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In Vitro and *In Vivo* Antioxidant Capacity of *Camellia sinensis* (Green Tea) and Its Neuroprotective Effects Against Dichlorvos-Induced Oxidative Stress in Wistar Rats

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ABSTRACT

Dichlorvos (DDVP), an organophosphate pesticide, due to its continuous use has been associated with oxidative stress-mediated neurotoxicity. However, green tea rich in polyphenols may confer protection against DDVP-induced neuronal damage. This study evaluated the *in vitro* antioxidant capacity of green tea extract (GTE) and its protective effects against DDVP-induced oxidative and histopathological alterations in Wistar rats. GTE antioxidant capacity was measured using DPPH and FRAP *in vitro*. Six groups of Wistar rats were used (n=5): Control, DDVP (8 mg/ kgbw), DDVP + GTE (50 mg/ kgbw), DDVP + GTE (100 mg/kgbw), and GTE-only groups (50 and 100 mg/ kgbw). Antioxidant's parameters and histopathology of the brain were measured at the end of the 21 days experimental period. Result obtained showed that GTE exhibited concentration-dependent antioxidant activity, DPPH activity increasing from 31.3% to 57.8% and FRAP absorbance from 0.0869 to 0.58. DDVP exposure significantly ($p < 0.05$) depleted the total protein concentration, SOD, CAT, GST, GPx, and GSH levels, with concomitant significant ($p < 0.05$) elevation of the MDA and nitrite concentrations. The hippocampal cytoarchitecture was severely disrupted showing signs of neuronal degeneration. Treatment with GTE significantly mitigated the observed depletion in the biochemical parameters and also attenuated the histological alterations induced by DDVP exposure in the hippocampal region of the brain. This study suggest that green tea extract possesses possible potent antioxidant activity and protects against DDVP-induced oxidative brain injury, supporting its further investigation as a potential natural intervention for mitigating pesticide-associated oxidative brain damage.

1. INTRODUCTION

Dichlorvos (2,2-dichlorovinyl O, O-dimethyl phosphate (DDVP)) is a widely used organophosphate pesticide primarily used for controlling livestock parasites and agricultural pests (El *et al*., 2020; Sarlak *et al*., 2021). However, its extensive use has raised significant public health concerns due to its toxic effects particularly on the nervous system (Quintino-Ottonicar *et al*., 2023). DDVP is rapidly absorbed via the respiratory system, gastrointestinal tract, and skin, making both humans and animals highly susceptible to its toxic effects following occupational or environmental exposure. On absorption, DDVP causes the overproduction of reactive oxygen species (ROS), thereby overwhelming the endogenous antioxidant defense systems and ultimately causing cellular oxidative damage (Salem *et al*., 2023). This oxidative damage has been implicated in the development of psychological and cognitive dysfunctions (Michaelis *et al*., 2017). In Nigeria, DDVP exposure remains a public health concern, as evidenced by its detection in food commodities, including a 56% detection rate in foods from Lagos (Ingenbleek *et al*., 2019), and

and reported cases of suspected consumption of DDVP-contaminated rice associated with deaths in families in Imo and Anambra States (Clement *et al*., 2021). These reports underscore the need for effective interventions to mitigate the health hazard associated with DDVP-exposure.

Medicinal plants and their derived products are an important source of therapeutic agents and are widely used in the prevention and management of various diseases, particularly in low- and middle-income countries (Ogbole *et al*., 2018). Green tea, derived from *Camellia sinensis* (L.), is among the most widely consumed beverages and has attracted considerable attention because of its diverse health-promoting properties, including anti-obesity, anti-inflammatory, anticancer, and neuroprotective activities (Suzuki *et al*., 2016; Yokogoshi, 2016). These beneficial effects are largely attributed to its high content polyphenolic compounds, particularly catechins, which exhibit potent antioxidant activity by scavenging free radicals, enhancing endogenous antioxidant enzyme activities, and suppressing pro-oxidant enzymes (Shirai *et al.,* 2020). Catechins protect against oxidative damage by inhibiting the oxidation of lipids, proteins, and DNA while strengthening the endogenous antioxidant defense system (Truong & Jeong, 2021; Rajak *et al*., 2023). Consistent with these properties, epigallocatechin-3-gallate (EGCG), a major catechin in green tea, was reported to mitigate diazinon-induced neurotoxicity through modulation of antioxidant and inflammatory pathways (Onukak *et al.,* 2025), suggesting the potential of green tea against pesticide-induced neurological injury.

