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Original Article

Biochemical Changes Induced by the Pesticide Thiacloprid in Rats: The Protective Role of Curcumin Against Environmental Pollutants

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Abstract

This study assessed how effective curcumin is against Thiacloprid toxicity in male rats. Thiacloprid exposure led to obvious oxidative stress, indication showing the significantly increased levels of lipid peroxidation, diminished GSH levels, and severely inhibited activities of the enzymatic antioxidant defense system. In addition, Thiacloprid altered plasma pituitary-gonadal hormones, induced testicular histopathological changes and caused a significant reduction in sperm parameters (sperm count, motility, viability), combined with systemic hepatotoxicity and biochemical derangement. On the other hand, co-administration of Curcumin ameliorated these toxicological hazards by harmonizing cellular redox equilibrium as well as enhancing antioxidant enzyme activities, maintaining histopathology of testicular architecture and modulating hormonal and biochemical parameters. These findings demonstrate the potent therapeutic potential of Curcumin as a natural protective bioprotectant against pesticide-induced organ toxicity and reproductive failure.

Key words: Thiacloprid; Rats; Curcumin; analysis.

Introduction

Thiacloprid, an insecticide, has a close relationship to imidacloprid's composition and mode of action. It functions by blocking nicotinic acetylcholine receptors, which disrupts the insect's nervous system (1). Thiacloprid has both cytotoxic and genotoxic effects; it significantly increases the production of micronuclei in the marrow cells of rat bone and human peripheral blood lymphocytes (2). Thiacloprid-induced endocrine disturbance caused by alcoholism may lead to tumors and glandular hypertrophy (3). Several studies has indicated a relationship within reproductive with pesticide exposure problems via disturbing neuro transmission and endocrine mechanisms, which changes physiological conditions, of reproductive organs and disrupts spermatogenesis (4). in imitating natural endogenous hormones with interfering and their actions in the endocrine systems, this contaminants alter sexual identity, fertility, or may be behavior by acting such as endocrine disruptors (EDCs) (5).

Curcumin has long been used in Asian medicine for its medical properties (6)**.** It is because of this that we put our curiosity in curcumin, a bright yellow phenolic pigment taken from the rhizome of *Curcuma longa*. Its antihyperglycemic and anticancer benefits have been attributed to its beneficial biological activities as an antioxidant, anti-inflammatory, antidiabetic, and antitumor agent (7). Curcumin has been observed to afford protection against cardiovascular oxidative stress-mediated

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cardiomyopathy, neuropathy, nephropathy, and hepatotoxicity, along with testicular dysfunction. (8). Curcumin, a pro-oxidant, is a potent bioprotectant with numerous therapeutic applications when exposed to transition metal ions (Cu and Fe). This treatment successfully neutralizes ROS, including superoxide anion, hydroxyl radical, singlet oxygen, peroxynitrite, and nitric oxide (9). But it's unclear if curcumin helps with testicular damage via lowering nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (10).

Methodology

1. Curcumin was obtained from Corbate, UK, as a pure powder.

2. Sigma - Aldrich (UK) delivered 99.6% pure thiacloprid, an organic molecule with the molecular formula C10H11ClN4.

Animals and experimental design

Forty male albino Wistar rats (170–190 g) were acclimatized for two weeks under standard laboratory conditions (12-h light/dark cycle, wire-bottom cages) with ad libitum access to distilled water and a standard pellet diet (55% corn starch, 20% casein, 5% salt mixture, 5% vitaminized starch; Egyptian Company of Soap and Oils, Kafr - Elzayat, Egypt). The study was approved by the Regional Animal Welfare Committee and is compliant with NIH guidelines for animal experimentation. After acclimatization, forty rats were divided randomly into 4 equal groups (n = 10 for each group) and orally treated once daily for three weeks as follows:

Group I (Control): Received distilled water.

Group II (Curcumin): Administered curcumin at 800 mg/kg body weight (11).

Group III (Thiacloprid): Administered thiacloprid at frac {1} {10} {LD}_ {50} (156{mg/kg} body weight; oral rat {LD}_ {50} = 1563mg/kg) (12).

Group IV (Curcumin + Thiacloprid): Co-administered curcumin (800mg/kg) and thiacloprid (156 mg/kg).

