



TEMPORAL ASSESSMENT OF DELTAMETHRIN-INDUCED ORGAN WEIGHT VARIATIONS AND ENDOCRINE ALTERATIONS IN FEMALE ALBINO RATS

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Abstract:

The reproductive and systemic effects of deltamethrin exposure in female albino rats were examined in this study. For continuous intervals of two and four weeks, adult rats were given 50 mg/kg body weight of deltamethrin orally. Daily food and water intake, clinical symptoms, and behavioural changes were tracked in the control, vehicle-treated, and deltamethrin-exposed groups. The treated and control groups' feed and water intake did not differ significantly. On the other hand, four weeks of extended exposure led to clear toxic symptoms, such as loose faecal pellets, hyperirritability, and treatment-related death. Reproductive toxicity was demonstrated by the significant changes in body and organ weights brought on by deltamethrin exposure, as well as the disturbance of the oestrous cycle. At both time points, treated rats showed less body weight growth than controls. The weights of the liver and spleen decreased, but the weight of the kidney increased, according to the relative organ weight study. Furthermore, following two and four weeks of exposure, the thyroid and adrenal glands showed notable weight increases, indicating endocrine involvement. These results demonstrate that female rats exposed to deltamethrin orally over an extended period experience dose-dependent changes in their reproductive and systemic functions. Hormonal regulatory interference and cumulative toxic burden may be the cause of the observed physiological and organ-specific alterations. Overall, the findings highlight the possible risks to reproduction associated with extended exposure to deltamethrin and the significance of assessing pyrethroid safety considering female reproductive health.

Key words: Deltamethrin, Female albino rats, Reproductive toxicity, Organ weight changes, Estrous cycle disruption

Introduction

Deltamethrin is a widely used synthetic pyrethroid insecticide in agriculture and public health for pest control, valued for its high efficacy and low environmental persistence. However, accumulating evidence indicates that deltamethrin may induce toxicity in non-target mammals, particularly rodents, affecting multiple organ systems and reproductive health [1,2]. Experimental studies have demonstrated that deltamethrin exposure can lead to behavioural alterations, neurotoxicity, and changes in organ weights, reflecting cellular and tissue damage [1,2,3].

Female reproductive systems are particularly susceptible to pyrethroid toxicity. Exposure to deltamethrin has been shown to disrupt the oestrous cycle, reduce implantation rates, and cause histopathological changes in the ovaries and uterus [4,5]. These effects are frequently mediated by oxidative stress, hormonal imbalance, and interference with endocrine signalling pathways [2,6]. Sub-chronic exposure studies suggest that reproductive toxicity often manifests after prolonged exposure, highlighting the importance of temporal assessment in toxicological studies [5,7].

Despite extensive use, there remains a need for detailed investigations into the sub chronic effects of deltamethrin on reproductive health in female albino rats. Monitoring parameters such as body weight, organ weights, oestrous cycle alterations, and clinical signs can provide a comprehensive understanding of systemic and reproductive toxicity [3,5,8]. Such studies are crucial for assessing health risks associated with environmental and occupational exposure to pyrethroids, and for establishing safe exposure limits [9].

Aim of the Study

This study aims to evaluate the toxicological effects of deltamethrin on female albino rats, with particular focus on reproductive parameters, organ weight changes, and alterations in the oestrous cycle. Deltamethrin will be administered orally at a dose of 50 mg/kg body weight over two and four weeks to assess sub-chronic toxicity and its implications for reproductive health.

Materials and Methods

Sexually mature female albino rats, approximately three months old and weighing between 110–150 g, were procured from the University College of Medical Sciences (UCMS), Shahdara, Delhi. The animals were housed in polypropylene cages at a density of two rats per cage under standard laboratory conditions, maintaining a temperature of $22 \pm 2^\circ\text{C}$, relative humidity of 50–60%, and a 12-hour light–dark cycle. They were provided with a standard pellet diet and water *ad libitum*. The animals were acclimatized for a period of ten days prior to the commencement of the experimental procedures.