This study aims to validate further, the antioxidant potential of green tea hydroextract (GTE) against dichlorvos-induced oxidative stress. Evaluates the *in vitro* antioxidant capacity of GTE and investigates its protective effects against dichlorvos-induced oxidative stress, specifically in the brain tissues of Wistar rats.

MATERIALS AND METHODOLOGY

Chemicals

Dichlorvos (100 g active ingredient/L) was procured from Saro Agrosciences LTD, Oyo State, Nigeria. Green tea used is a product of Qualitea, Qualitea Ceylon (PVT) Ltd, Colombo, Sri Lanka. Superoxide dismutase (SOD), catalase (CAT), malondialdehyde (MDA), reduced glutathione (GSH), glutathione peroxidase (GPx), glutathione-S-transferase (GST), and nitrite assay kits were all products of Fortress Diagnostics Limited, Uk. All other chemicals and reagents were of analytical grade and prepared in the laboratory using distilled water.

Experimental Design

Thirty Wistar rats of 135 ± 15 g were obtained and raised in a well-ventilated environment with an ambient temperature of 25 ± 2 °C, humidity of 55 ± 5 % and a 12/12 h light and dark cycle in the Central Animal House of Osun State University, Osogbo, Nigeria. The rats were fed rat chow and water *ad libitum*. All experimental procedures were conducted in accordance with the institutional and internationally accepted guidelines for the care and use of laboratory animals and in accordance with the ARRIVE guidelines. Six groups of Wistar rats were

used (n=5): Control (given only food and water), DDVP, DDVP + GTE (50 mg/kgbw), DDVP + GTE (100 mg/kgbw), and GTE-only groups (50 and 100 mg/kgbw). *Oral administration of DDVP (8 mg/kg body weight) was carried out at three-day intervals, following the previously described protocol of Saka et al. (2021). GTE was administered daily and prepared by infusing 15 g of dried green tea leaves in 1 L of water at 90 °C for 3 min, following the method of Kumar and Yadav (2021).* The oral administration lasted twenty-one (21) days. Ethical approval for the research was obtained from Osun State Ministry of Health, Osogbo, Osun State, Nigeria (OSHREE/PRS/569T/901).

In Vitro Analysis of GTE

The *in vitro* antioxidant capacity of GTE was evaluated using the 2,2-diphenyl-1-picrylhydrazyl (DPPH) radical scavenging assay and the ferric reducing antioxidant power (FRAP) assay following the methods described by Ademoye *et al*., (2018) and Benzie & Strain (1996), respectively.

Preparation of Brain Homogenates

At the end of the experimental period, rats were euthanized using cervical dislocation and the brains excised, rinsed in ice-cold 1.15 % KCl, blotted dry, and homogenised using a teflon homogeniser in ice-cold 0.1 M phosphate buffer (pH 7.4). The homogenates were centrifuged at 10,000 *x g* for 20 min, at 4 ºC and the supernatants were collected and stored at -20 ºC until biochemical analysis.

Biochemical Analysis

Total protein concentration was measured using the method described by Gornall *et al.* (1949). The activities of CAT and SOD were determined according to the methods of Claiborne (1984) and Misra & Fridovich (1972), respectively. GPx and GST activities were analysed following the modified procedures of Matkovics *et al*. (1997) and Habig *et al.* (1974), respectively. GSH levels were estimated using the method of Beutler *et al*. (1963). Lipid peroxidation was evaluated by MDA levels according to Draper and Hadley (1990), while nitrite concentration was determined using the protocol described by Miranda *et al*. (2001).