Blood sample collection and tissue preparations

At the conclusion of the methodology period, all animals were euthanized humanely. Blood samples with heparin as an anticoagulant were collected for haematological analyses. After centrifugation at 860 × g for 20 min, plasma was isolated and stored at −80 °C until the parameters of interest in plasma. The freshly isolated testes were washed with the chilled physiological saline solution (0.9%) and gradually dissected to separate from surrounding adipose and connective tissues. The testes were then minced and homogenized with a Potter–Elvehjem homogenizer in chilled 0.25 M sucrose buffer at a concentration of 10% (w/v). Homogenates were centrifuged for 20 min at 4 °C and 10,000 × g to discard the cellular debris, and the supernatants retrieved were stored at −80 °C until analysis of the selected parameters.

Markers of oxidative stress in testes

The assay for malondialdehyde (MDA) as a measure of lipid peroxidation index in testicular homogenate was done using thiobarbituric acid-reactive substances (TBARS) and was measured at 532 nm employing thiobarbituric acid [2,6-dihydroxypyrimidine-2-thiole; TBA]. Quantitative determination of TBARS was done basically according to a method established by (13). SOD activity was determined as described previously (14). The glutathione peroxidase (GPx) activity was measured according to the method of Sheehan and Dennis (15) in testicular homogenate. The activity of glutathione S-transferase (GST) was measured as previously described in [16]. The activity of catalase (CAT) was assessed in testicular homogenate using the Luck method by the catalysis of hydrogen peroxide decomposition 17. The content of reduced glutathione (GSH) was determined using the method described by Griffith, 1980, with metaphosphoric acid and5,5′-dithiobis (2-nitrobenzoic acid) (DTNB) for color development and reading its density at 412

nm; All assays were performed as per the method of (18). All the above assays were performed as per the manual instructions of Bio Diagnostic Kit, Egypt.

Determination of reproductive hormone Testosterone

Testosterone levels were measured utilizing a quantitative ELISA kites measurementr of testosterone in rats; the assays were done strictly according to **(19).**

Semen characteristics

Preparation of the epididymis for assessment of sperm count, motility & morphology, as well as Computer Assisted Semen Analysis (CASA System; Germany) with Olympus microscope (Olympus, Tokyo, Japan) was utilized according to the method of [20]. From each rat, a total of 200 spermatozoa were assessed and scored according to the strict morphology criteria based on (21), as normal or abnormal.

Findings and Discussion

Figure 1 demonstrated a notable rise in the concentrations of TBARS and MDA in the testes. Curcumin + Thiacloprid-treated rats had significantly lower TBARS and MDA concentrations than the Thiacloprid-treated group, but Thiacloprid-treated rats had higher LPO indicators than the control group. However, glutathione (GSH) levels were much lower in rats that had received thiacloprid. Furthermore, co-administration of curcumin with thiacloprid significantly raised GSH levels in rats treated with thiacloprid. After receiving curcumin alone, the testes' GSH content considerably increased while their TBARS greatly dropped.

The administration of Thiacloprid induced severe testicular oxidative stress, evidenced by elevated lipid peroxidation and significant depletion of non-enzymatic cellular defenses **(22).** Curcumin co-treatment effectively mitigated the damage, protecting membrane integrity against pesticide toxicity and restoring intracellular redox balance **(23, 24).** This protective action is consistent with previous findings on Curcumin's power to quench free radicals and maintain cellular antioxidant status **(25).**

Figure 2 shows antioxidant enzyme activity in the Testes (SOD, CAT, GPx, GR, and GST). The activity of antioxidant enzymes was significantly decreased in rats treated with thiacloprid compared to the untreated controls. But those rats fed curcumin plus thiacloprid did show a significant increase in antioxidant enzyme activity when compared to the thiacloprid group. In contrast, the activity of antioxidant enzymes in the testis of rat only received curcumin was tremendously increased.

Thiacloprid induced a significant decrease in the enzymatic antioxidant defenses in the chemically exposed testis, reason why animals are subjected to severe oxidative stress and tissue injury (25). Co-treatment with Curcumin remarkably maintained the enzyme activities, which helps to enhance biological barriers against pesticide toxicity (22, 23). This protective effect is consistent with Curcumin's well-documented ability to reverse chemical-induced interference in critical enzyme systems that results in cellular redox dysregulation (24).

Results shown in (Figure 3) indicate the presence of pro-inflammatory cytokines (TNF-α & IL-6) in the testes. The results were significantly improved when the thiacloprid groups were compared to the control group. P53 in the testes dramatically decreased in the thiacloprid-treated rats, and the number of rats receiving curcumin treatment was significantly lower than that of the control group. Furthermore, compared to Thiacloprid alone, P53 changed very little in Thiacloprid + Curcumin.

