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Research Article
Integrated *in vivo* and *in silico* Assessment of Paraquat-Induced Toxicity in *Drosophila melanogaster*

[1]Monday Alexander Etuh, [2,3]Oluwatosin Imoleayo Oyeniran and [1]Dung D. Pam

[1]Department of Zoology (Applied Entomology and Parasitology Unit), University of Jos, Jos, Nigeria
[2]Department of Physiology, Faculty of Basic Medical Science, College of Health Sciences, Nile University of Nigeria, Abuja, Nigeria
[3]Department of Kinesiology and Integrative Physiology, Michigan Technological University, Houghton, Michigan, United States

Abstract

Background and Objective: The extensive use of paraquat, a highly toxic herbicide, poses a major risk to non-target organisms through the generation of reactive oxygen species (ROS). This study investigated paraquat-induced toxicity in *Drosophila melanogaster* using a combination of *in vivo* behavioral and biochemical assays alongside *in silico* molecular modeling to evaluate its effects on detoxification and cholinergic systems. **Materials and Methods:** Fruit flies were exposed to varying paraquat concentrations ranging from 2.5-10 mg/10 g diet for seven days to determine the Median Lethal Concentration (LC_{50}), followed by a 72 hrs sublethal exposure at 2.5 mg/10 g diet. Negative geotaxis behavior, acetylcholinesterase (AChE), glutathione-S-transferase (GST) and catalase activities were measured to assess oxidative stress and neurobehavioral impacts. Molecular docking was performed using PyRx and Discovery Studio to identify potential interactions between paraquat and target enzymes. **Results:** The LC_{50} was determined as 3.261 mg/10 g diet. Sublethal exposure significantly increased mortality ($p<0.05$) and reduced GST and AChE activities, while negative geotaxis and catalase activity showed non-significant declines, indicating oxidative stress-induced impairment. Docking analysis revealed weak binding of paraquat to GST (-4.5 kcal/mol) and moderate binding to AChE (-6.9 kcal/mol), with stronger interactions observed for positive controls such as NBDHEX (-5.3 kcal/mol) and galantamine (-9.2 kcal/mol). **Conclusion:** These findings demonstrate that paraquat disrupts both detoxification and neural systems in *D. melanogaster* through oxidative stress and specific molecular interactions, highlighting its ecological and toxicological hazards. This study supports the use of *D. melanogaster* as a sensitive model organism for environmental toxicology studies and underscores the need for further experimental validation to confirm molecular docking findings.

Key words: Paraquat, Non-target organism, *D. melanogaster*, *in vivo*, *in silico* modeling, ligands, glutathione-S-transferase, acetylcholinesterase

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Corresponding Author: Alexander M. Etuh, Department of Zoology (Applied Entomology and Parasitology Unit), University of Jos, Jos, Nigeria

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Reductions in biodiversity and alterations in insect distribution have been associated with the extensive use of synthetic pesticides, including insecticides and herbicides[1,2]. These chemicals can affect non-target organisms, disrupt ecological processes and impair insect behavior and reproductive fitness[3]. Paraquat is a widely used, non-selective bipyridyl herbicide of particular toxicological concern because of its high biological toxicity and ability to generate reactive oxygen species (ROS)[4]. Its strong binding to soil particles influences its environmental fate, although residues may persist in agricultural environments and may be transported through contaminated soil particles and runoff[4]. Exposure may occur through occupational handling and application, as well as accidental or environmental contact, making paraquat an important public-health and environmental contaminant[4]. Paraquat toxicity is largely associated with redox cycling and excessive ROS generation, which can overwhelm antioxidant defenses and cause oxidative damage to cellular components[5]. In *Drosophila melanogaster*, paraquat exposure has been associated with oxidative stress, ultrastructural changes, DNA damage and neurological dysfunction[6,7]. *Drosophila melanogaster* is a well-established model organism with conserved genetic, cellular and biochemical pathways, making it valuable for investigating toxicant-induced molecular, biochemical and behavioral effects[8].

Despite growing evidence of paraquat toxicity, the relationship between paraquat-induced behavioral impairment, alterations in detoxification enzymes and potential molecular interactions with key target proteins remains insufficiently characterized in *D. melanogaster*. In particular, few studies have integrated behavioral and biochemical responses with molecular docking to provide a mechanistic link between paraquat exposure and its biological effects. This represents an important research gap because identifying potential interactions with enzymes involved in neurotransmission and detoxification may help explain the physiological consequences of paraquat exposure.