Histopathological Examination

The excised brain tissues were fixed in 10 % neutral buffered formalin. Following fixation, the tissues were dehydrated through a graded series of ethanol, cleared in xylene, and embedded in paraffin wax. Paraffin-embedded tissues were sectioned into thin serial sections using a microtome, stained using hematoxylin and eosin (H&E), and examined under a light microscope for histopathological alterations (Slaoui and Fiette, 2011).

Statistical Analysis

For statistical analysis, GraphPad Prism version 8.0 (GraphPad Software, USA) was utilized. One-way analysis of variance (ANOVA) and Tukey's multiple comparison post hoc test were used to analyze the data. The mean ± standard deviation (SD) is used to present the results. A priori power analysis was carried out, and $P < 0.05$ was chosen as the threshold for statistical significance.

Results

In Vitro Antioxidant Potential of GTE

The *in vitro* antioxidant capacity of green tea extract (GTE) was evaluated via DPPH and FRAP assays (Figure 1). GTE demonstrated concentration-dependent radical scavenging activity, with DPPH inhibition increasing from 0.5% at 31.3 µg/mL to 57.8% at 1000 µg/mL (IC50 ≈ 912.95 µg/mL). A corresponding progressive increase was observed in the FRAP assay, reaching a peak absorbance of 0.58 at 1000 µg/mL. Across both assays, the antioxidant efficacy of GTE remained consistently below that of the reference standard, ascorbic acid.

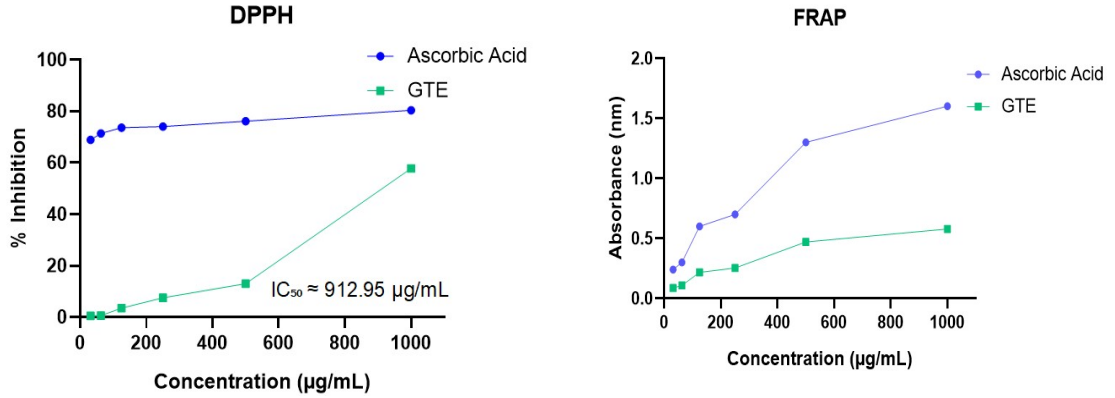


Figure 1: *In vitro* antioxidant capacity of green tea extract compared with ascorbic acid. (a) DPPH radical scavenging capacity *(b) Ferric reducing antioxidant power (FRAP)*

Brain Total Protein Alterations

To assess the impact of toxicity and treatment on protein synthesis and cellular integrity, total protein concentration in the brain tissue was quantified (Figure 2). DDVP exposure induced a marked decline in brain total protein levels relative to the control group ($p = 0.013$). While co-treatment with the lower dose of GTE (50 mg/kgbw) did not completely reverse this depletion, the 100 mg/kgbw dose effectively restored total protein concentrations ($p = 0.0056$) back to baseline control levels. Furthermore, administering GTE alone at either dose caused no adverse deviations, maintaining protein profiles statistically identical to the control group. Quantitative values and statistical indicators are detailed in Table 1.

Brain Oxidative Stress and Antioxidant Status

Exposure to DDVP induced severe oxidative stress in the brain tissue of experimental animals (Figures 3 and 4). This was evidenced by a profound depletion of the antioxidant defense system, marked by a significant reduction in the activities of SOD, CAT, GST, and GPx, alongside a sharp drop in GSH concentration compared to the control group ($p <$

0.001). Concurrently, DDVP exposure triggered significant lipid peroxidation and nitrosative stress, as reflected by elevated levels of MDA ($p = 0.0044$) and nitrite ($p = 0.0025$).

