Al-Mukhtar Journal of Basic Sciences 23 (2): 87-95, 2025
Doi: https://doi.org/10.54172/ytye2x47

Research Article

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Antioxidant Role of *Cleome Droserifolia* Extract on Cyhalothrin-Induced Oxidative Stress in Male Albino Rats

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Received:
09 July 2025

Accepted:
29 August 2025

Publish online:
31 August 2025

Abstract

Cyhalothrin (Cy) is a pyrethroid pesticide used globally to control pests in farming and inside homes. *Cleome droserifolia* extraction (Cd) is a potential antioxidant that protects tissues from oxidative damage. The current study looks at the antioxidant properties of Cd on Cy-caused oxidative harm in rats. A group of 20 male Wistar rats were separated among 4 distinct groups: Group I acted as the control; group II administered Cy i.p. just (6.2 mg/kg b.wt.); group III obtained Cd solely (100 mg/kg b.wt., p.o.) for 8 weeks; and group IV administered Cd as a form of protection every day for 8 weeks, then received Cy (i.p.) 3 times each week during 2 weeks. The findings indicated that Cy produced a considerable decline in body weight and markedly decreased serum superoxide dismutase (SOD), glutathione (GSH), and catalase (CAT). Furthermore, Cy elicited a considerable rise in serum hydrogen peroxide (H_2O_2), nitric oxide (NO), and malondialdehyde (MDA). In contrast with Cy-treated rats, Cd in the protective rats significantly recovered the changes in body weight, antioxidant, and oxidative stress indicators. In conclusion, the findings of this investigation demonstrated that Cd extract has antioxidant properties against oxidative stress in Cy of male albino rats.

Keywords: *Cleome Droserifolia*; Cyhalothrin; Antioxidant; Oxidative Stress; Rat.

INTRODUCTION

Cyhalothrin (Cy), a Pyrethroid pesticide, works wonderfully against a variety of pests (Fetoui et., al., 2008; Wang & Wang, 2017). Its act by disrupting the proper functioning of the neurological system in an organism, leading to in immobility or death (Velmurugan et., al., 2007), Cy can be hazardous to animals (Atamanalp et., al., 2002; Sakr and Rashad, 2023), by inducing oxidative damage, which results in the formation of free radicals, alterations in antioxidant activity, and lipid peroxidation (LPO) (Ender & Onder, 2006; Yadav et., al., 2023). While Cy can't create reactive oxygen species directly, it indirectly generates various radicals such as superoxide radical (O_2^-), and hydroxyl radical ($OH^·$), thus causing damage to proteins, lipids, and DNA by oxidation (Kale et., al., 1999; Abdul-Hamid et., al., 2020).

Meanwhile, antioxidants/oxidation inhibitors are regarded as vital supplements due to their numerous health advantages, and they are commonly employed in the food sector as LPO-inhibiting agents (Scherer & Godoy, 2009). Synthetic antioxidants collect throughout the bodi-

ly tissues, leading to destruction of the liver and cancer. Such issues do not occur when antioxidants obtained from spices and herbs are employed. The extracted substances are harmless, maybe nutritious, and have medicinal properties (Velioglu et., al., 1998 and Rubilar *et., al*., 2006*)*. Amongst these plant resources, *Cleome droserifolia* was chosen for the current investigation. The watery extract of *Cleome droserifolia* (Cd) has been used as hepatoprotective, hypoglycemic, antioxidant, antihistaminic, antimicrobial agent, relaxant, and tranguilizing effects (El-Askary, 2005 & Azab, 2025). Cd is an essential component of antioxidants (Rice Evans, 2004; Nagy and Mohamed, 2015); organic antioxidants improve serum antioxidant activity and lower the possibility of certain disorders (Prior *et., al*., 2005). Cd's secondary metabolic products, including flavonoids and phenolics, are powerful free radical hunters (El-Naggar et., al., 2005; Al-Zail & Kamies, 2021).

The present investigation will look into how antioxidants from *Cleome droserifolia* extract (Cd) can protect male rats from the harmful effects of cyhalothrin (Cy).