Therefore, this study aims to investigate paraquat-induced toxicity in *D. melanogaster* by integrating *in vivo* behavioral and biochemical assessments with *in silico* molecular docking. Acetylcholinesterase (AChE) and glutathione-S-transferase (GST) were selected as key molecular targets because of their roles in neurotransmission and cellular detoxification, respectively. Molecular docking was used to examine potential interactions of paraquat with these enzymes, while behavioral and biochemical assays were used to assess its physiological effects. By integrating these levels of evidence, this study seeks to provide a more comprehensive understanding of the molecular and physiological mechanisms underlying paraquat toxicity in *D. melanogaster.*

MATERIALS AND METHODS

Study area: This study was conducted at the Drosophila Laboratory of the Africa Centre of Excellence in Phytomedicine Research and Development (ACEPRD), University of Jos, Plateau State, Nigeria, from August to November 2025.

Reagents: Throughout the experiment, analytical-grade chemicals were used. The source of paraquat was an agrochemical store. Sigma Aldrich (USA) provided the CDNB (1-chloro-2,4-dinitrobenzene, 5,5), GSH (reduced glutathione), DTNB (5,5'-dithiobis (2-nitrobenzoic acid)) and acetylcholine iodide. However, Medicom, Jos, Plateau State, Nigeria, provided the Sodium Dihydrogen Phosphate ($NaH_2PO_4.2H_2O$), Randox protein kit, Potassium Hydrogen Phosphate (K_2HPO_4) and Ethylenediaminetetraacetic Acid (EDTA).

D. melanogaster. *D. melanogaster* (Harwich strain) was bred and maintained under stable conditions at the Africa Centre of Excellence in Phytomedicine Research and Development, Nigeria, with a consistent temperature (23±1°C) and humidity (60%), using 12 hrs light and dark cycles. The Drosophila medium was prepared with yellow cornmeal, brewer's yeast, agar and a preservative, following the method described by Etuh *et al.*[9].

Behavioral changes

Median Lethal Concentration (LC_{50}) and short duration (72 hrs) exposure: Two- to three-day-old *D. melanogaster* were treated with 2.5 mg PQ/10 g, 5 mg PQ/10 g, 7.5 mg PQ/10 g and 10 mg PQ/10 g of Paraquat[8]. Mortality score was taken 24-hourly for 7 days. The technique of ingestion[9] was used for a brief exposure (72 hrs). Fifty flies were treated in triplicate with 2.5 mg PQ/10 g diet and control (diet alone) at concentrations below the median lethal limit (LC_{50}). Temperature and relative humidity were controlled throughout this experiment. For 72 hrs, the mortality score was calculated every 24 hrs. Before being sacrificed, homogenised (1 mg of fruit fly weight: 10 µL of 0.1 M phosphate buffer, pH 7.0) and centrifuged (10 min at 4000 rpm; (temperature, 4°C)), the surviving flies were used

for the negative geotaxis assay. Acetylcholinesterase and antioxidant activity (Glutathione-S-transferase, or GST and Catalase) assays were performed using the obtained supernatant.

Negative geotaxis: This behaviour indicates the health of the *D. melanogaster* locomotor system. For this test, a clear, vertical glass tube of 15 cm (diameter and height, respectively) was utilized. Ten flies were selected at random and put in the tube, to put it briefly. The tube was tapped gently on a soft, level surface after one minute (to allow for recuperation and acclimatisation). The proportion of flies that managed to ascend the marked 6-cm height in 6 sec was noted and scored[8].

Biochemical changes

Total plasma protein: According to Pam *et al.*[8] and Nwanna *et al.*[10], the protein content of the 2.5 mg Paraquat-exposed and control fruit flies was measured using a Sigma Randox protein kit.

Glutathione-S-transferase: The reagents utilized for this assay were CDNB and reduced glutathione. Ethylenediaminetetraacetic Acid (EDTA) and 0.25 M phosphate buffer (pH 7.4) were used to prepare reduced glutathione, while CDNB was prepared in absolute ethanol. The reaction mixture consisted of the collected supernatant (fly sample), GSH and CDNB. A blank was prepared by replacing the fly sample with distilled water. For two minutes, absorbance determination occurred at 340 nm every ten seconds. As µmol CDNB conjugate formed/min/mg protein, the activity was computed[11].