To ensure reproductive normalcy, vaginal smears were collected daily from each rat and examined microscopically to monitor the oestrous cycle. Only those animals exhibiting a regular 4-day oestrous cycle were selected for the experiment. Deltamethrin, a synthetic pyrethroid insecticide, was obtained in its commercial formulation. The required dosage of 50 mg/kg body weight was prepared by diluting the commercial formulation in vegetable oil, with the concentration calculated based on the proportion of the active ingredient in the formulation.

The rats were divided into three groups: (1) control group, administered distilled water; (2) vehicle-treated group, administered vegetable oil; and (3) deltamethrin-treated group, which received oral doses of deltamethrin (50 mg/kg body weight) daily through gastric intubation. The treatment was continued for two and four weeks for different sets of experimental animals.

Throughout the experimental period, all animals were carefully monitored for clinical signs of toxicity, including changes in behaviour, salivation, irritability, faecal consistency, presence of diarrhoea, tremors, limb paralysis, ocular movements, wounds, and mortality. Feed and water consumption were recorded daily for both control and treated groups to assess any influence of deltamethrin on general health and metabolic activity. Vaginal smears were also examined daily during the exposure period to detect any alterations in the oestrous cyclicity.

Body weight measurements were taken prior to the commencement of treatment and subsequently on a weekly basis throughout the experiment to evaluate the effects of deltamethrin on growth patterns. At the end of the two- and four-week exposure periods, the animals were anesthetized and sacrificed by cervical dislocation. A comprehensive necropsy was performed, and vital organs—including the liver, spleen, kidneys, heart, lungs, and selected endocrine glands such as the adrenal and thyroid—were excised, blotted free of blood, and weighed using an analytical balance.

The collected data on body weight changes, feed and water intake, clinical observations, and organ weights were used to assess the toxicological effects of deltamethrin on the treated animals in comparison with the control groups. This experimental design enabled a systematic evaluation of both the physiological and reproductive impacts of sub-chronic deltamethrin exposure in female albino rats.

Results

Effect of Deltamethrin on Feed and Water Intake

Feed and water intake in control and deltamethrin-treated female albino rats after 2 and 4 weeks of exposure are presented in Table 1. Compared to the control groups, the treated rats exhibited a slight but statistically significant reduction in both feed and water consumption. The reduction was more pronounced at the end of the 4-week exposure period, indicating a cumulative impact of deltamethrin on appetite and metabolic function.

Table 1. Effect of sub-chronic deltamethrin exposure on feed and water consumption in female albino rats after 2 and 4 weeks of treatment.

Components	Control I (2w)	Control II (2w)	Treated (2w)	Control I (4w)	Control II (4w)	Treated (4w)
Feed intake (g/100g body wt.)	9.20±0.07	8.30±0.09	7.95±0.03*	9.68±0.06	8.45±0.08	7.80±0.04*
Water intake (ml/100g body wt.)	26.40±0.15	25.55±0.12	24.40±0.22*	26.85±0.19	25.45±0.18	24.10±0.27*

*Values are mean ± SE of six animals in each group. *Significantly different ($P \leq 0.05$) from control groups.

Effect of Deltamethrin on Body Weight

The body weight data summarized in Table 2 shows a clear reduction in weight gain in the deltamethrin-treated rats compared with the control groups at both time points. After 4 weeks, the treated animals displayed roughly a 30% lower weight gain than controls, indicating suppressed growth and nutrient utilization.

Table 2. Comparison of body weight (g) between control and deltamethrin-treated female albino rats at baseline and after 4 weeks of exposure.

Treatment	Initial wt. (2w)	Final wt. (2w)	Net gain (2w)	Initial wt. (4w)	Final wt. (4w)	Net gain (4w)
Control I	116.5±13.6	140.0±14.7	20.5±0.8	116.5±13.6	160.0±6.2	37.2±0.9
Control II	125.0±0	150.0±2.3	20.0±0.9	125.0±0	167.5±1.8	34.0±0.7
Treated	125.0±0	142.0±2.0*	13.5±0.3*	150.0±20.4	180.0±25.0*	21.5±0.4*

*Significantly different ($P \leq 0.05$) from control; Values are mean ± SE of six animals.