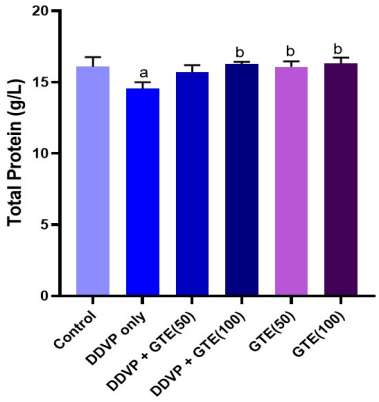


Figure 2: Total protein concentration in the brain of dichlorvos-intoxicated rats treated with green tea extract.

Results are represented as Mean ± SD (n=5). [a] – values are significantly different ($p<0.05$) from the control group; [b] – values are significantly different ($p<0.05$) from the DDVP group.

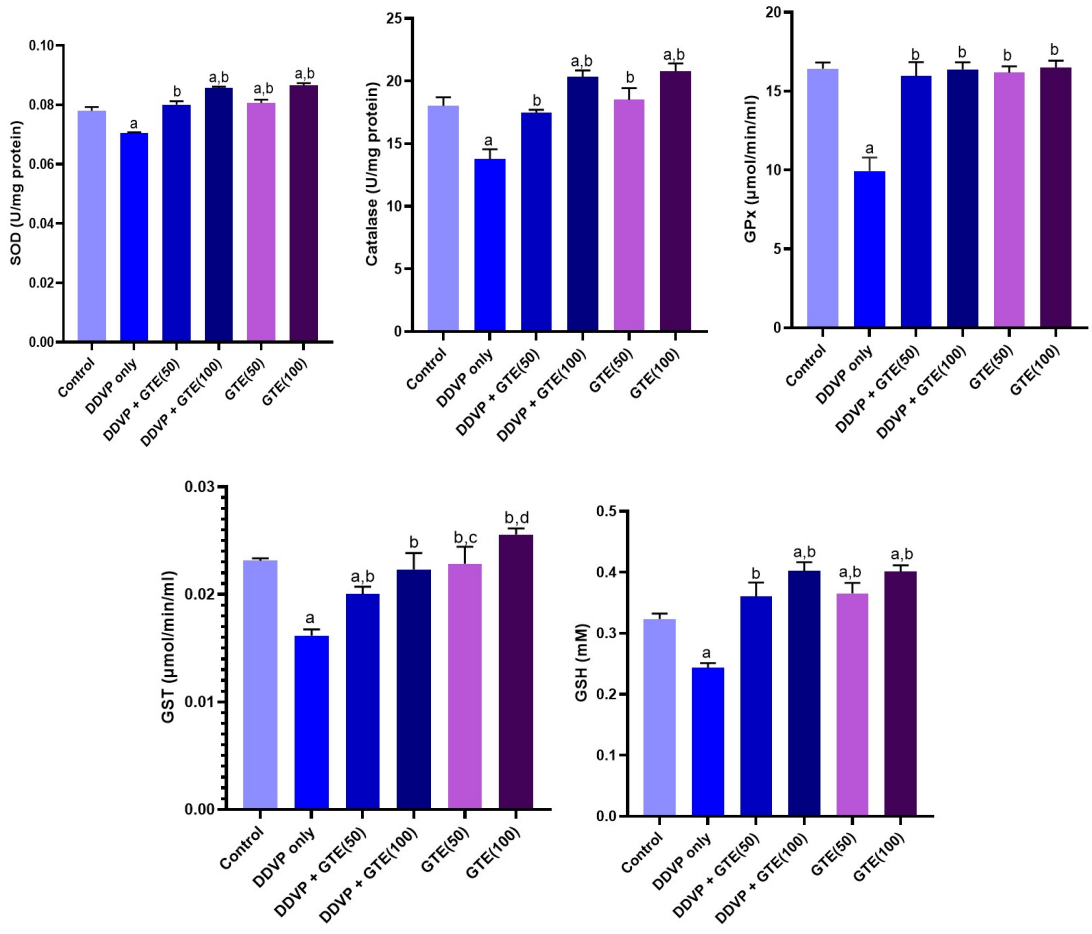


Figure 3: Antioxidant markers in the brain of dichlorvos-intoxicated rats treated with green tea extract. (a) *SOD activities (b) Cat activities (c) GPx activities (d) GST activities (e) GSH.*

