

Modulatory Effects of Methanol Extract of *Spondias mombin* leaves on Arsenic-Induced Neurotoxicity in *Drosophila melanogaster*

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Abstract

Arsenic, a common environmental toxicant causes neurotoxicity via oxidative stress, impaired antioxidant defenses, and lipid peroxidation. *Spondias mombin*, a tropical plant with abundant bioactive phytochemicals, shows antioxidant, anti-inflammatory, and neuroprotective potential, though its role against arsenic neurotoxicity is underexplored. This study evaluated the modulatory effects of the methanolic extract of *Spondias mombin* on arsenic-induced neurotoxicity in *Drosophila melanogaster*. Adult flies were divided into four groups (control, *Spondias mombin* only, arsenic only, arsenic + *Spondias mombin*), each with five vials of 50 flies. The control group received a standard diet; the *Spondias mombin* group received 300 mg/kg of the extract; the arsenic group received 10 mg/kg of arsenic; and the combined group received both treatments. Post-exposure, oxidative stress and neurotoxicity markers, total protein, acetylcholinesterase (AChE), catalase (CAT), glutathione S-transferase (GST), and malondialdehyde (MDA) were evaluated. Arsenic significantly lowered total protein, CAT, and GST while elevating AChE and MDA, confirming oxidative damage and neurotoxicity. *Spondias mombin* co-treatment reversed these changes, restoring protein and antioxidant enzymes and reducing AChE and MDA levels. Improved biochemical profiles in the combined group indicate the extract's protective role against arsenic-induced neurotoxicity through modulation of oxidative stress markers. These findings affirm the neuroprotective potential of *Spondias mombin* in *Drosophila melanogaster*, supporting its role in mitigating arsenic-induced neurotoxicity.

Keywords: Arsenic, *Spondias mombin*, Neurotoxicity, *Drosophila melanogaster*, Oxidative stress

1. Introduction

Neurotoxicity refers to the harmful effects of chemical substances on the structure and function of the nervous system, leading to disruptions in neuronal signaling, brain development, and behaviour [1, 2]. Exposure to neurotoxic agents may alter neural cells directly or interfere with metabolic processes essential for normal brain function, ultimately resulting in

cognitive, motor, and behavioural impairments [3]. With increasing industrialization and environmental contamination, exposure to neurotoxic substances has become a growing public health concern, particularly in regions with limited environmental regulation and monitoring.

Among environmental toxicants, arsenic is a naturally occurring metalloid widely distributed in the earth's crust and commonly

found in soil, water, and mineral deposits [4]. Historically described as the “king of poisons,” arsenic remains one of the most toxic and pervasive environmental pollutants [5]. Human exposure occurs primarily through contaminated drinking water, food, and occupational activities such as mining, smelting, and agricultural practices. Chronic exposure to inorganic arsenic has been associated with a broad spectrum of adverse health outcomes, including dermatological disorders, cardiovascular diseases, and increased risk of several cancers [6]. Importantly, prolonged exposure to arsenic has also been linked to neurological dysfunction, particularly in vulnerable populations such as children and individuals living in contaminated environments [6].

Arsenic exerts profound effects on the central nervous system. It readily crosses the blood–brain barrier and accumulates in different brain regions, where it disrupts neuronal structure and function [6]. Experimental and epidemiological studies have shown that arsenic exposure is associated with cognitive deficits, motor dysfunction, and increased susceptibility to neurodegenerative and neurodevelopmental disorders [7]. A key mechanism underlying arsenic-induced neurotoxicity is oxidative stress. By generating reactive oxygen species and impairing antioxidant defense systems, arsenic promotes lipid peroxidation, protein damage, and neuronal apoptosis [6]. Given the high metabolic demands of the brain, these oxidative insults render neural tissues particularly vulnerable to arsenic toxicity.

In recent years, increasing attention has been directed toward the search for natural compounds with neuroprotective potential against environmental toxicants. Medicinal plants rich in bioactive phytochemicals have emerged as promising sources of antioxidants and neuroprotective agents. *Spondias mombin* (Linn, a fruit-bearing tree belonging to the family Anacardiaceae, is widely used in traditional medicine across tropical regions. Extracts of *Spondias mombin* have been reported to possess antioxidant, anti-inflammatory, antimicrobial, and neuroprotective properties [8]. These effects are largely attributed to its rich phytochemical composition, including flavonoids, tannins, phenolic acids, alkaloids, and saponins, which are known to modulate oxidative stress and inflammatory pathways [8].