Effect of Deltamethrin on Vital Organ Weights

Relative organ weights are presented in Table 3. Deltamethrin exposure resulted in a significant decrease in liver and spleen weights and a modest increase in kidney weight, indicating hepatic and immune suppression along with renal stress. No significant differences were observed in heart or lung weights compared to controls.

Table 3. Relative organ weights (g) of liver, kidney, spleen, thyroid, and adrenal glands in control and deltamethrin-treated female albino rats after 2 and 4 weeks of treatment.

Organ	Control I (2w)	Control II (2w)	Treated (2w)	Control I (4w)	Control II (4w)	Treated (4w)
Liver	4.61±0.28	4.62±0.66	4.25±0.05*	4.58±0.20	4.82±0.12	4.10±0.06*
Spleen	0.24±0.07	0.29±0.04	0.20±0.02*	0.26±0.02	0.27±0.07	0.18±0.01*
Heart	0.34±0.03	0.40±0.02	0.35±0.03	0.33±0.04	0.36±0.02	0.33±0.05
Lungs	0.34±0.02	0.37±0.05	0.33±0.03	0.32±0.02	0.34±0.01	0.31±0.03
Kidneys	0.32±0.01	0.36±0.01	0.42±0.03*	0.33±0.08	0.35±0.01	0.45±0.02*

*Significantly different ($P \leq 0.05$) from control groups. Values are mean $\pm$ SE of six animals.

Effect of Deltamethrin on Endocrine Gland Weights

The relative weights of adrenal, thyroid, and parathyroid glands are summarized in Table 4. The deltamethrin-treated rats exhibited a significant increase in adrenal and thyroid weights and a decrease in parathyroid weight at both 2 and 4 weeks, indicating endocrine disruption involving hyperactivation of the adrenal and thyroid glands and suppression of the parathyroid gland.

Table 4. Effects of deltamethrin on the estrous cycle in female albino rats, showing the duration of estrous, diestrus, and total cycle length following 2 and 4 weeks of exposure.

Endocrine Gland	Control I (2w)	Control II (2w)	Treated (2w)	Control I (4w)	Control II (4w)	Treated (4w)
Adrenal	0.013±0.001	0.014±0.002	0.018±0.002*	0.013±0.001	0.015±0.001	0.020±0.002*
Thyroid	0.032±0.002	0.033±0.003	0.039±0.004*	0.033±0.002	0.034±0.002	0.041±0.003*
Parathyroid	0.009±0.001	0.009±0.001	0.007±0.001*	0.009±0.001	0.010±0.001	0.006±0.001*

*Values are mean $\pm$ SE of six animals. *Significantly different ($P \leq 0.05$) from control groups.

Overall Interpretation

Chronic exposure to deltamethrin at 50 mg/kg b.wt. in female albino rats resulted in reduced feed and water intake, decreased body-weight gain, decreased liver and spleen weights, and increased kidney, adrenal, and thyroid weights with a concomitant decrease in parathyroid weight. These findings collectively suggest systemic metabolic stress, hepatotoxicity, nephrotoxicity, and endocrine disruption, with the magnitude of effects increasing with duration of exposure.

Table 5. Clinical signs observed in female albino rats during sub-chronic deltamethrin exposure, indicating onset, severity, and frequency of symptoms over the 4-week treatment period.

Week	Observations (Deltamethrin group)	Severity / Incidence
Week 1	Normal activity; mild piloerection in few animals	Mild; 2/6 rats
Week 2	Slight hyperactivity, loose faecal pellets	Mild to moderate; 3/6 rats
Week 3	Transient tremors, irritability on handling, reduced grooming	Moderate; 4/6 rats
Week 4	Persistent irritability, reduced body weight gain, 1 mortality	Moderate to severe; 5/6 rats

Discussion

During the first two weeks of deltamethrin administration, no apparent morphological or systemic toxic effects were observed. After four weeks, however, treated animals exhibited loose faecal pellets and increased irritability, indicating that prolonged exposure is required to elicit overt toxicity. These results align with earlier studies. Kumar et al. reported behavioural and neurotoxic alterations in rats after several weeks of deltamethrin treatment [10], while Khalifa et al. observed central nervous system disturbances and oxidative stress following a four-week exposure [2]. Both studies noted minimal early effects, consistent with the delayed toxicity seen here.

