REDVET - Revista electrónica de Veterinaria - ISSN 1695-7504
Vol 25, No.1 (2024)
http://www.veterinaria.org
Article Received: 04- 04- 2024, Revised:15-05-2024, Accepted: 25- 06- 2024

Toxicological And Behavioural Effects Of Deltamethrin In Albino Rats

Anil Kumar[1*]

[1*]Assistant Professor, Department of Anatomy and Neurobiology, College of Medicine and Health Science (CoMHS), National University of Science and Technology (NUST), Sohar, Al Tareef. 391, P. O. Box: 321, Sultanate of Oman, Sultanate of Oman, +968-95393982, E-mail: anilkumar@nu.edu.om

***Corresponding author**: Dr. Anil Kumar
*Assistant Professor, Department of Anatomy and Neurobiology, College of Medicine and Health Science (CoMHS), National University of Science and Technology (NUST), Sohar, Al Tareef. 391, P. O. Box: 321, Sultanate of Oman, Sultanate of Oman, +968-95393982, E-mail: anilkumar@nu.edu.om

ABSTRACT:
Pesticides, especially deltamethrin—a Type-II synthetic pyrethroid widely utilized in agriculture and residential settings—are among the most concerning hazardous agents intentionally introduced into our environment, prompting serious apprehensions regarding their possible detrimental impacts on human health. This study examined the harmful effects of deltamethrin on behaviour and motor coordination in adult Wistar albino rats (250-270 gm), which were administered the substance intraperitoneally at a dosage of 0.5 mg/kg body weight for one month. Behavioural and toxicological indicators, encompassing body weight, rectal temperature, open field behaviour, social contact, landing foot splay, and neuromuscular function (forelimb and hindlimb grip assessments), were monitored. Experimental rats displayed mild to moderate symptoms, including reduced food consumption, salivation, motor incoordination, looping, and weaving. Rats treated with deltamethrin exhibited a statistically significant reduction in body weight, diminished locomotor and rearing frequency, prolonged immobility duration, decreased social interaction, enlarged foot angles, and delayed reflex onset, indicating notable variability in toxicity and motor coordination.

Keywords: Deltamethrin, Behavioural changes, Pyrethroid, Pesticides, Toxicity

Introduction:

Deltamethrin, a synthetic pyrethroid of type II that possesses insecticidal properties, is a commonly employed ectoparasiticide in public health and agricultural protection programs [1]. Deltamethrin is notably neurotoxic, as evidenced by extensive animal studies. It disrupts the sodium channel gating mechanism, which is essential for the generation and conduction of nerve impulses [2].

Recent studies conducted on mice, rats, rabbits, and guinea pigs have reported toxicity through dermal, oral, and inhalational routes. This toxicity is characterized by excessive salivation, impaired limb function, ataxia, loss of righting reflex, lethality, paraesthesia, choreoathetosis, tremors, and at times, paralysis and convulsions [3-6]. Deltamethrin decreased the average weight of live fetuses when administered during major organogenesis [7]. During the manufacturing and handling process, occupational hazards may include transient cutaneous and mucous membrane irritation, itching, dizziness, anomalous facial sensations, and allergic reactions [8].

The indiscriminate and injudicious use of pesticides poses a threat to human health, as the entry of pesticide residues into the food chain and the exceeding of maximal limits can result in adverse effects [9]. Given its extensive agricultural application and associated harmful effects, this study aims to evaluate the behavioural, morphological, and histopathological effects of repeated oral administration of deltamethrin in albino rats.

MATERIALS AND METHODS

Albino rats

The albino rats, both male and female, were obtained from the animal hospital of the University College of Medical Sciences (UCMS) and Guru Teg Bhadur (G.T.B) Hospital in Delhi. They were approximately 90 days old and weighed 250-270 gm. Free access to food and water was provided to the animals, who were confined in propylene cages measuring 32 X 40 X 18 cm and maintained at a controlled temperature of 22-24°C with a 12-hour light/dark cycle. The investigations were conducted in accordance with the guidelines of the Institutional Animal Ethical Committee (IAEC) of UCMS and G.T.B. Hospital, Delhi, as well as national regulations. Before the treatment commenced, the rodents were subjected to acclimatization for a period of one week.