Understanding how natural compounds can modulate neurotoxicity is important for developing safer and more accessible therapeutic strategies [9]. Environmental exposure to arsenic is often chronic and difficult to avoid in many communities because it occurs through unavoidable sources in daily life such as drinking water and food consumption, thereby making prevention alone insufficient [10]. Therefore, exploring

plant-based modulators that can mitigate arsenic-induced neural damage is not only scientifically relevant but also clinically useful. Such studies can provide insights into protective mechanisms, support evidence-based use of medicinal plants, and contribute to the development of affordable interventions for populations at risk.

The fruit fly, *Drosophila melanogaster*, provides a valuable experimental model for investigating arsenic-induced neurotoxicity and neuroprotection. Its well-characterised nervous system, conserved molecular pathways involved in oxidative stress responses, and genetic similarity to humans make it particularly suitable for studying the neurotoxic effects of environmental agents such as arsenic. In addition, its suitability for behavioural and biochemical assays enables the evaluation of both functional and molecular outcomes of toxic exposure and therapeutic intervention [11]. Therefore, the present study investigated the modulatory effects of the methanolic extract of *Spondias mombin* on arsenic-induced neurotoxicity in *Drosophila melanogaster*. By examining behavioural, biochemical, and histological outcomes, this study seeks to deepen understanding of the interactions between environmental toxicants and plant-derived neuroprotective agents, with potential implications for toxicology, neuroscience, and public health.

2. Materials and Methods

2.1 Ethical approval

Ethical clearance for this research was granted by the Babcock University Health Research Ethics Committee (BHUREC) under the protocol reference (BHUREC 235/25). All procedures involving *Drosophila melanogaster* were performed in compliance with international standards for welfare and the institutional policies of Babcock University.

2.2 Fly care and husbandry

Wild-type *Drosophila melanogaster* was obtained and maintained in the Eureka *Drosophila melanogaster* Laboratory, Department of Anatomy, Benjamin S. Carson School of Medicine, Babcock University, Ogun State, Nigeria. Flies were reared on standard cornmeal medium and maintained at 21–25 °C under 60–70% relative humidity with a 12 h light/dark cycle. Cultures were monitored regularly, and fresh media were supplied as required.

2.3 Chemicals, reagents, and equipment

All reagents used were of analytical grade. Distilled water, cornmeal, yeast, agar-agar, nipagin, ethanol, and arsenic were used for fly culture and treatment. Methanol was used for plant extraction. Biochemical reagents for protein estimation and enzyme assays were prepared according to standard protocols. Laboratory ware included Petri dishes, culture vials, and conical flasks. Equipment included a homogenizer, centrifuge,

micropipettes, CO₂ anesthetisation setup, refrigerator, sensitive weighing balance, and light microscope.

2.4 Preparation of cornmeal diet

The cornmeal medium was prepared adopting a previously established method [12]. 800 mL of distilled water was boiled, followed by the addition of yeast (26 g) and agar-agar (13 g) with continuous stirring. Cornmeal (90 g) was mixed separately with 400 mL of distilled water to form a slurry and added to the boiling mixture. Dextrose (25 g) was incorporated and the mixture stirred thoroughly. After cooling for 2–3 min, methyl paraben (5 g) previously dissolved in ethanol was added, mixed uniformly, and dispensed into culture vials. Media were allowed to cool and solidify before use.

2.5 Plant material and methanolic extraction of *Spondias mombin*

Fresh leaves of *Spondias mombin* were collected from Ilishan-Remo, Ogun State, Nigeria, and certified (authentication number: PSBHT-237) by the Plant Science and Biotechnology, Departmental Herbarium and Taxonomic (PSBHT) Unit, Adekunle Ajasin University, Akungba Akoko, Ondo State, Nigeria. The leaves were air-dried at room temperature, pulverized into fine powder, and soaked in 100% methanol for 72h with intermittent stirring to enhance extraction. The mixture was filtered using Whatman filter paper, and the filtrate was allowed to evaporate at room temperature. The resulting crude methanolic extract was collected, weighed, and stored until use [13].

2.6 Experimental design and treatment

Flies were randomly-assigned into four experimental groups. Each group consisted of five vials containing 50 flies per vial (250 flies per group), and treatments were administered for 21 days under standard culture conditions. The groups were categorized as follows:

Group 1 (Control): Animals in this group received only the standard cornmeal diet without any supplemental treatment.

Group 2 (*S. mombin*): This group was administered a standard diet supplemented with *S. mombin* methanolic extract at a dosage of 200 mg/kg of the diet [14].

Group 3 (Arsenic): To induce toxicity, animals were fed a standard diet supplemented with Sodium Arsenic at a concentration of 10 mg/kg of the diet.