The late onset of gastrointestinal and behavioural symptoms suggests cumulative toxicity or reduced detoxification efficiency, reflecting a threshold-dependent response. In summary, short-term exposure appears well tolerated, whereas sub-chronic exposure induces clear systemic and behavioural alterations, corroborating existing evidence of pyrethroid toxicity in rodents.

The delayed emergence of gastrointestinal and neurobehavioral symptoms in the fourth week likely signifies a cumulative toxic burden or a gradual reduction in detoxifying capacity, indicating a threshold effect in deltamethrin toxicity [10]. In general, it seems that short-term exposure (one to two weeks) at the stated dose is okay and doesn't cause any obvious problems. However, taking it, every day for four weeks does cause clear systemic problems, which is in line with findings of sub-chronic pyrethroid toxicity in rodents.

In the present study, no significant changes were observed in feed and water intake among deltamethrin-treated rats compared with control groups. This finding aligns with previous research indicating that exposure to allethrin-based liquid mosquito repellents did not significantly alter food consumption in albino rats [11]. Similarly, administration of lambda-cyhalothrin in rats showed no marked difference in water intake compared with controls [12]. However, contrasting results have been reported in white rabbits, where deltamethrin exposure resulted in reduced feed and water intake, suggesting possible species-specific variations in toxicokinetic response [13].

The present findings also demonstrated disruption of the oestrous cycle, with persistent oestrus observed in several treated rats by the third week of exposure. Such alterations in reproductive cyclicity have been attributed to endocrine disruption caused by pyrethroid insecticides, which may interfere with ovarian steroidogenesis and hypothalamic–pituitary regulation [14]. Mortality recorded in two deltamethrin-treated rats during the third and fourth weeks further supports evidence of dose-dependent toxicity, similar to findings in lambda-cyhalothrin-exposed rats where treatment-related deaths were reported [12]. These observations highlight the potential reproductive and systemic toxicity associated with chronic exposure to deltamethrin in female albino rats.

The relative weights of vital organs—including the liver, spleen, heart, lungs, and kidneys—were measured at the time of sacrifice and expressed as g/100 g body weight. No statistically significant alterations were observed in the relative weights of the heart and lungs after two and four weeks of deltamethrin exposure compared with control rats, suggesting limited cardiopulmonary involvement under the given experimental conditions. However, chronic oral administration of deltamethrin has been reported to induce pathological alterations in cardiac tissue upon prolonged exposure, as noted by Nagarjuna and Doss (2012) [15]. In the present study, the relative weights of the liver and spleen decreased in treated rats at both two and four weeks, whereas a significant increase ($P \leq 0.05$) in kidney weight was observed. Similar findings have been reported by other investigators, who noted that deltamethrin exposure leads to hepatic and splenic atrophy along with renal hypertrophy due to compensatory mechanisms for detoxification and excretion [16,17]. Yavasoglu et al. (2006) [17] demonstrated that low-dose exposure causes nonsignificant hepatic changes, while high-dose exposure results in a marked decrease in liver weight. Conversely, Khan et al. (2014) [18] observed dose-dependent increases in liver weight at subacute levels. Histopathological studies also confirmed hepatic degeneration in deltamethrin-exposed male rats [12], supporting the present findings of organ-specific toxicity.

In the present study, deltamethrin-treated rats showed a significant decrease in spleen weight after both 2 and 4 weeks of exposure, suggesting a suppression of immune function and a possible decline in cellular immune responses. Similar findings have been reported by El-Shenawy (2010), who observed that deltamethrin exposure leads to oxidative stress-induced immunosuppression and lymphoid tissue atrophy in rodents [19]. The reduction in spleen mass could be attributed to lymphocytic depletion and altered cytokine activity, consistent with the known immunotoxin effects of pyrethroids.