Exposure to Thiacloprid induced severe reproductive toxicity and disruption of the pituitary-gonadal hormonal axis in male rats **(26).** Concurrent administration of Curcumin significantly restored

hormone levels and preserved testicular function against pesticide toxicity **(22, 23).** This restorative effect demonstrates Curcumin's broad capacity to mitigate endocrine dysfunction and protect tissue physiology from chemical damage **(27).**

Thiacloprid medication significantly decreased the amount of testosterone in rat serum when compared to the control, according to data shown in Figure 4. Rats given curcumin alone showed negligible changes in the tested parameters. Otherwise, the concomitant use of curcumin and thiacloprid resulted in a considerable increase in rat serum testosterone compared to the thiacloprid-treated group, reversing these hormonal levels.

Thiacloprid toxicity produced severe histopathological alterations in testicular architecture and a significant decline in sperm count, motility, and viability **(25).** Co-administration with Curcumin effectively attenuated these structural damages, preserving seminiferous tubule morphology and improving epididymal sperm characteristics **(22,28).** These restorative effects underscore Curcumin's therapeutic capacity to protect germ cells and preserve male reproductive cytoarchitecture against pesticide-induced damage **(23).**

Data represented in Figure 5 depict that the percentages of sperm abnormalities were much higher and sperm count and motility were severely reduced in rats administered thiacloprid as compared to the control. In contrast, rats treated with curcumin had only a few and relatively minor changes in the investigated parameters. Alternatively, the combination of curcumin and thiacloprid modified sperm quality compared to the group additionally treated with thiacloprid.

The exposure to thiacloprid resulted in a high level of hepatotoxicity, as suggested by significant changes in serum liver enzyme markers (25). The biochemical disturbances induced by treatment with pyrrolizidine alkaloids were significantly ameliorated when combined with curcumin, restoring hepatic biomarkers towards physiological control (22, 24). Also, this protective mechanism signifies the systemic applicability of curcumin's effect against the toxic effects of pesticides on certain organs and organ systems involved in metabolism (29).

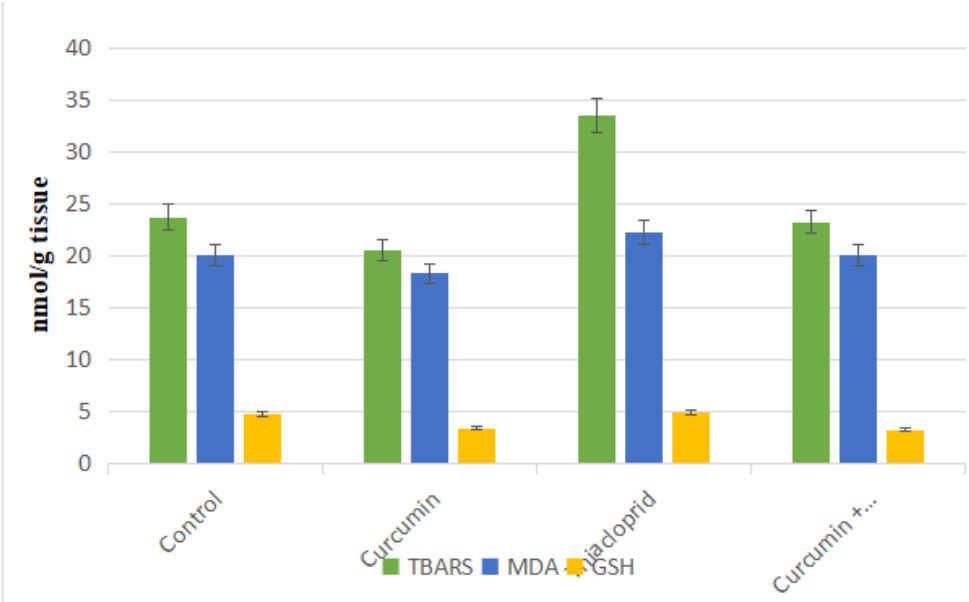

Figure 1: Effect of Curcumin and Thiacloprid and their combination (Curcumin + Thiacloprid) on the amount of reactive compounds with thiobarbituric acid, malondialdehyde (MDA), and reduced glutathione concentration in rat testes