Group 4 (*S. mombin* + Arsenic): This group received a dual-supplemented diet containing both *S. mombin* methanolic extract (200 mg/kg) and Sodium Arsenic (10 mg/kg) [15].

2.7 Survival assay

Newly emerged adult flies were used for survival analysis. Flies were maintained on their respective treatment diets, and food was replaced every five days. The number of live and dead flies was recorded daily for 21 days to assess survival across experimental groups [16].

2.8 Behavioural assays

2.8.1 Negative geotaxis assay

Locomotor performance was evaluated using the negative geotaxis assay [17]. Ten flies from each vial were gently anesthetized using CO₂ and transferred into labelled vertical glass vials (15 cm length, 1.5 cm diameter). A 6cm mark was made on each vial, and flies were gently tapped to the bottom. The number of flies that climbed above the 6 cm mark within 10s was recorded.

2.8.2 Larval crawling assay

Third instar larvae were collected using a damp paintbrush and placed on a slightly moistened Petri dish. Larval contractions, distance moved, and contraction rate were observed and recorded to assess locomotor behaviour [18].

2.9 Biochemical analyses

Biochemical analyses were performed on brain homogenates to evaluate oxidative stress markers and enzyme activities. Following behavioural assessments, adult flies were anesthetized with CO₂ and homogenized in cold phosphate buffer solution. Following behavioural assessments, adult flies were anesthetized with CO₂ and homogenized in cold phosphate buffer solution. The homogenates were centrifuged at 13,000 rpm for 5 min at 4 °C, and the resulting supernatants were collected and used immediately or stored at –20 °C until analysis [19]. Total protein concentration was determined using the Biuret method, with absorbance measured at 540 nm [20]. Catalase activity was assay following the method by Beers and Sizer [21], by monitoring the decomposition of hydrogen peroxide at 240 nm and expressing activity as μmol of H₂O₂ consumed per minute per milligram of protein [21]. Acetylcholinesterase activity was determined using the method of Ellman, by measuring absorbance at 412 nm and expressing enzyme activity as $\mu\text{mol}/\text{min}/\text{mg}$ protein [22]. Lipid peroxidation was assessed by measuring malondialdehyde (MDA) levels using the thiobarbituric acid reactive substances (TBARS) method as previously described [19]. Malondialdehyde concentration was expressed as mmol MDA formed per milligram of protein using a molar extinction coefficient of $1.56 \times 10^5 \text{ M}^{-1} \text{ cm}^{-1}$ [23]. Glutathione-S-transferase (GST) activity was determined according to the method of Habig and Jakoby, by monitoring the conjugation of reduced glutathione with CDNB at 340 nm [24].

2.10 Statistical Analysis

Data were analysed using one-way analysis of variance (ANOVA) to assess significant differences among multiple groups under various treatments, followed by Tukey's post-hoc test using GraphPad Prism version 8.5. Results were expressed as mean $\pm$ SEM, and statistical significance was set at $p < 0.05$.

3. Results

3.1 Survival Assay

Figure 1a and 1b show result of survival assay of the flies following arsenic neurotoxicity. The lifespan of flies was determined by recording the number of dead flies per day through out the duration of the experiment. The result from this study shows that arsenic (ASEN) was associated with increased mortality of the flies by day 8, with the percentage survival rate showing a significant decrease in this group compared to the control groups (CONT, SM). In the group administered *S. mombin* and arsenic there was no significant change. The percentage survival rate in this group was similar to that of the arsenic group and was also significantly reduced when compared to the control group

3.2 Behavioural Assay

Figure 2 shows the result of behavioural assay. There was a decrease in the locomotor activity in the arsenic group and also in group co-administered arsenic with *S. mombin* group compared to the control and *S. mombin* group after 7 days. Treatment with *S. mombin* and arsenic was associated with increase the locomotor activity the flies.

3.3 Larva crawling activity

Figure 3a and 3b show larval crawling activity measured as the distance travelled and contraction of *Drosophila* larva respectively across experimental groups. The result showed a decrease in the distance and contraction in the arsenic group when compared to the control. In the group treated with *S. mombin* and arsenic there was improved larva performance evidenced by increased the distance covered and the number of contraction when compared to the control group.

3.4 Biochemical Assay

3.4.1 Brain Protein Levels

Figure 4 shows the effect of *S. mombin* on protein levels in whole brain homogenate. Results showed that ASEN administration was associated with a significant decrease in total protein level when compared to other experimental groups. In the groups administered *S. mombin* and arsenic there was a significant increase in total protein level in the brain lysate compared to the arsenic groups.