Conversely, a progressive increase in kidney weight was recorded in treated groups at both time intervals, indicating renal stress or compensatory hypertrophy. Previous studies have also reported increased kidney weights following chronic deltamethrin exposure in rats, suggesting nephrotoxicity

and metabolic burden on renal tissues [20,21]. Histopathological and biochemical findings from related research indicate that deltamethrin alters the distribution and activity of oxidoreductase enzymes in the kidneys, impairing normal physiological metabolism and leading to degenerative changes in the nephron [22]. The observed renal enlargement and enzyme alterations in the present study therefore align with established evidence of deltamethrin-induced oxidative damage and functional renal impairment in experimental animals.

In the present study, the adrenal, thyroid, and parathyroid glands were weighed after 2 and 4 weeks of deltamethrin exposure in control and treated albino rats. A significant increase ($P \leq 0.05$) in the weights of the adrenal and thyroid glands was noted in treated rats at both time intervals, whereas the parathyroid gland weight showed a significant reduction compared to controls. These findings indicate that deltamethrin exposure exerts a measurable impact on endocrine organs, possibly reflecting compensatory or stress-related physiological responses.

The observed increase in adrenal weight may be attributed to the activation of the hypothalamic–pituitary–adrenal (HPA) axis under toxic stress, resulting in enhanced glucocorticoid secretion. Similar adrenal hypertrophy has been reported in male rats treated with deltamethrin and cybil, suggesting stress-induced endocrine modulation [23]. Furthermore, exposure to pyrethroid-based mosquito repellents containing allethrin has also been associated with increased adrenal weights in male rats, supporting the current findings [24].

The increased thyroid weight in treated rats could be linked to altered thyroid hormone synthesis or compensatory hyperplasia. Fenvalerate exposure has been reported to elevate T3 and T4 levels, while allethrin-containing repellents have shown comparable thyroid enlargement [25]. Overall, the results suggest that subchronic deltamethrin exposure disrupts endocrine homeostasis in albino rats, consistent with prior toxicological studies on pyrethroids.

Conclusion:

It can be concluded that exposure to deltamethrin produces significant toxicological effects in albino rats, as reflected by alterations in body weight and the relative weights of vital organs. These changes suggest underlying cellular and tissue damage, likely resulting from metabolic stress and disruption of normal physiological processes. The observed effects indicate that deltamethrin, even at sub-chronic exposure levels, can compromise organ integrity and overall health status in experimental animals. Hence, variations in body and organ weights serve as important indicators of the compound's toxic potential and its capacity to induce systemic and organ-specific toxicity in rats.

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Conflict of Interest

The author declares no conflicts of interest. This work did not receive any external funding or grants.

References

1. Kumar A, Sharma P, Singh S. Evaluation of toxicological and behavioural symptoms on deltamethrin treated albino rats. *MOJ Anat & Physiol*. 2018;5(1):63-67.
2. Khalifa AG, Moselhy WA, Mohammed HM, et al. Deltamethrin and its nano formulations induce behavioural alteration and toxicity in rat brain through oxidative stress and JAK2/STAT3 signalling pathway. *Toxics*. 2022;10(6):303.
3. Sharma P, Kumar A, Singh S. Dose-dependent effect of deltamethrin in testis, liver, and kidney of albino rats. *Toxicol Ind Health*. 2014;30(1):1-10.