Catalase: The reagent utilized for this assay was hydrogen peroxide and was prepared employing a 0.05 M phosphate buffer (pH 7.4). The reaction mixture consisted of the collected supernatant (fly sample) and prepared hydrogen peroxide. A blank was prepared by replacing the fly sample with distilled water. For two minutes, the absorbance was determined at 240 nm every ten seconds. To calculate the activity, µmol H_2O_2 decomposed/min/mg protein was used[8].

Acetylcholinesterase: The reagents utilized for this assay were DTNB and acetylcholine. DTNB was prepared in 0.1 M phosphate buffer (pH 7.4), while acetylcholine was produced using distilled water. The reaction mixture consisted of collected supernatant (fly sample), distilled water, 0.1 M phosphate buffer, prepared DTNB and acetylcholine. A blank was prepared by replacing the fly sample with distilled water. For two minutes, the absorbance was determined at 412 nm every ten seconds. Acetylcholine hydrolysis activity was quantified as µmol/min/mg protein[12].

***In silico* modeling:** To dock the 3D crystal structure of the protein targets, acetylcholinesterase (PDB ID: 6XYU) and glutathione-S-transferase (PDB ID: 1M0U), obtained from the protein database were utilised and the technique was outlined by Nwamma *et al.*[10] and Iorjiim *et al.*[13]. The ligands, Paraquat (CID: 15939), Glutathione (CID: 124,886), Acetylcholine (CID: 187), 6-(7-Nitro-2,1,3-benzoxadiazol-4-ylthio)hexanol (NBDHEX) (CID: 9,817,686) and Galantamine (CID: 9651), were extracted from the PUBCHEM database. The PyRx tool was used to convert the MOL SDF format to a PDBQT file and the optimisation algorithm was used to minimise energy using the force field set at UFF. A PyRx and AutoDock Vina were used for the molecular docking processes, depending on the scoring function choice[13]. The Discovery Studio 2017R2 visualiser was used to analyse the target protein's manner of interaction with ligands.

Statistical analysis: The mean difference between the exposed group and control data, presented as Mean±SD, was calculated using a t-test (unpaired). A non-linear regression dose-response curve was used to calculate the LC_{50}. The significance level of $p<0.05$ was established using GraphPad Prism Version 8.

RESULTS

Median Lethal Concentration (LC_{50}): Exposure of *D. melanogaster* to paraquat revealed an LC_{50} of 3.261 mg PQ/10 g diet after 7 days, with 91.7% mortality at 10 mg PQ/10 g (Fig. 1). A 2.5 mg PQ/10 g concentration was selected for sublethal exposure studies.

72 hrs mortality: Short-term exposure at 2.5 mg PQ/10 g induced significantly higher mortality ($p<0.05$) compared to controls, with rates ranging from 6.7% to 37.3% (Fig. 2).

Total plasma protein: Total plasma protein was slightly elevated in paraquat-exposed flies compared to control, though not significantly ($p>0.05$) (Fig. 3).

Locomotor activities and AChE: Acetylcholinesterase activity and climbing ability declined in paraquat-exposed flies, with AChE activity significantly reduced ($p<0.05$) but climbing decline non-significant ($p>0.05$) (Fig. 4a-b).