Research methodology

Normal feed and water were administered to all animals' ad libitum throughout the duration of the study. The animals were divided into two groups: control (Group I) and experimental (Group II), each of which contained ten animals. Deltamethrin was administered intraperitoneally to adult Wistar albino rats at a dose of 0.5 mg/kg body weight for a month. The test concentrations were achieved by dilutions made with peanut oil. Based on the percentage of active

constituents in commercial formulations, the test concentrations were determined. Control rodents were administered an equivalent quantity of peanut oil via intraperitoneal injection, devoid of any pesticides. Every 10 days, the body weights of all animals were recorded.

Toxicological manifestations
Both control and experimental rats were closely monitored throughout the study for any behavioural changes and toxic symptoms. Detailed records were kept of the nature, severity, and timing of these symptoms.

Behavioural modifications
Physical parameters of general behavior were assessed daily. Traditional analyses of drug induced behavior start off with a ready list of arbitrarily defined behavioural acts (such as sniffing, head down, nose-poking, or rearing). The objective of those analyses is to discover the frequency, duration, and sequence of the acts during the drugs action [10-11].

Open field investigations
The open field apparatus, based on Broadhurst's design, features a circular arena with a diameter of 96 cm, surrounded by a 25 cm high white wall and partitioned into 25 sections by black stripes [12]. A 40-watt white lamp, situated 72 cm above the floor, delivered continuous illumination during the studies. Handheld counters and stopwatches were employed to quantify locomotion (number of floor units traversed), rearing frequency (number of instances an animal stood on its hind limbs), and immobility (total duration without spontaneous movements). Rats were individually positioned in the centre of the open-field arena, and their activity was monitored for three minutes. To mitigate circadian effects on behavior, control and experimental animals were switched. The device was sanitized with a 5% alcohol/water solution prior to each session to remove olfactory biases. Control and experimental rats were evaluated between 8:00 AM and 12:00 PM, following a minimum acclimatization period of 90 minutes in environments mirroring the test circumstances.

Test of social interaction
The social interaction assessment occurred in the open field equipment. Two days before, each rat participated in a 10-minute acclimatization session within the test arena. On the subsequent day, rats were matched by weight (with a maximum difference of 10 grams) and monitored for social contact during a duration of 10 minutes. Two hours following the administration of deltamethrin to experimental rats or peanut oil to control rats, the paired rats were positioned in the center of the test arena to evaluate social interaction. The cumulative duration (in seconds) allocated to active social activities, including sniffing, following, grooming, kicking, boxing, biting, and manoeuvring under or over the partner, was documented for 7.5 minutes. The device was sanitized with a 5% ethanol solution before to each test. Control and experimental rat pairs were cycled, with tests administered between 8:00 AM and 12:00 PM.

Rectal temperature measurement
A metallic sensor that was lubricated with Vaseline was employed to register the rectal temperature using a thermometer (Rat Rectal Temperature Probe). During the peak effects of deltamethrin on open field behaviors, measurements were obtained before and 120 minutes after the respective treatments. The thermometer was disinfected with a 5% ethanol solution prior to each use. Measurements were conducted between 8:00 AM and 12:00 PM to prevent interference from circadian variation, and control and experimental rodents were alternated.

Foot splay upon landing
The footprint test is utilized to evaluate gait, indicating peripheral nerve injury (neuropathy). The animal is dropped from a height, and the distance between its hind feet upon landing is measured. Splay values are quantified in millimetres (mm). Foot splay appeared independent of body weight, as the measurements did not rise over time despite the rats' growing weights [13-14].

Grip tests for neuromuscular function in the forelimb and hindlimb
The animal's capability to suspend its forelimb, the period of suspension, and its behavior during hanging were examined. Muscle strength was quantified using a grip strength meter (Columbus Instruments International Company, USA), which evaluates neuromuscular function by measuring the maximum force exerted by an animal when gripping specifically constructed pull bar assemblies. The meter employs precision force gauges to digitally record and show the maximum force [15].