Results are represented as Mean ± SD (n=5). [a] – values are significantly different ($p<0.05$) from the control group; [b] – values are significantly different ($p<0.05$) from the DDVP group; [c] – values are significantly different ($p<0.05$) from DDVP + GTE (50); [d] – values are significantly different ($p<0.05$) from DDVP + GTE (100).

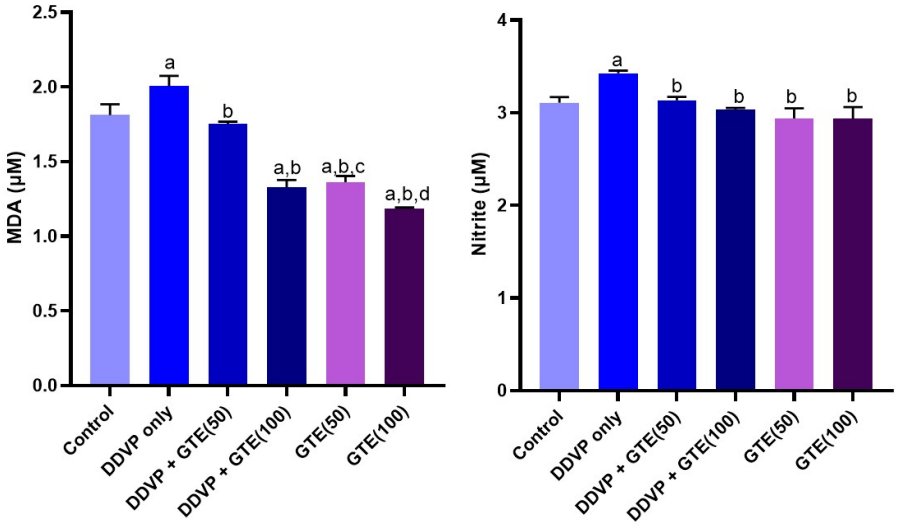


Figure 4: Oxidative stress markers in the brain of dichlorvos-intoxicated rats treated with green tea extract. (a) *MDA concentration (b) Nitrite concentration.*

Results are represented as Mean ± SD (n=5). [a] – values are significantly different ($p<0.05$) from the control group; [b] – values are significantly different ($p<0.05$) from the DDVP group; [c] – values are significantly different ($p<0.05$) from DDVP + GTE (50); [d] – values are significantly different ($p<0.05$) from DDVP + GTE (100).

Protective Effect of GTE against DDVP-Induced Oxidative Stress

Administration of GTE at both 50 and 100 mg/kgbw effectively mitigated DDVP-induced oxidative damage. GTE co-treatment significantly restored all compromised antioxidant enzyme activities and GSH levels ($p < 0.01$), while successfully suppressing the elevated concentrations of MDA and nitrite back toward or below baseline control levels.

Animals treated with GTE alone exhibited an excellent safety profile and, in several parameters, enhanced baseline antioxidant capacity. Both baseline doses of GTE significantly lowered lipid peroxidation (MDA) and increased GSH concentration compared to the control group. While GST and GPx activities remained statistically unchanged relative to controls, the higher dose of GTE (100 mg/kgbw) significantly upregulated baseline SOD and CAT activities ($p = 0.0028$), underscoring its intrinsic antioxidant-boosting potential. Detailed values, means, and statistical breakdowns are summarized in Table 1.