3.4.2 Catalase Activity

Figure 5 shows the effect of *S. mombin* on catalase activity following arsenic induced neurotoxicity. Result showed a significant decrease in catalase activity with ASEN compared to other experimental groups. In groups co-administered *S. mombin* and arsenic there was a significant increase in brain catalase activity compared to arsenic alone group.

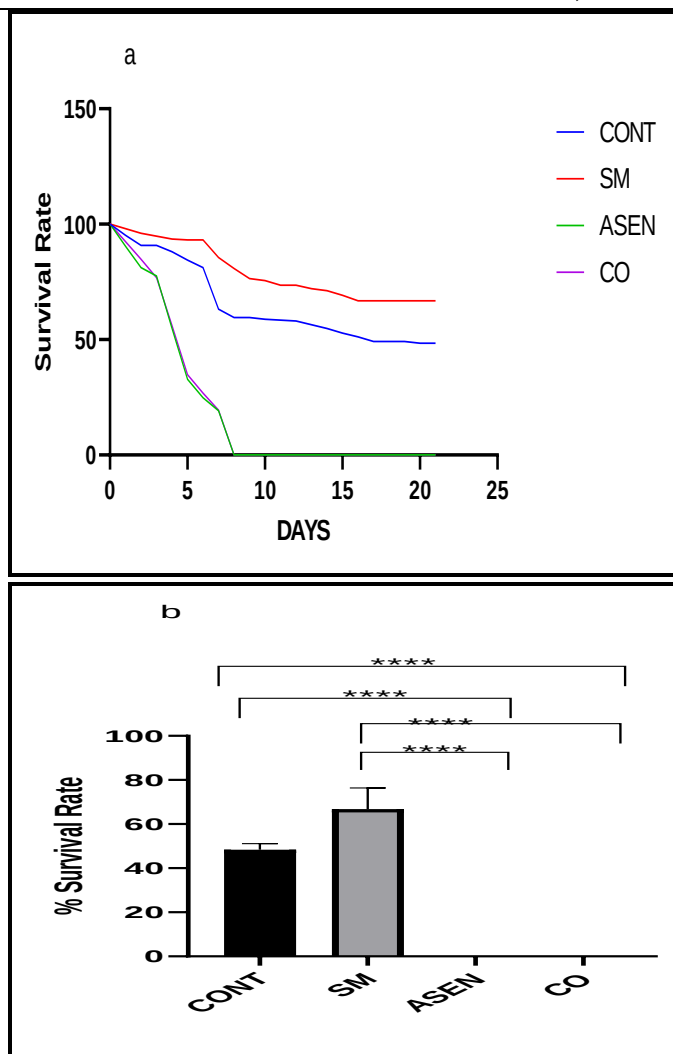


Figure 1. (a & b): Showing the percentage of survival rate across the groups. $p < 0.05$, $** = p < 0.01$, $*** = p < 0.001$, $**** = p < 0.0001$

3.4.3 Acetylcholinesterase Activity

Figure 6 shows the effect of *S. mombin* on brain levels of acetylcholinesterase. There was a significant increase in acetylcholinesterase activity ASEN compared to other experimental groups. In groups co-administered *S. mombin* with arsenic a significant decrease in acetylcholinesterase activity was observed compared to the arsenic alone group.

3.4.4 Malondialdehyde Levels

Figure 7 shows the effect of *S. mombin* on brain levels of malondialdehyde (MDA). Arsenic administration was associated with an increase in lipid peroxidation measured as MDA compared to other experimental groups. In the groups co-administered *S. mombin* and arsenic there was a significant decrease in brain MDA levels compared to the arsenic alone group.

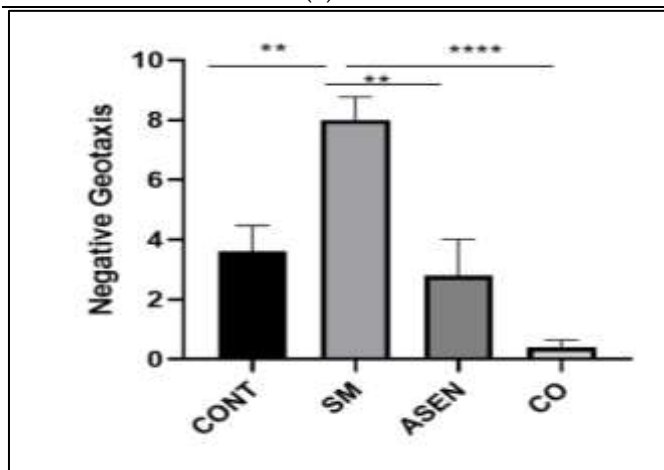


Figure 2. Showing the effects of Spondias mombin on locomotor activity across the groups. $p < 0.05$, **= $p < 0.01$, ***= $p < 0.001$, ****= $p < 0.0001$.