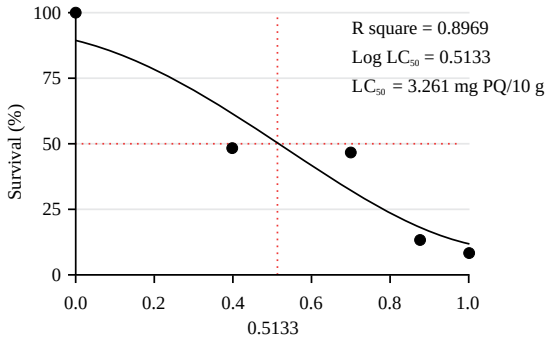


Fig. 1: 7 day LC_{50} of paraquat in *D. melanogaster*

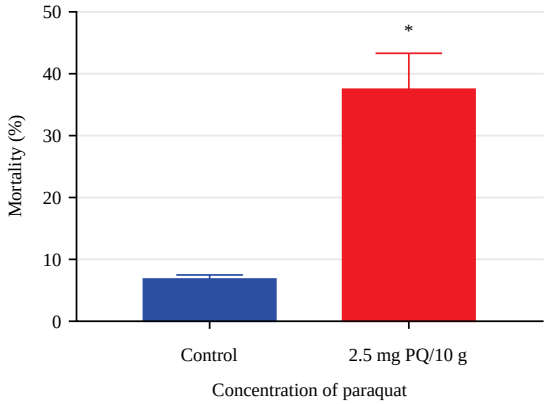


Fig. 2: 72 hrs mortality rate of *D. melanogaster* exposed to paraquat

*: Significantly elevated ($p<0.05$) compared to control

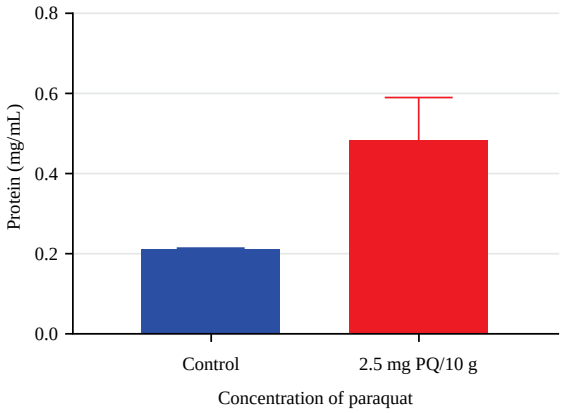


Fig. 3: Protein content of *D. melanogaster* after 72 hrs of paraquat exposure

Antioxidant enzymes: The GST activity significantly decreased ($p<0.05$) in exposed flies, while catalase activity decreased non-significantly ($p>0.05$) (Fig. 5a-b)

***In silico* docking:** Paraquat showed weak binding affinity to GST (-4.5 kcal/mol) and moderate affinity to AChE (-6.9 kcal/mol), compared to stronger binding by inhibitors NBDHEX (-5.3 kcal/mol) and galantamine (-9.2 kcal/mol), respectively (Table 1).

The GST-ligand interactions revealed that GSH bound to GST via hydrogen bonds with GLN 96, HIS 103, GLN 109, SER 110, ASN 142 and ARG 145 (Fig. 6a-b), NBDHEX formed hydrogen bonds with GLN 96, MET 57, HIS 108, SER 110 and a pi-akyl contact with LEU 60 (Fig. 7a-b), Paraquat interacted via pi-pi stacking with PHE 55 and pi-akyl contact with VAL 57, ALA 59 and LEU 60 (Fig. 8a-b). NBDHEX shared GLN 96 and SER 110 with GSH, while paraquat only shared LUE 60 with NBDHEX.

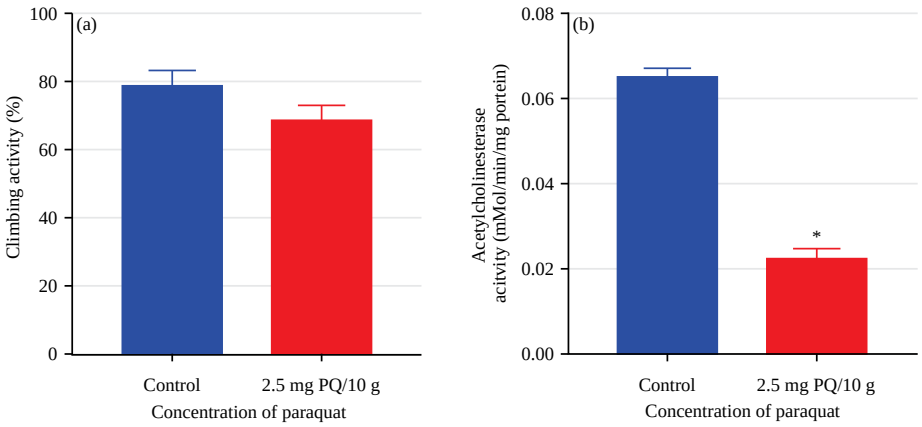


Fig. 4(a-b): Locomotor activities (a) Negative geotaxis and (b) Acetylcholinesterase activities of *D. melanogaster* after 72 hrs of paraquat exposure