MATERIALS AND METHODS

Animals

A group of 20 male Wistar rats of adult age and albino type, with an average body weight of 120±10 g/animal, representing 2 -3 months of age, were employed in the current study.

Cyhalothrin

Sigma-Aldrich, located in St. Louis, Missouri, USA, was the source of cyhalothrin (Cy). The drug was injected intraperitoneally (i.p.) with a concentration equivalent to 1/10 of the median lethal dose (LD50), specifically, 6.2 mg/kg/b.w (Fetoui et., al., 2013).

Natural antioxidant (*Cleome droserifolia*)

During the actual day of the experiment, the dried extract was dissolved in water that had been distilled. Then a dosage of 100 mg/kg/b.w were gavaged orally (El-Naggar et., al., 2005).

Experimental procedure

The animals were separated to 4 distinct groups (5 rats in each group) as following: Group I: control, that got distilled water solely during the period of study (p.o.) daily; Group II: Cy group, which was given cyhalothrin only (6.2 mg/kg b.w.,i.p.) 3 times a week for 2 weeks; Group III: Cd group, in which the animals received *Cleome droserifolia* extract only (100 mg/kg b.w.,p.o.) in distilled water daily during 8 weeks; Group IV (Protective by Cd): animals were given Cd (p.o.) daily for 8 weeks. In the 7^{th} week, they received Cy (i.p.) 3 times a week for 2 weeks. Blood samples were taken through the orbital sinus at the final stage of the experiment and put in a sterile centrifuge tube. Serum was collected from specimens after they had been spun for 15 minutes at 3000 rotations per minute to analyze antioxidant biomarkers and oxidative stress.

Determination of total body weight

Animals of the control and treated groups were weighed prior to the time of treatment and again prior to sacrifice.

Determination of antioxidant biomarkers

The enzyme activities of catalase (CAT), superoxide dismutase (SOD), and glutathione (GSH) in blood were measured using the technique of (Nishikimi et., al. 1972) and the methodology of (Aebi, 1984). Colorimeter measures analysis using the method of kits provided by (BIODIAGNOSTIC).

Determination of oxidative stress markers

Malondialdehyde (MDA), nitric oxide (NO), and hydrogen peroxide (H_2O_2) levels were determined calorimetrically in the serum depending on the methodologies of (Ellman, 1959); Ohkawa et., al. 1979; Green et., al. 1982). Colorimeter measures analysis using the method of kits provided by (BIODIAGNOSTIC).

Statistical analysis

All of the findings were analyzed using SPSS, a statistical program for Windows, version 17.0. The standard error of this difference was estimated using analysis of variance (ANOVA), which displays the variance or difference between different means (Tello & Crewson, 2003).

RESULTS

Effect of Cd on Cy-induced alteration in total body weight

From the present investigation, table (1) clearly shows that the data concerning the control rats (GI) and Cd group (GII) demonstrated a rise in body weights. The mean body weight at the beginning of the experiment was 120±10 g/animal and reached (149.88±1.32) upon completion of the experiment. In contrast, there was an extraordinarily substantial decline in the average value of Cy rats (GII) that recorded (90.52±4.30) compared to the control group. Furthermore, the protective group by Cd (GIV) showed a substantial rise ($p<0.05$) in total body weight (144.94±2.39) as opposed to the Cy group.

Table:(1). The protective role of Cd on total body weight in treated groups with Cy.

Groups		Total body weights (g)
Group I	Control	$149.88±1.32^{ab}$
Group II	Cy	$90.52±4.30^{c}$
Group III	Cd	$155.74±2.33^{a}$
Group IV	Protection by Cd	$144.94±2.39^{b}$

Results are presented as means ± S.E. (n=5 per group).
Averages in columns without similar elevated script letters (a, b, c) varied substantially ($p<0.05$).

Effect of Cd on Cy-induced alteration in antioxidant biomarkers

The results provided by Table (2) demonstrated a very highly substantial reduction in GSH, SOD, and CAT of Cy group (GII) with a mean value of (0.401±0.04, 2.131± 0.07 and 0.488±1.01), respectively, as contrasted with the untreated group. While a very considerable increase was noticed in the average antioxidant biomarkers of the protective group by Cd (GIV) with a mean value of (1.032±0.08, 5.481±1.09, and 0.949±1.41), respectively, relative to the Cy group. Furthermore, the average of Cd alone group **(**GIII**)** did not demonstrate any significant variations from the normal animals.

