treatment with *O. sanctum* restored antioxidant status and reduced oxidative stress (Table 3).

Table 3. Effect of Atrazine and *O. sanctum* on Oxidative Stress Markers

Parameter	Control	Atrazine	Atrazine + 100 mg/kg	Atrazine + 200 mg/kg
MDA nmol/mg protein	2.05 ± 0.09	4.78 ± 0.16	3.28 ± 0.11	2.52 ± 0.10
SOD U/mg protein	8.9 ± 0.3	5.0 ± 0.2	6.7 ± 0.3	8.0 ± 0.3
CAT µmol/ min/mg protein	56.2 ± 1.9	32.8 ± 1.6	46.1 ± 1.8	52.4 ± 1.7
GSH nmol/mg protein	8.5 ± 0.2	4.4 ± 0.2	6.1 ± 0.2	7.4 ± 0.3
GPx U/mg protein	13.2 ± 0.4	7.6 ± 0.3	10.1 ± 0.4	11.9 ± 0.4

Histopathological Alterations

Severe liver and kidney damage was observed in atrazine-treated animals, while *O. sanctum* treatment improved tissue architecture in a dose-dependent manner (Table 4).

Table 4. Histopathological Observations

Group	Liver	Kidney
Control	Normal	Normal
Atrazine	Necrosis, fatty changes	Tubular damage
Atrazine + 100 mg/kg	Mild damage	Partial recovery
Atrazine + 200 mg/kg	Near normal	Near normal

DISCUSSION

The present study demonstrates that sub-chronic exposure to atrazine induces significant physiological, biochemical, and histopathological alterations in experimental animals, primarily mediated through oxidative stress and metabolic dysregulation. The observed reduction in body weight and general activity reflects systemic toxicity and impaired energy metabolism. Similar findings have been reported in pesticide-exposed models, where reduced growth is attributed to altered nutrient utilization and metabolic stress (Kori *et al.,* 2019).

The significant elevation in blood glucose levels in atrazine-treated animals indicates disruption of carbohydrate metabolism. This hyperglycemic condition may be linked to oxidative damage to pancreatic β-cells or interference with insulin signaling pathways. Comparable results have been reported by Seth *et al.* (2011), who demonstrated that toxicant-induced oxidative stress can impair glucose homeostasis and insulin function.

Alterations in lipid metabolism were evident from the increased serum cholesterol levels following atrazine exposure. This may be attributed to hepatic dysfunction and impaired lipid clearance, as the liver plays a central role in cholesterol metabolism. Similar elevations in cholesterol have been documented in pesticide-induced toxicity, suggesting a common mechanism involving oxidative damage to hepatic tissues (Kori *et al.,* 2019). The restoration of cholesterol levels following *Ocimum sanctum* treatment indicates its potential role in stabilizing hepatic function and regulating lipid metabolism.

The decline in plasma protein levels further supports the presence of hepatic injury, as protein synthesis is primarily regulated by hepatocytes. Reduced protein content may result from impaired transcriptional and translational processes due to toxic insult. In parallel, the elevation of acid phosphatase (ACP) activity observed in the atrazine group indicates lysosomal membrane destabilization and cellular damage. These findings are consistent with earlier reports highlighting enzyme leakage as a marker of tissue injury (Manjunatha & Srinivasan, 2006).

Oxidative stress appears to be a central mechanism underlying the toxic effects of atrazine. The significant increase in lipid peroxidation, as indicated by elevated MDA levels, reflects enhanced membrane damage due to reactive oxygen species (ROS). Concurrently, the reduction in antioxidant enzymes such as superoxide dismutase, catalase, glutathione, and glutathione peroxidase indicates depletion of the endogenous defense system. These results are in agreement with Lim (2005), who emphasized the role of oxidative stress in pesticide-induced cellular damage.

Regulation of *Ocimum sanctum* extract significantly ameliorated these alterations, demonstrating its strong antioxidant and cytoprotective properties. The improvement in biochemical parameters, along with restoration of antioxidant enzyme levels, suggests that Tulsi effectively scavenges free radicals and enhances endogenous defense mechanisms. The presence of bioactive compounds such as flavonoids and eugenol may contribute to membrane stabilization, enzyme regulation, and overall cellular protection (Gupta *et al.,* 2002; Pattanayak *et al.,* 2010).

Histopathological observations further corroborate the biochemical findings. Severe structural damage observed in the liver and kidney of atrazine-treated animals, including necrosis, fatty degeneration, and tubular injury, confirms the extent of organ toxicity. These pathological changes are consistent with earlier studies reporting atrazine-induced tissue damage (Campos-Pereira *et al.,* 2012). In contrast, Tulsi-treated groups exhibited marked improvement in tissue architecture, with near-normal morphology observed at higher doses, indicating effective protection and recovery.

The protective effects of *Ocimum sanctum* were found to be dose-dependent, with the 200 mg/kg dose showing greater efficacy across all parameters. This suggests that the therapeutic potential of Tulsi is closely linked to its concentration and bioavailability, supporting previous findings on dose-dependent antioxidant activity of medicinal plants.

The study provides a comprehensive understanding of atrazine-induced toxicity and highlights the potential of *Ocimum sanctum* as a natural intervention. By integrating biochemical, oxidative, and histological analyses, the findings strengthen the evidence that oxidative stress is a key mediator of pesticide toxicity and that plant-based antioxidants can play a crucial role in mitigating such effects.

CONCLUSION

The present study clearly demonstrates that sub-chronic exposure to atrazine induces significant physiological, biochemical, and structural alterations in experimental animals. The marked reduction in body weight, along with hyperglycemia, hypercholesterolemia, decreased plasma protein levels, and elevated acid phosphatase activity, indicates severe metabolic imbalance and organ dysfunction. These effects are strongly associated with enhanced oxidative stress, as evidenced by increased lipid peroxidation and depletion of key antioxidant enzymes such as SOD, CAT, GSH, and GPx.

Histopathological findings further confirmed the toxic impact of atrazine, revealing pronounced damage to liver and kidney tissues, including necrosis, fatty degeneration, and tubular injury. Together, these observations establish oxidative stress as a central mechanism underlying atrazine-induced toxicity.

Regulation of *Ocimum sanctum* extract significantly mitigated these adverse effects in a dose-dependent manner. Tulsi supplementation restored body weight, normalized biochemical parameters, improved antioxidant defense systems, and preserved tissue architecture. The higher dose (200 mg/kg) was particularly effective, showing near-complete recovery in most parameters.

The study highlights the potent antioxidant and cytoprotective properties of *Ocimum sanctum* and supports its potential as a natural therapeutic agent against pesticide-induced toxicity. These findings also emphasize the importance of plant-based interventions in reducing the health risks associated with environmental toxicants like atrazine.

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