*: Significantly declined compared to control group

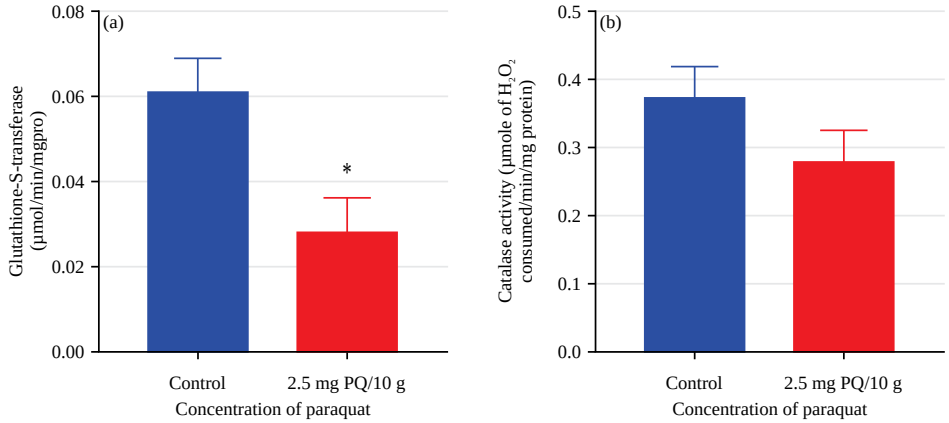


Fig. 5(a-b): Antioxidant system (a) Glutathione-S-transferase and (b) Catalase activities of *D. melanogaster* after 72 hrs of paraquat exposure

*: Significantly declined compared to the control group

Table 1: Binding energy of ligands (Paraquat, GSH, NBDHEX, galantamine, AChE) to glutathione-s-transferase and acetylcholinesterase

		Binding energy (Kcal/mol)	
Compounds	Pubchem CID	GST	AChE
Substrates			
GSH	124886	-5.1	-
Acetylcholine	187	-	-4.6
Inhibitors			
NBDHEX	9817686	-5.3	-
Galantamine	9651	-	-9.2
Paraquat	15939	-4.5	-6.9

GST: Glutathione-S-transferase, AChE: Acetylcholinesterase, GSH: Reduced glutathione, NBDHEX: 6-(7-nitro-2,1,3-benzoxadiazol-4-ylthio)hexanol, PubChem CID, PubChem Compound Identifier. Binding energy values are expressed in kcal/mol and represent the predicted docking affinity of each compound toward the indicated target enzyme. -: No docking interaction evaluated/not applicable. Lower (more negative) binding energy indicates stronger predicted binding affinity

AChE-ligand interaction revealed that acetylcholine formed hydrogen bonds with GLY 151 and ALA 239 and carbon hydrogen (C-H) bonds with TYR 71 and PHE 371 (Fig. 9a-b). Galantamine formed a hydrogen bond TYR 374, carbon hydrogen (C-H) bonds with TYR 370, HIS 480, GLY 150, pi-pi stacking with TRP 83 and pi-alkyl contact with TYR 71 (Fig. 10a-b). Paraquat exhibited pi-pi stacking with TYR 374, PHE 371, TYR 71, TYR 370 and carbon hydrogen (C-H) bond with ASP 375 (Fig. 11a-b). Paraquat shared TYR 71, TYR 370, PHE 371 and TYR 374 with galantamine and acetylcholine at the binding pocket.

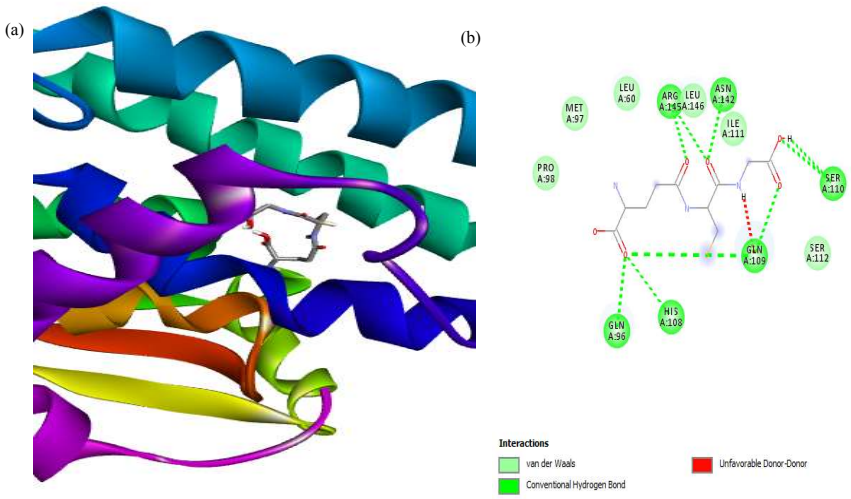


Fig. 6(a-b): (a) 3D and (b) 2D of molecular interaction of Glutathione-S-transferase amino acid residues with GSH