Table: (2). The protective role of Cd on antioxidant biomarkers in treated groups with Cy.

Groups		Parameters		
		GSH nmol/ml	SOD IU/L	CAT mM/L
Group I	Control	$1.237±0.03^{ab}$	$7.341± 1.04^{a}$	$1.547±0.02^{a}$
Group II	Cy	$0.401±0.04^{c}$	$2.131± 0.07^{c}$	$0.488±1.01^{c}$
Group III	Cd	$1.444±0.16^{a}$	$7.623±0.21^{a}$	$1.657±0.21^{a}$
Group IV	Protection by Cd	$1.032±0.08^{b}$	$5.481±1.09^{b}$	$0.949±1.41^{b}$

Results are presented as means ± S.E. (n=5 per group).
Averages in columns without similar elevated script letters (a, b, c) varied substantially ($p<0.05$).

Effect of Cd on Cy-induced alteration in oxidative stress markers

Table (3) shows a remarkable rise in blood levels ($p<0.05$) of nitric oxide (NO) (1.152±0.05), hydrogen peroxide (H_2O_2) (11.411± 0.17) and malondialdehyde (MDA) (4.201±0.06) in Cy group (GII) relative to control groups, but significant decline ($P<0.05$) was showing in serum oxidative stress markers of protective group as opposed to the control rats by mean values 0.611±0.08, 4.562±0.13, 0.939±0.02, respectively. In any case, the Cd-alone group **(**GIII**)** did not exhibit any noteworthy variations from the untreated group.

Table: (3). The protective role of Cd on oxidative stress markers in treated groups with Cy.

Groups		Parameters		
		NO (nmol/ml)	H_2O_2 (µM/l)	MDA (nmol/ml)
Group I	Control	0.611±0.08^{b}	4.562±0.13^{c}	0.939±0.02^{c}
Group II	Cy	1.152±0.05^{a}	11.411±0.17^{a}	4.201±0.06^{a}
Group III	Cd	0.498±0.65^{b}	3.469±0.05^{c}	1.082±0.04^{c}
Group IV	Protection by Cd	0.901±0.04^{ab}	8.909±0.11^{b}	2.950±0.06^{b}

Results are presented as means ± S.E. (n=5 per group).
Averages in columns without similar elevated script letters (a, b, c) varied substantially ($p<0.05$).

DISCUSSION

The current investigation found a significant reduction in total body weight in the group of rats that received Cy alone (GII) in comparison to the control (GI). The drop in body weight in male rats administered Cy is most likely related to diminished appetite and/or metabolic disruption resulting from significant damage to hepatic tissue. This suggestion is consistent with (Madbouly, 2003) and (Prashanthi et., al. 2006), who said that an insecticide generated noticeable harm in the hepatic cells and impacted the processes of metabolism in the liver, and that result was attributed to lower consumption of food and water in treated mice DZN group. Furthermore, in this investigation, Cy considerably reduced SOD, CAT and GSH, while considerably raising H_2O_2, NO, and MDA. Cy toxicity could be caused by the production of cyanohydrins, which are volatile in biological conditions and degrade into cyanide and aromatic compounds, potentially acting as an inducer of free radicals in humans. These results align with (Madkour, 2012; Sakr & Rashad, 2023) in the evaluation of antioxidant properties in Cy-treated animals. Pesticides cause oxidative damage, as a result, free radicals are formed, the amount of oxidation inhibitors changes, and lipid peroxidation occurs. It forms different radicals like (O^{2-}) and ($OH^{\cdot}$), producing harm to lipids, amino acids, and DNA through oxidation (Ender & Onder, 2006; Wang & Wang, 2017).