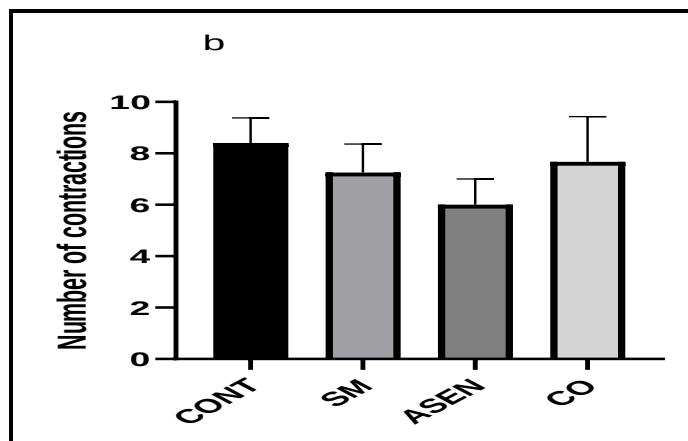
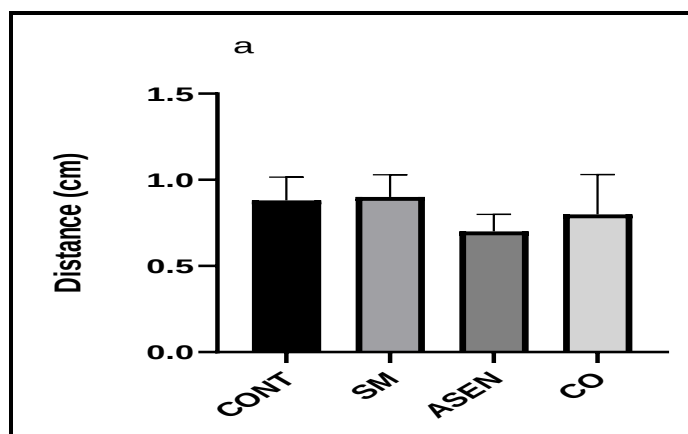


Figure 3. (a & b): Showing the effects of *S. mombin* on larva crawling across all groups. Values presented as Mean $\pm$ S.E.M ($p < 0.05$).

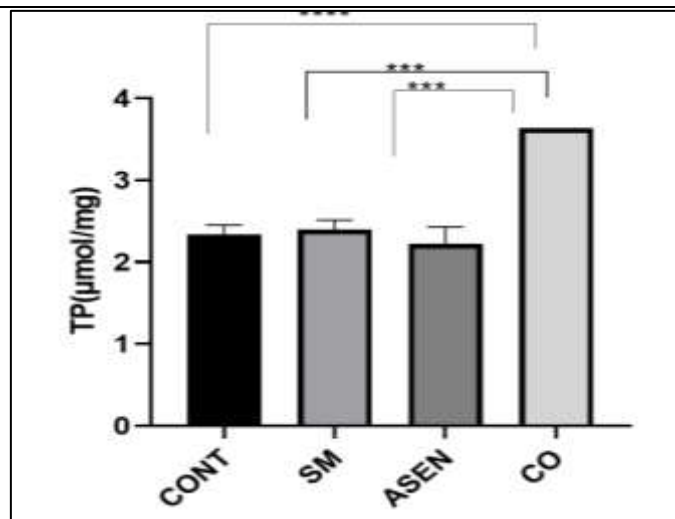


Fig 4. shows total protein level in the brain lysate across treatment groups. Values are expressed in Mean $\pm$ S.E.M, significance was established at ($p < 0.05$). * = $p < 0.05$, **= $p < 0.01$, ***= $p < 0.001$, ****= $p < 0.0001$.

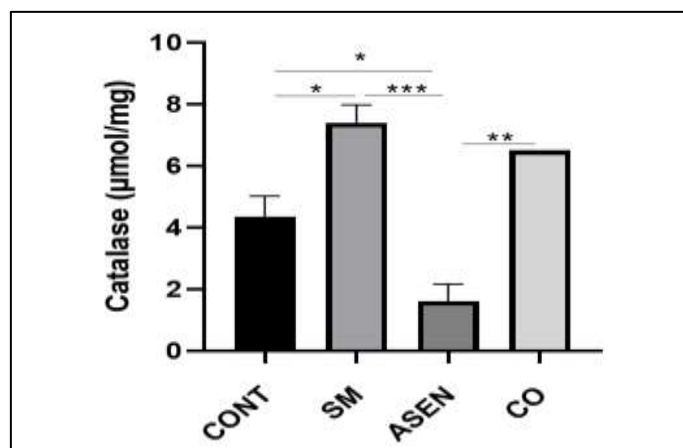


Fig 5. Shows the activity of catalase in the brain lysate across treatment groups. * = $p < 0.05$, **= $p < 0.01$, ***= $p < 0.001$, ****= $p < 0.0001$