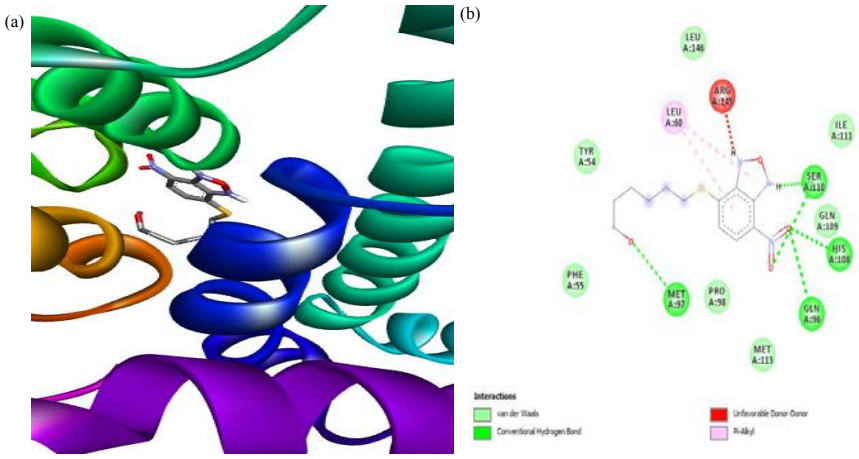


Fig. 7(a-b): (a) 3D and (b) 2D of molecular interaction of Glutathione-S-transferase amino acid residues with 6-(7-Nitro-2,1,3-benzoxadiazol-4-ythio)hexanol (NBDHEX)

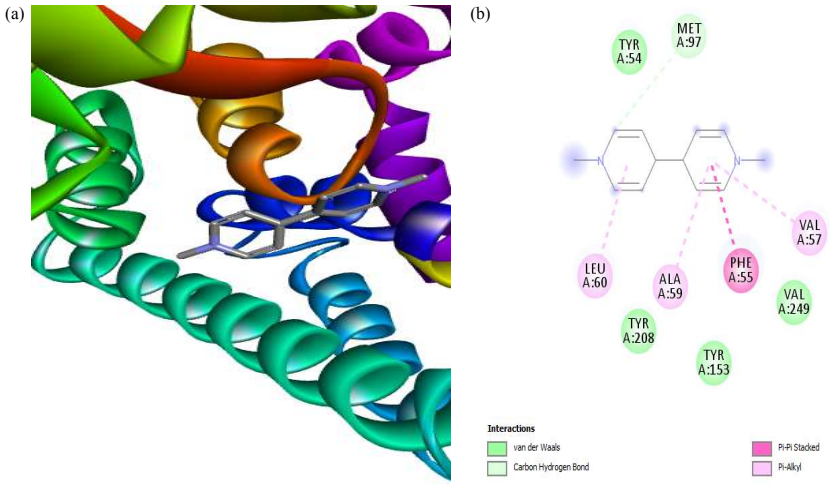


Fig. 8(a-b): (a) 3D and (b) 2D of molecular interaction of Glutathione-S-transferase amino acid residues with paraquat

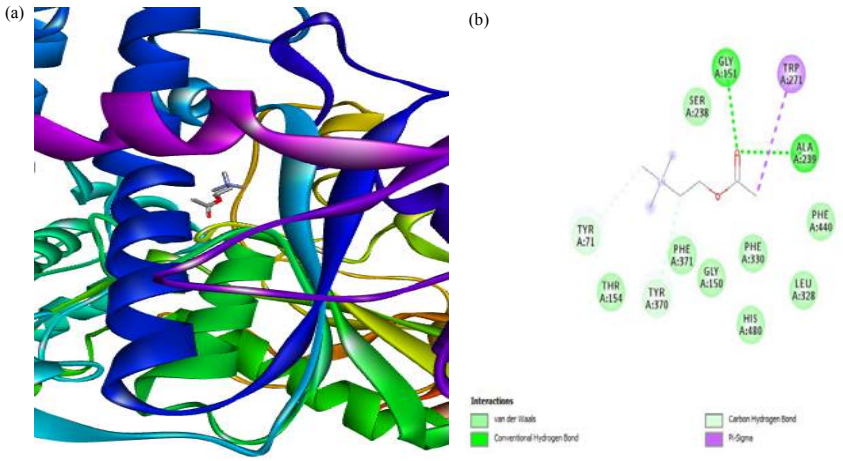


Fig. 9(a-b): (a) 3D and (b) 2D of molecular interaction of acetylcholinesterase amino acid residues with acetylcholine