A considerable elevate in tissue MDA concentration and a decline in tissue GSH amount as well as SOD function, result from the inability of the defense mechanism of oxidation inhibitors to offset the inflow of radicals induced by the Cy exposure (Fetoui et., al., 2010). Lack of tissue GSH and SOD constitutes one of the key causes that allow for lipid peroxidation and resulting harm to tissues (Huang et., al., 2003; Abdul-Hamid et., al., 2020). Using the findings from the current investigation, (El-Demerdash, 2007; Fetoui et., al. 2009; Yadav et., al. 2023) found that Cy injection led to a considerable rise in MDA generation. fatty acids peroxidation occurs when free radicals react with lipids, and it is thought to be a key feature of the cellular injury brought by free radical attack (Hoek & Pastorino, 2002; Abdul-Hamid et., al., 2020).

The protective groups by Cd (GIV) showed a significant increase in total body weight as compared

to the Cy group (GII). Similar results were demonstrated by (El-Shenawy & Abdel-Nabi, 2004), who discovered a significant improvement in body weight in diabetic rats given Cd extract. This demonstrates that the Cd extract decreases the formation of oxygen radicals that are free inside the tissues, leading to diminished oxidative harm to cells and higher amounts of antioxidant activity possible in Cy rats. In line with earlier literature, (Kumar et., al. 2009; EL-Khawaga et., al. 2010), the cause is linked to the presence of various flavonoids in Cd extract. Furthermore, in the current research, therapy of Cd extract caused substantial enhancements in CAT, GSH, SOD, MDA, NO, and H_2O_2. This extract's antioxidant activities can be described in part by the existence of glycosidic flavonoids, including quercetin, rutin, kaempferol, luteolin, isorhamnetin, and phenolic acids, which were previously found (Abdel Motaal et., al., 2011; Aparadh et., al., 2012).

The current data are consistent with those published (El-Naggar et., al. 2005), who revealed that Cd extract at rates of 100 and 200 mg/kg demonstrated considerable antiperoxidative action in alloxan rats with diabetes. Their results show a considerable increase in lipid peroxidation in rats that receive alloxan, which is reduced by Cd extract. Similarly, to the current results, (Fushiya et., al. 1999) identified, separated, and studied the inhibitory impact of two novel flavonoids from Cd extract on nitric oxide generation in macrophages that were stimulated in vitro. Additionally, (Cao et., al., 1998; El-(Shenawy & Abdel-Nabi, 2004; Nagy & Mohamed, 2015) showed that giving Cd extract resulted in an important rise in the amount of GSH in rats suffering from diabetes. That might suggest that the extract enhances GSH manufacturing and/or lowers oxidative stress, resulting in less GSH breakdown. likewise, levels of MDA were considerably lower in diabetic rats than in healthy control rats.

The raised overall antioxidant power of diabetic blood rats following Cd extract administration could be related to antioxidant uptake. Furthermore, treating rats with diabetes using Cd extract increased the rate of action of glucose-6-phosphate dehydrogenase (G6PDH). G6PDH serves a crucial part in glutathione production, based on the following research information: G6PDH regulates the proper amount of NADPH. Consequently, NADPH sustains GSH concentrations in hepatocytes, and GSH defends cells from ROS-induced oxidative harm. Cd extract induced an elevate in hepatic GSH concentration may improve the GSH/GSSG balance and reduce liver lipid peroxidation, hence improving physiological functioning (Mehta et., al., 2000). Evidence suggests that certain phytochemicals found in this extract, such as flavonoids, play a major role in treating or retarding a wide spectrum of diseases and are reported to possess anti-oxidative and anti-inflammatory properties (Al-Zail & Kamies., 2021)

CONCLUSION

The findings of this investigation showed that the *Cleome droserifolia* (Cd) extract has antioxidant protection effects against oxidative stress of cyhalothrin (Cy) of male albino rats.

ACKNOWLEDGEMENT

I would like to thank my institution, Omar Al-Mukhtar University, and the Faculty of Science, especially the Department of Zoology, for their moral support and for providing a suitable environment for study.

ETHICS

Authors should address any ethical issues that may arise after the publication of this manuscript.

Duality of interest: The authors declare no conflict of interest

Author contributions: Contribution is equal between authors.

Funding: The authors did not receive any funding (institutional, private, and/or corporate financial support)

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