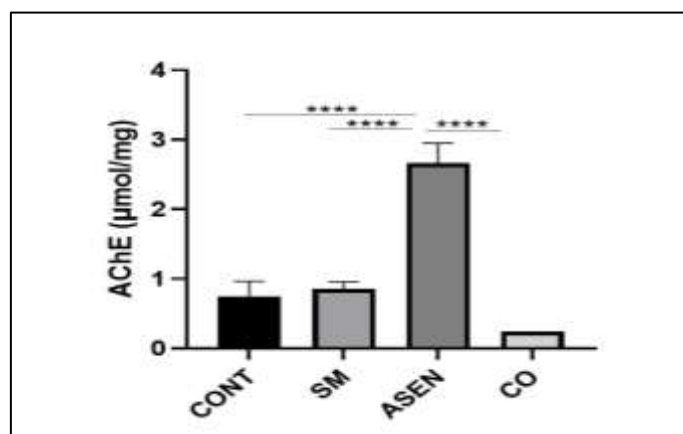


Fig. 6. Shows the activity of acetylcholinesterase in the brain lysate across treatment groups. Values are presented as Mean $\pm$ S.E.M. Significance was established at ($p < 0.05$). * = $p < 0.05$, **= $p < 0.01$, ***= $p < 0.001$, ****= $p < 0.0001$

3.4.5. Glutathione -S Transferase

Figure 8 shows the effect of *S. mombin* on brain levels of glutathione -S Transferase (GST). Result showed that the administration of ASEN was associated with a significant decrease brain GST level compared to other experimental groups. In the group co-administered *S. mombin* and arsenic there was a significant increase in the brain GST level compared to the arsenic alone group.

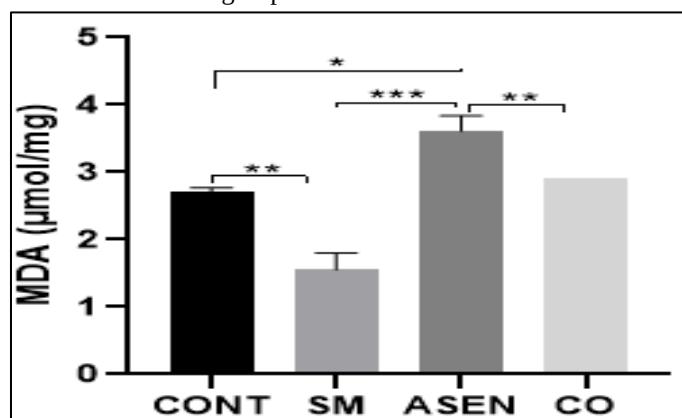


Figure 7. Shows the level of MDA in the brain lysate of across treatment groups. Significance was established at ($p < 0.05$). * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, **** = $p < 0.0001$

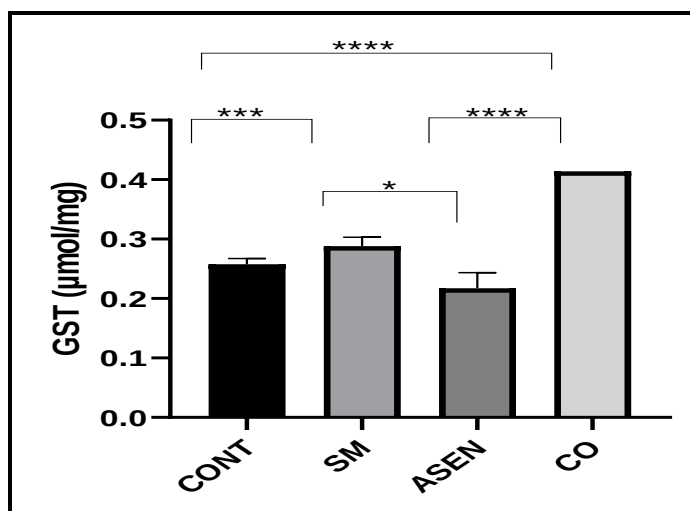


Figure 8. Shows the level of GST in the brain lysate across treatment groups. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, **** = $p < 0.0001$

Discussion

This study examined the ability of methanolic extract of *Spondias mombin* to modify arsenic-induced neurotoxicity in *Drosophila melanogaster* using survival, behavioural, and biochemical assessments. *Drosophila melanogaster* is a well-established model for neurotoxicity studies because it shares conserved cellular and molecular pathways involved in oxidative stress, neurotransmission, and neuronal degeneration with higher organisms [25, 26]. The results of this study showed

that arsenic exposure produced marked toxic effects on survival, behaviour and brain biochemistry, while treatment with *S. mombin* provided partial protection, mainly at the biochemical and larval behavioural.