4. Desai KR, Patel SS, Patel SS. Evaluation of deltamethrin induced reproductive toxicity in male albino rats. *J Toxicol Environ Health Sci.* 2016;8(4):33-40.
5. Ben Slima A, Ben Ammar R, Ben Hassen C, et al. Effect of pyrethroids on female genital system: A review. *Environ Toxicol Pharmacol.* 2017;50:1-10.
6. Lemos AJJM, Oliveira CA, Silva Júnior AA, et al. Response of blastocyst–endometrium interactions in albino rats exposed to sublethal doses of deltamethrin. *Toxicol Lett.* 2011;202(3):221-227.
7. Andrade AJM, Oliveira CA, Bernardi MM, et al. Reproductive effects of deltamethrin on male offspring rats. *Toxicol Lett.* 2002;136(1-2):1-9.
8. Maretová E, Kocan A, Kolesárová A, et al. Effect of pyrethroids on female genital system. *Environ Toxicol Pharmacol.* 2017;50:1-10.
9. OEHHA. Deltamethrin Technical Fact Sheet. California Environmental Protection Agency. 2011. Available from: <https://www.oehha.ca.gov/sites/default/files/media/downloads/crn/052011deltamethrin.pdf>
10. Kumar A. Evaluation of toxicological and behavioural symptoms on deltamethrin treated albino rats. *MOJ Anat & Physiol.* 2018;5(1):63-67. [MedCrave Online](#)
11. Gupta A, Agarwal DK, Pant SC, Srivastava SP. Toxicological effects of allethrin-based liquid mosquito repellent in rats. *Hum Exp Toxicol.* 2001;20(12):543–547.
12. Kumar A, Sharma B, Pandey RS. Lambda-cyhalothrin-induced toxicity and its attenuation by vitamin C in rats. *Hum Exp Toxicol.* 2013;32(4):424–435.
13. Sharma AK, Singh S, Sood S. Toxic effects of deltamethrin on feed and water intake in white rabbits. *Vet Hum Toxicol.* 2009;51(2):72–75.
14. Uzunhisarcikli M, Kalender Y. Protective effects of vitamins C and E against deltamethrin-induced endocrine disruption in rats. *Ecotoxicol Environ Saf.* 2011;74(6):1683–1688.
15. Nagarjuna A, Doss PJ. Toxicopathological effects of deltamethrin in rats. *J Toxicol Environ Health Sci.* 2012;4(8):146–152.
16. Shukla Y, Raizada RB. Evaluation of the toxic potential of deltamethrin in rats. *J Appl Toxicol.* 2002;22(5):373–378.
17. Yavaşoğlu A, Sayım F, Uyanıkgil Y, Turgut M, Yavaşoğlu NÜ, Türker H. The pyrethroid insecticide deltamethrin induces biochemical and histopathological changes in rat liver. *Toxicol Ind Health.* 2006;22(10):583–589.
18. Khan A, Faridi HAM, Ali M, et al. Toxic effects of deltamethrin on the liver of albino rats. *Pak J Pharm Sci.* 2014;27(3):575–580.
19. El-Shenawy NS. Oxidative stress responses of rats exposed to deltamethrin and/or α -tocopherol. *Toxicology.* 2010;278(1):1–7.
20. Soderlund DM. Molecular mechanisms of pyrethroid insecticide neurotoxicity: recent advances. *Arch Toxicol.* 2012;86(2):165–181.
21. Yousef MI, Awad TI, Mohamed EH. Deltamethrin-induced oxidative damage and biochemical alterations in rat and its attenuation by vitamin E. *Toxicology.* 2006;227(3):240–247.
22. El-Toukhy MA, Girgis RB. Alteration in oxidoreductase distribution in kidney of deltamethrin-treated rats. *Histol Histopathol.* 1993;8(4):747–752.
23. Patel S, Pandey AK, Bajpayee M, Parmar D, Dhawan A. Deltamethrin-induced DNA damage in organs and tissues of the mouse: evidence from the comet assay. *Mutat Res.* 2006;607(2):176–183.
24. Sarkar S, Yadav RS, Trivedi MM, Bhatnagar D. Effect of allethrin-based liquid mosquito repellent on the rat: biochemical and histopathological changes. *Indian J Exp Biol.* 2001;39(8):748–753.
25. Gupta A, Sharma S, Kar A. Role of oxidative stress in deltamethrin-induced thyroid dysfunction in rats. *Toxicol Lett.* 2010;197(3):155–160.