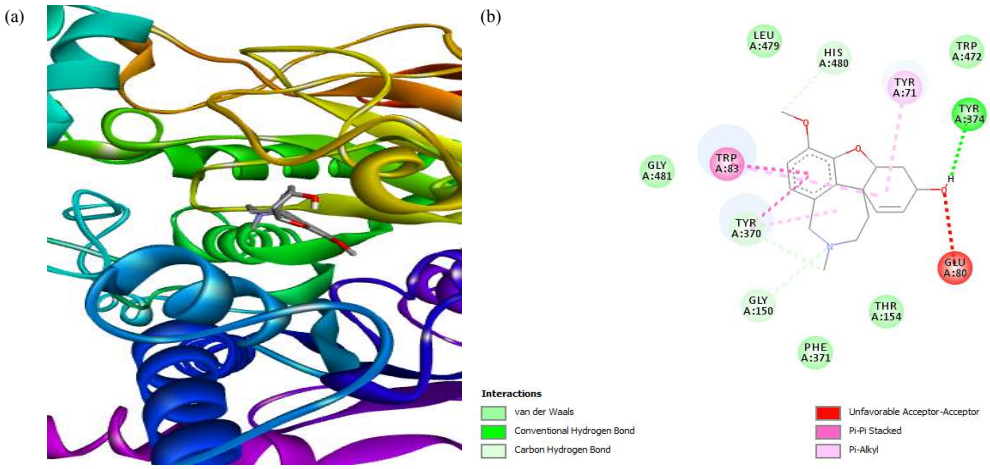


Fig. 10(a-b): (a) 3D and (b) 2D of molecular interaction of acetylcholinesterase amino acid residues with galantamine

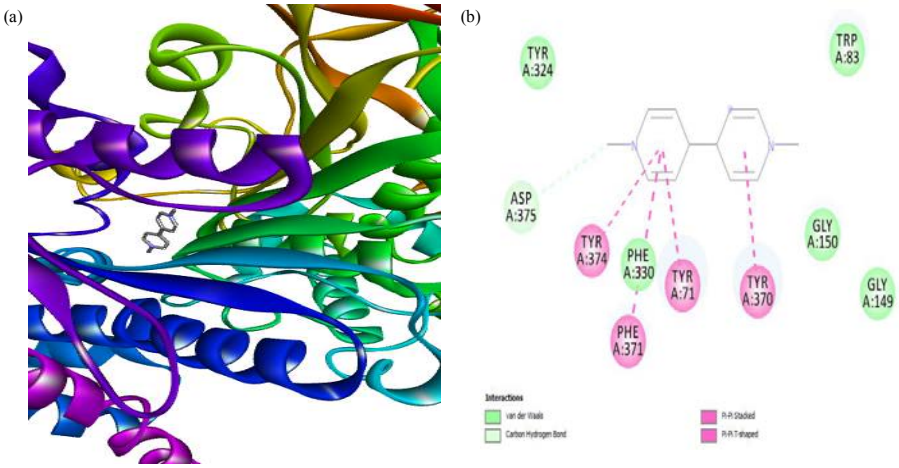


Fig: 11(a-b): (a) 3D and (b) 2D of molecular interaction of acetylcholinesterase amino acid residues with paraquat

DISCUSSION

The present study established a Median Lethal Concentration (LC_{50}) of 3.261 mg PQ/10 g diet for *Drosophila melanogaster*. Exposure to paraquat also resulted in increased mortality, particularly at the higher treatment concentration, demonstrating the acute toxic effect of paraquat in *D. melanogaster*. This finding is consistent with reports of paraquat-induced toxicity and mortality in experimental rodent models[5,14]. These findings indicate that paraquat induces both behavioral and biochemical toxicity, potentially through oxidative stress and reactive oxygen species (ROS) generation. The observed behavioral deficits and reductions in AChE and GST activities are consistent with previous reports of paraquat-induced neurotoxicity and disruption of detoxification pathways in Drosophila. Mehdi and Qamar[6] reported oxidative stress, DNA damage and ultrastructural alterations in the nervous system of paraquat-exposed *D. melanogaster*, while Chaouhan *et al*.[15] reported paraquat-induced neurodegeneration associated with mitochondrial dysfunction in Drosophila. Ramalho *et al*.[7] also reported neurotoxic effects of paraquat and its degradation products in *D. melanogaster*[16]. Furthermore, Ijaz *et al*.[17] reported that paraquat exposure can induce oxidative damage and disrupt antioxidant defense mechanisms, including effects on GST and catalase. These findings support the interpretation that oxidative stress may contribute substantially to the mortality, behavioral impairment and enzymatic alterations observed in the present study.