Arsenic exposure caused a severe reduction in lifespan, with complete mortality observed by day eight. This finding confirms the high toxicity of arsenic and reflects cellular stress associated with increased oxidative damage, as evidenced by altered antioxidant enzyme activities and elevated lipid peroxidation. Similar reductions in survival have been reported in previous studies, where arsenic exposure led to reduced survival rates and increased mortality in *Drosophila* and other experimental models [27-29]. Treatment with *S. mombin* after arsenic exposure did not improve survival, as mortality in the co-treated group was comparable to that of the arsenic-only group. This suggests that, under the conditions used in this study, *S. mombin* was unable to counteract the lethal effects of arsenic. The lack of survival benefit may be related to the severity of arsenic toxicity, the dose of the extract, or the timing of treatment. It may also be possible that *S. mombin* may confer protection at biochemical and functional levels without significantly affecting survival under acute arsenic exposure. Importantly, flies treated with *S. mombin* alone showed normal survival, indicating that the extract itself was not toxic.

Arsenic exposure also significantly impaired locomotor activity in adult flies, as shown by reduced performance in the negative geotaxis assay. Locomotor dysfunction is a common indicator of neurotoxicity and is often linked to damage in motor neurons, neuromuscular junctions, and dopaminergic pathways. Previous studies have shown that arsenic disrupts neurotransmitter signaling, induces mitochondrial dysfunction, and promotes oxidative damage in motor circuits, leading to reduced climbing ability in *Drosophila* and mammalian models [6, 28]. In this study, treatment with *S. mombin* did not significantly restore adult locomotor performance. This suggests that although the extract may improve biochemical markers of oxidative stress, such protection may not be sufficient to reverse established motor deficits in adult flies. Behavioural recovery often requires extensive neuronal repair, which may not occur within the treatment period used.

In contrast to the adult locomotor findings, larval crawling behaviour showed a more favourable response to *S. mombin* treatment. Arsenic significantly reduced the distance travelled and the number of contractions in larvae, indicating impaired neuromuscular coordination. These findings are consistent with previous studies demonstrating that arsenic interferes with neuronal signaling and muscle coordination through oxidative stress and mitochondrial dysfunction [27, 29]. Notably, treatment with *S. mombin* following arsenic exposure improved

larval crawling performance, with increased distance travelled and contraction frequency compared to the arsenic-only group. This improvement suggests that developing neural systems may be more responsive to antioxidant intervention than mature ones. The enhanced larval performance may be due to the ability of *S. mombin* to reduce oxidative damage and support neuronal function during early developmental stages.

Biochemical findings further support the neurotoxic effects of arsenic and the protective role of *S. mombin*. Arsenic exposure caused a significant reduction in total protein levels in the brain, reflecting impaired protein synthesis, increased protein degradation, or neuronal loss. Protein depletion is a common feature of neurotoxicity and indicates compromised cellular integrity. Similar reductions in brain protein content have been reported in previous studies following arsenic exposure [27, 29]. Treatment with *S. mombin* significantly restored total protein levels, suggesting preservation of neuronal structure and improved cellular stability. This protective effect may be linked to the antioxidant properties of *S. mombin*, which help limit oxidative damage to cellular proteins [30, 31].

Arsenic also disrupted antioxidant defense systems, as shown by reduced catalase and GST activities and increased MDA levels. The decrease in catalase and GST suggests weakened enzymatic protection against reactive oxygen species and toxic metabolites, while the elevated MDA level indicates increased lipid peroxidation and membrane damage. These changes confirm that oxidative stress plays a major role in arsenic-induced neurotoxicity, as widely reported in previous studies across different experimental models [6, 27, 32]. Treatment with *S. mombin* significantly increased catalase and GST activities and reduced MDA levels, demonstrating its ability to strengthen antioxidant defenses and limit oxidative damage. These effects are likely due to the presence of flavonoids, phenolics, and other bioactive compounds in *S. mombin*, which have been shown in previous studies to scavenge free radicals and enhance endogenous antioxidant systems [30, 33].