Table 1: Effects of DDVP and Green Tea Extract (GTE) on Brain Biochemical Parameters

Biochemical Parameter	Control	DDVP Only	DDVP + GTE (50 mg/kg)	DDVP + GTE (100 mg/kg)	GTE Only (50 mg/kg)	GTE Only (100 mg/kg)
Total Protein *(mg/g tissue)*	16.08 ± 0.68	14.55 ± 0.44 [a]	15.71± 0.48	16.27 ± 0.15 [b]	16.07 ± 0.39	16.32 ± 0.40
SOD *(U/mg protein)*	0.78 ± 0.001	0.071 ± 0.0002 [a]	0.081 ± 0.001 [b]	0.086 ± 0.0005 [b]	0.081 ± 0.001	0.087 ± 0.001 [b]
CAT *(U/mg protein)*	18.03 ± 0.68	13.75 ± 0.80 [a]	18.51 ± 0.92 [b]	20.32 ± 0.54 [b]	18.51 ± 0.93	20.79 ± 0.61 [b]
GST *(U/mg protein)*	0.023 ± 0.0002	0.016 ± 0.001 [a]	0.020 ± 0.001 [b]	0.022 ± 0.002 [b]	0.022 ± 0.002	0.026 ± 0.002
GPx *(U/mg protein)*	16.42 ± 0.40	9.94 ± 0.86 [a]	15.96 ± 0.88 [b]	16.38 ± 0.45 [b]	16.17 ± 0.40	16.50 ± 0.43
GSH *(µmol/mg protein)*	0.32 ± 0.009	0.24 ± 0.008 [a]	0.36 ± 0.022 [b]	0.40 ± 0.014 [b]	0.37 ± 0.017 [b]	0.40 ± 0.011 [b]
MDA *(nmol/mg protein)*	1.81 ± 0.071	2.00 ± 0.071 [a]	1.76 ± 0.013 [b]	1.33 ± 0.049 [b]	1.36 ± 0.041 [b]	1.19 ± 0.007 [b]
Nitrite *(µmol/mg protein)*	3.11 ± 0.61	3.43 ± 0.027 [a]	3.14 ± 0.036 [b]	3.03 ± 0.017 [b]	2.94 ± 0.11	2.93 ± 0.13

Data expressed as Mean ± SD. [a] $p < 0.05$ compared significantly against the Control group; [b] $p < 0.05$ compared significantly against the DDVP-only group.

Hippocampal Histopathology

Histopathological evaluation of the hippocampus via H&E staining revealed profound structural variations across the experimental groups (Plate 1). Brain sections from DDVP-exposed rats exhibited extensive cellular injury, characterized by severe neuronal degeneration, pyknotic nuclei, and a highly disorganized neuropil matrix. Conversely, co-treatment with graded doses of GTE effectively mitigated this neurotoxicity. Animals receiving GTE demonstrated a dose-dependent preservation of tissue integrity, culminating in the 100 mg/kgbw cohort displaying a near-complete restoration of normal hippocampal architecture and pyramidal cell alignment.