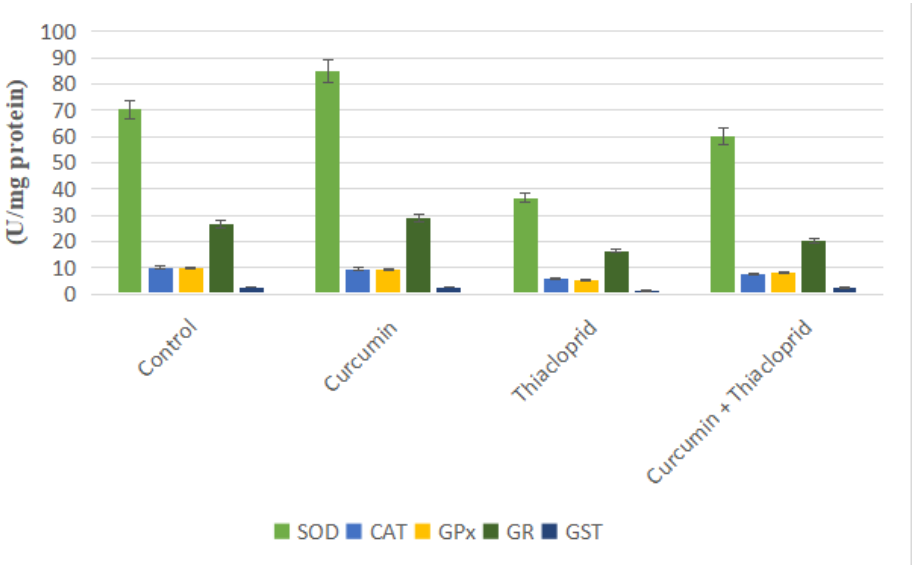

Figure 2: Effect of Curcumin, Thiacloprid and their combination (Curcumin + Thiacloprid) on the activities of antioxidant enzymes in rat testes

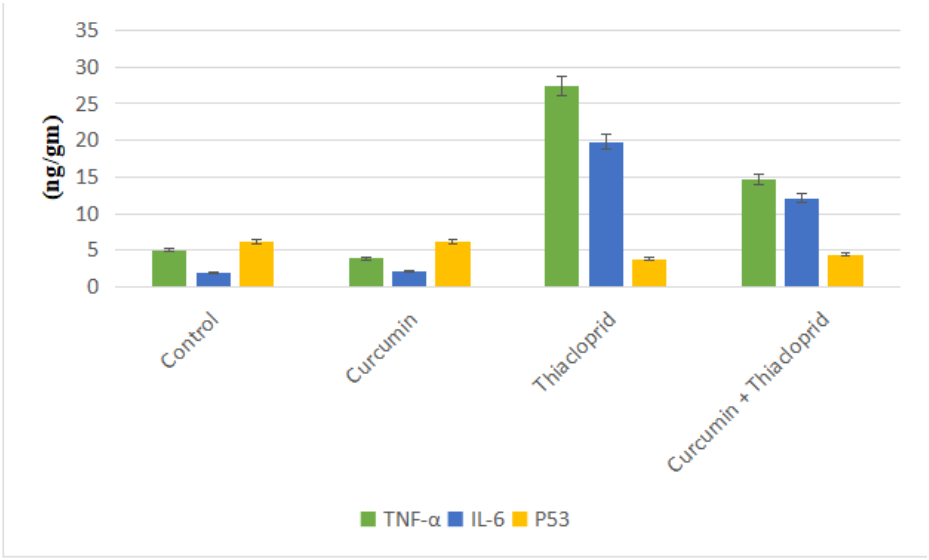

Figure 3: The impact of Curcumin, Thiacloprid, and their combination (Curcumin, Thiacloprid) on (TNFα), (IL-6), and (P53) in testes of male rats