Cholinergic dysfunction was also evident following arsenic exposure, as indicated by a significant increase in acetylcholinesterase (AChE) activity. Elevated AChE activity can lead to excessive breakdown of acetylcholine, resulting in impaired synaptic transmission and neuronal communication. Such alterations have been associated with cognitive and motor dysfunction in previous studies examining heavy metal and chemically induced neurotoxicity [27, 34]. Treatment with *S. mombin* significantly reduced AChE activity in arsenic-exposed flies, suggesting normalization of cholinergic function. This finding is consistent with earlier reports showing that *S. mombin* modulates AChE activity and supports neurotransmitter balance in experimental models of neurotoxicity [33, 31].

The findings of this study show that arsenic induces severe neurotoxicity in *Drosophila melanogaster*, affecting survival, behaviour and brain biochemistry. While *S. mombin* did not prevent arsenic-induced mortality or fully restore adult locomotor function, it significantly reduced oxidative stress, improved antioxidant and detoxification enzyme activities, normalized acetylcholinesterase levels, and enhanced larval motor performance. These findings suggest that *S. mombin* offers meaningful neuroprotection at the cellular and biochemical levels, even though it may not be sufficient to counteract the acute lethality of arsenic. The study highlights the potential of *S. mombin* as a natural agent for reducing arsenic-induced neural damage and supports further investigation into its active components, optimal dosing strategies, and mechanisms of action.

Strengths of the Study

A major strength of this study lies in the use of *Drosophila melanogaster* as a well-validated model for investigating neurotoxicity and neuroprotection. The fly model offers conserved molecular pathways involved in oxidative stress, neurotransmission, and neuronal degeneration, allowing meaningful interpretation of arsenic-induced neural damage and the modulatory effects of *Spondias mombin* [21, 22].

Another notable strength is the integration of multiple experimental endpoints, including survival analysis, behavioural assays, biochemical evaluations, and histological examination. This comprehensive approach provided a broad assessment of arsenic toxicity and allowed the neuroprotective effects of *S. mombin* to be evaluated at functional, biochemical, and structural levels. The consistency between biochemical markers of oxidative stress (MDA, catalase, GST), behavioural outcomes (larval crawling), and histological findings strengthens the reliability of the conclusions drawn.

Additionally, the study assessed both adult and larval stages, offering insight into developmental differences in susceptibility to arsenic toxicity and responsiveness to antioxidant intervention. The observed improvement in larval locomotor activity following *S. mombin* treatment highlights the relevance of developmental stage in neuroprotective studies and adds depth to the findings.

Despite its strengths, this study has some limitations that should be acknowledged. First, *S. mombin* treatment did not significantly improve survival or adult locomotor performance in arsenic-exposed flies. This suggests that the dose, duration, or timing of extract administration may not have been optimal for reversing severe arsenic-induced damage. A dose-response or pre-treatment design may have yielded different outcomes.

Second, the study focused on biochemical and behavioural endpoints without direct molecular analyses such as gene expression profiling or signaling pathway assessment. As a result, the precise molecular mechanisms underlying the observed antioxidant and neuroprotective effects of *S. mombin* could not be fully elucidated.

Another limitation is the use of a crude methanolic extract rather than isolated bioactive compounds. While the extract showed beneficial effects, the specific constituents responsible for these effects remain unidentified. Furthermore, although *Drosophila melanogaster* is a powerful model for neurotoxicity research, findings from insect models may not fully translate to mammalian or human systems, highlighting the need for further validation in higher organisms.

Conclusion

This study demonstrates that arsenic exposure induces pronounced neurotoxicity in *Drosophila melanogaster*, as shown by reduced survival, impaired motor performance, increased oxidative stress, disrupted antioxidant defenses, altered cholinergic activity, and significant neuronal degeneration. Treatment with methanolic extract of *Spondias mombin* provided partial neuroprotection by improving antioxidant enzyme activity, reducing lipid peroxidation, restoring total protein levels, normalizing acetylcholinesterase activity, enhancing larval motor function, and preserving brain histoarchitecture.

Although *S. mombin* did not fully rescue arsenic-induced mortality or adult locomotor deficits, its ability to attenuate oxidative damage and support neuronal integrity highlights its therapeutic potential. These findings suggest that *Spondias mombin* may serve as a valuable natural agent for reducing arsenic-related neural damage, particularly at the biochemical and cellular levels. Further studies focusing on dose optimization, molecular mechanisms, and validation in mammalian models are recommended to fully explore its neuroprotective capacity.

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None

Availability of data and materials

Data generated during and analysed during the course of this study are available from the corresponding author on request.

Competing interests

All authors of this paper declare that there is no conflict of interest related to the content of this manuscript.

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