The molecular docking analysis in the present study demonstrated interactions between paraquat and the selected target enzymes, AChE and GST, providing a possible molecular explanation for the biochemical and behavioral effects observed *in vivo*. The predicted interaction with AChE is consistent with the findings of Alcaro *et al*.[18], who demonstrated through molecular modelling and enzymatic studies that paraquat and related compounds can interact with AChE and potentially influence its activity. This finding provides support for the possibility that the interaction predicted in the present study may contribute to the observed reduction in AChE activity and associated locomotor impairment. The molecular interaction of paraquat with AChE therefore provides a plausible link between the computational and biochemical findings obtained in this study.

The present study also demonstrated an interaction between paraquat and GST, with the observed binding affinity providing a possible molecular basis for the alteration in GST activity following exposure. This finding is consistent with Ritika *et al*.[19], who demonstrated through molecular docking that GST can interact with toxic agrochemicals and xenobiotic compounds, supporting its relevance as a molecular target in chemical toxicity. The predicted GST interaction in the present study therefore complements the biochemical findings and suggests that paraquat may interfere with detoxification processes at the molecular level.

The docking analysis further identified non-covalent interactions, including hydrogen bonds, π-π stacking and alkyl interactions, between paraquat and residues within the binding sites of AChE and GST. These interactions may contribute to ligand recognition and binding stability and may help explain the differences in binding affinities observed between the two enzymes. Such interactions are commonly evaluated in molecular docking studies to characterize ligand-protein recognition and binding behavior[20].

This study has several limitations. First, the molecular docking approach provides theoretical predictions of ligand-protein interactions and does not fully account for protein flexibility, solvent effects, cellular metabolism, or the complex biological environment. Therefore, the predicted interactions between paraquat and AChE or GST cannot be considered definitive evidence of enzyme inhibition. Second, the relatively short exposure duration used in the present study limits conclusions regarding chronic exposure, long-term accumulation and adaptive physiological responses to paraquat. Third, although *D. melanogaster* is a well-established model for toxicological research, differences in metabolism, physiology and detoxification mechanisms between insects and mammals may limit the direct extrapolation of the findings to humans. Future studies should therefore incorporate longer exposure periods, multiple concentrations and direct biochemical enzyme-inhibition assays to validate the molecular docking predictions and further clarify the mechanisms underlying paraquat toxicity.

CONCLUSION

Paraquat exposure induced significant toxic effects in *D. melanogaster*, including increased mortality, impaired locomotor performance and alterations in AChE and GST activities. Molecular docking predicted interactions between paraquat and both enzymes, providing a possible molecular basis for the observed biochemical and behavioral effects. However, docking predictions alone cannot confirm enzyme inhibition or establish causality. Further studies using longer exposure periods, multiple exposure concentrations and direct biochemical enzyme-inhibition assays are required to validate these findings. Overall, this study demonstrates the value of integrating *in vivo* behavioral and biochemical

assessments with *in silico* molecular docking to provide a broader understanding of paraquat-induced toxicity in *D. melanogaster*.

ETHICAL APPROVAL

This study received approval from the Ethical Review Board, University of Jos, Plateau State, Nigeria.

SIGNIFICANCE STATEMENT

This study demonstrates that paraquat exposure can adversely affect both detoxification and cholinergic functions in *Drosophila melanogaster*, providing evidence of its potential ecological and toxicological risks. The observed reductions in glutathione-S-transferase and acetylcholinesterase activities, together with impaired behavioral performance, highlight the usefulness of *D. melanogaster* as a model for investigating pesticide-induced oxidative and neurotoxicity. The molecular docking findings further provide preliminary insights into possible interactions of paraquat with key enzymes. These findings contribute to a better understanding of paraquat toxicity and support further experimental investigation of its molecular mechanisms and environmental impacts.

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