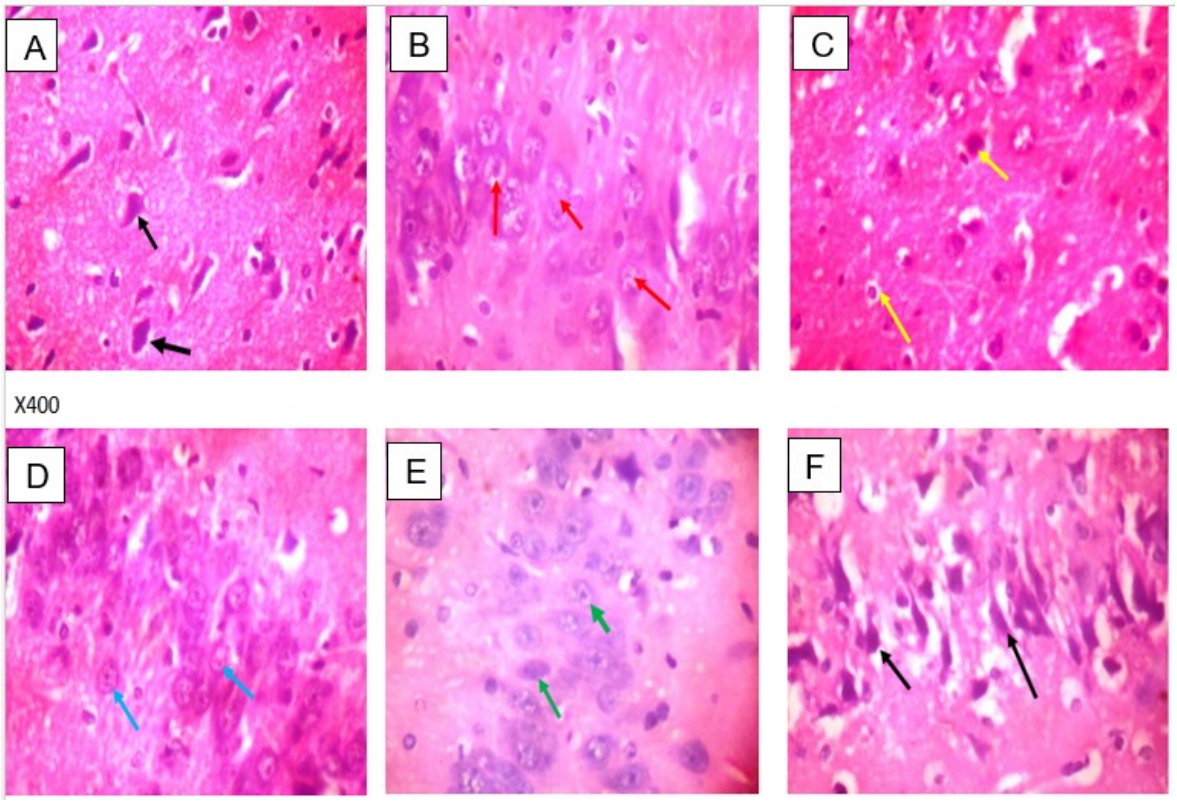


Plate 1: Photomicrographs of H&E-stained hippocampal sections from DDVP-exposed rats.

A: Control group showed normal hippocampal cytoarchitecture with well-preserved pyramidal neurons exhibiting distinct cell membranes, centrally located nuclei, and prominent nucleoli. The neuropil appeared compact and uniformly organized, with no evidence of neuronal degeneration, vacuolation, or inflammatory cell infiltration.

B: DDVP-intoxicated group exhibited marked histopathological alterations characterized by neuronal degeneration (Red arrows), shrunken neurons with pyknotic and hyperchromatic nuclei, cytoplasmic eosinophilia, and prominent vacuolation. The neuropil appeared disorganized with widened intercellular spaces.

C: DDVP-intoxicated rats treated with GTE (50 mg/kgbw) showed partial restoration of hippocampal architecture. Neuronal morphology was moderately improved with reduced neuronal shrinkage and vacuolation (yellow arrows), although mild nuclear condensation and cytoplasmic alterations persisted.

D: DDVP-intoxicated rats treated with GTE (100 mg/kgbw) demonstrated marked histological recovery, characterized by well-preserved neuronal morphology, organized neuropil, minimal vacuolation, and only occasional degenerating neurons.

E and F: Rats treated with GTE alone (50 and 100 mg/kgbw) exhibited normal hippocampal histoarchitecture comparable to the control group, with intact neuronal cell bodies, well-defined nuclei, and organized neuropil.

Discussion

In the present study, GTE exhibited concentration-dependent DPPH radical-scavenging and ferric-reducing antioxidant activities, indicating its electron-donating properties and ability to neutralize free radicals (Cömert & Gökmen, 2017). The DPPH and FRAP assays are widely used to evaluate the *in vitro* non-enzymatic antioxidant capacity of medicinal plants (Alfarrayeh *et al*., 2022). The antioxidant activity of GTE is primarily attributed to its abundant polyphenolic constituents, which act as potent free radical scavengers and reducing agents (Magiera *et al.,* 2022; Allagui *et al*., 2022). The present results are consistent with those of Bartoszek *et al*. (2018), who reported that green tea exhibits strong antioxidant activity due to its rich phenolic composition. Nevertheless, GTE has a lower antioxidant potency than that of the reference antioxidant, ascorbic acid, which may be largely due to synergistic interactions among its bioactive components.