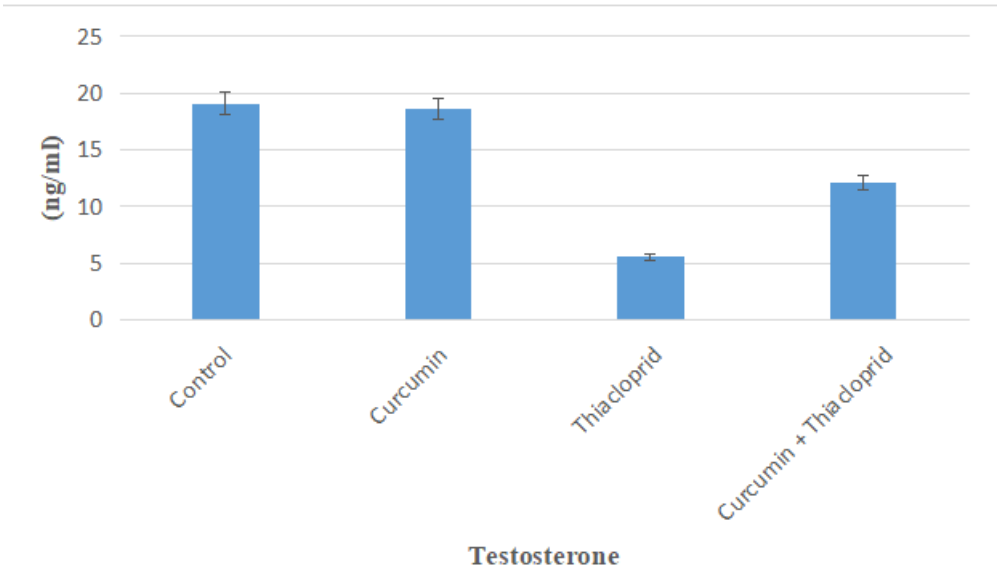

Figure 4: Effect of Curcumin, Thiacloprid, and their combination (Curcumin, Thiacloprid) on hormone levels in rat serum

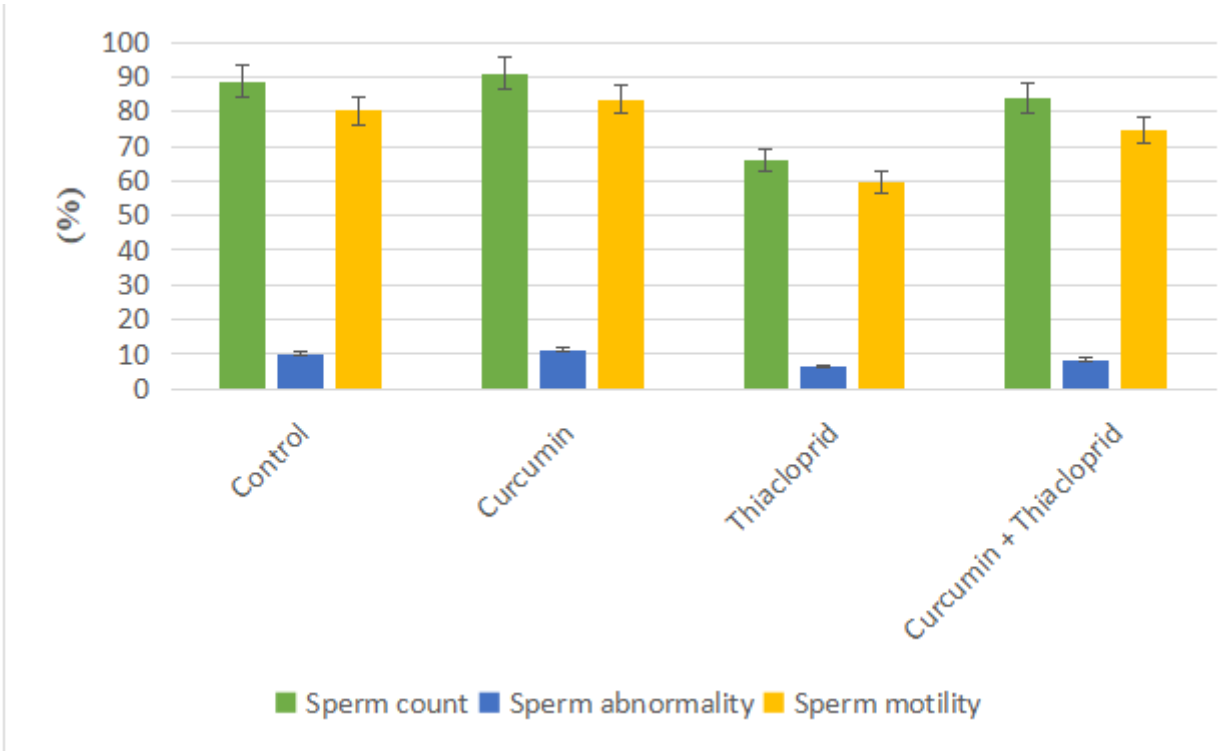

Figure 5: Effect of Curcumin, Thiacloprid, and their combination (Curcumin, Thiacloprid) on sperm quality

Conclusion

Our results revealed that Thiacloprid exposure induces very severe multi-organ toxicity in male rats and contributes to testicular oxidative damage, hormonal dysregulation, decreased sperm quality, histopathological changes of the testis, and liver dysfunction. In contrast, concurrent administration of Curcumin markedly diminishes these toxicological effects by acting as a potent cellular antioxidant and preserving tissue cytoarchitecture and homeostasis. Thus, Curcumin can be regarded as a potential natural bioprotective agent against

pesticide-induced oxidative stress and organ toxicity.

Conflict of interest: NIL

Funding: NIL

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