The brain is especially prone to oxidative injury because it has a relatively weak antioxidant defense system, high concentration of lipids, and a substantial demand for oxygen (Carocci *et al*., 2018). Excessive ROS production disrupts oxidative balance and has been implicated in the onset and progression of several neurological disease, including Alzheimer diseases (Okoroiwu & Iwara, 2018; Udodi *et al*., 2023). In the present study, DDVP exposure impaired the brain's antioxidant defense system leading to oxidative stress that may contribute to cellular damage and neuronal dysfunction (Trivedi *et al*., 2021; Jalili *et al*., 2022). Elevated nitrite levels observed following DDVP exposure suggest increased nitric oxide (NO) production, which readily reacts with superoxide anions to form peroxynitrite, a highly reactive oxidant capable of inducing lipid peroxidation, protein oxidation, DNA damage, and neuronal dysfunction (Ferrer-Sueta *et al*., 2018). Also, the observed increase in MDA levels may indicate enhanced lipid peroxidation of neuronal membrane phospholipids, suggesting increased oxidative damage following DDVP exposure (Ajayi *et al*., 2020). These findings correspond with the observations of Imam *et al.* (2018), who reported that DDVP exposure induced oxidative stress and markedly impaired antioxidant capacity. Similarly, Saka *et al*. (2024) observed increased MDA levels accompanied by decreased antioxidant enzyme activities following dichlorvos exposure. This indicates a similar pattern of oxidative imbalance following DDVP exposure and provides further support for the involvement of oxidative stress as major mechanisms underlying DDVP-induced neurotoxicity.

Conversely, treatment with GTE significantly boost the brain antioxidant status of the experimental animals, an effect largely attributed to its abundant polyphenolic constituents, which readily donate hydrogen atoms or electrons to neutralize ROS and terminate oxidative chain reactions (Truong & Jeong, 2021; Farhan, 2022). Furthermore, green tea polyphenols have been reported to enhance endogenous antioxidant defenses by boosting the

antioxidant enzymes activities, including SOD, CAT, GPx, and GST, while preserving GSH levels, thereby mitigating oxidative stress and maintaining cellular redox homeostasis (Martins *et al*., 2017; Ibrahim *et al*., 2019; Zhang *et al*., 2020). Consequently, the restoration of these antioxidant biomarkers observed in the rats brain in this study, suggests that GTE effectively protected the brain against DDVP-induced oxidative damage by reinforcing the endogenous antioxidant defense system.

Structural damage to the hippocampus is frequently associated with cognitive and behavioural deficits following exposure to neurotoxic agents such as dichlorvos (DDVP), owing to its essential role in learning, memory consolidation, emotional regulation, spatial cognition, and social behaviour (Todorov *et al*., 2019). DDVP exposure in the present study induced marked histopathological alterations in the hippocampus. However, treatment with GTE attenuated these histopathological changes, preserving the structural organization of the hippocampus and demonstrating its ameliorative effect against DDVP-associated brain oxidative and histological alterations. The preservation of hippocampal architecture observed following GTE treatment is likely attributable to its potent antioxidant activity, which limits oxidative damage and maintains neuronal integrity.

In conclusion, this study demonstrates that *Camellia sinensis* (green tea) hydroextract possesses robust neuro-ameliorative potential against dichlorvos-induced neurotoxicity. GTE exhibited potent *in vitro* radical scavenging activity and effectively rescued the in vivo brain antioxidant defense system. This was characterized by a significant reduction in lipid peroxidation and nitrosative stress, alongside the preservation of hippocampal cytoarchitecture. Taken together, these findings indicate that the neuroprotective efficacy of green tea is mediated through its capacity to upscale antioxidant defenses and suppress oxidative damage. Consequently, GTE represents a promising, accessible natural therapeutic agent for mitigating pesticide-induced oxidative brain injuries.

Recommendation

These findings suggest that green tea extract could serve as a valuable dietary countermeasure against pesticide-associated oxidative and neurotoxic damage. However, further mechanistic studies are required to establish optimal dosing regimens, intervention durations, and long-term safety profiles. Additionally, translational clinical research is warranted to determine whether the protective neuro-architectural effects observed in this Wistar rat model are fully applicable to human populations facing occupational or environmental pesticide exposure.

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