Statistical evaluations
Quantitative observations for all rats in both groups were documented, organized, and analysed utilizing an independent sample t-test. Body weight data for the experimental and control rats were compiled and evaluated utilizing Tukey's test. Statistical analysis was performed using SPSS 20.0 software, with a significance threshold of $P < 0.001$.

REDVET - Revista electrónica de Veterinaria - ISSN 1695-7504
Vol 25, No.1 (2024)
http://www.veterinaria.org
Article Received: 04- 04- 2024, Revised:15-05-2024, Accepted: 25- 06- 2024

Results

All control rodents maintained their normal, healthy, and active state throughout the treatment period. Conversely, rodents that were administered deltamethrin exhibited diminished activity and a lack of strength. All of the animals included in the investigation survived the thirty-day period. Compared to control rodents, rats treated with deltamethrin exhibited a reduction in body weight gain. The control group rodents' mean body weight was 178.50±7.47g prior to the experiment and 182.50±8.57g on the final day (Table 1). The mean body weight of the experimental group was 181.50±6.25g at the outset and 166.00±8.09g on the final day. Physical parameters were adversely impacted in deltamethrin-treated rats, and control rats were more active than treated rats. The behaviours that were observed were as follows: sniffing, keeping the head down, jumping, circling, rotating, looping (somersaulting from the cage top), and weaving (pacing to and fro over the same location, with frequent rears when turning).

Table 1: Body weight (gm) comparison between experimental and control rats

Experiment	Group	Mean	SD	P-value (One way ANOVA)	Significance (Tukey's test at 5% level)
Before the experiment	Control	178.5	7.47	>0.001*	The groups were not statistically significant
	Experimental	181.5	6.25		
Last day of experiment	Control	182	8.57	<0.001*	Experimental group was statistically significantly different control groups
	Experimental	166	8.09		

**p value≤0.001 means data are statistically significant*
μ, mean; S.D, standard deviation; gm, grams

The rats exhibited hyperactivity, which was characterized by sneezing, shuddering, groaning, and excessive salivation, for approximately 30 minutes following the initial dose of deltamethrin. The rats exhibited symptoms such as loose faeces and occasional vomiting, as well as a loss of appetite and a lethargic appearance in the days that followed.

The experimental rodents demonstrated a decreased locomotor frequency during the 120-minute session in comparison to the control group, as indicated by the open field test. The deltamethrin-treated rodents also exhibited a lower rearing frequency, which was indicative of a decrease in locomotor activity. It is important to note that the duration of immobility increased significantly because of the reduced locomotor and rearing frequencies. The control and treated groups both spent comparable amounts of time in the centre, suggesting that they were cognizant of the chamber's boundaries and could observe the surrounding area.

Social interaction was markedly diminished in the experimental group relative to the control group (Student's t-test, $p < 0.001$). Deltamethrin administration did not influence rectal temperature across any of the groups, with temperatures varying from 32.0 to 36.2°C.

The Landing foot splay analysis test revealed that the experimental rat exhibited a greater foot angle compared to the control rat. Additionally, the experimental rats displayed increased agitation and moved more slowly along the walkway. The animal's capacity to suspend its forelimb, the duration of suspension, and its behavior during this period were examined. Marked decreases in forelimb and hindlimb suspension duration (seconds) were seen in male experimental rats relative to the control group. Furthermore, a postponed emergence of specific reflexes was noted in rats subjected to deltamethrin exposure.

Discussion

Deltamethrin administered at a dosage of 0.5 mg/kg body weight induced different degrees of mild to moderate toxic symptoms and behavioural alterations in albino rats. The rats exhibited hyperactivity immediately following the initial dose of deltamethrin. In the following days, they exhibited lethargy, diminished appetite, loose stools, and intermittent vomiting. Deltamethrin is recognized as neurotoxic, affecting axons in both the peripheral and central nervous systems by interaction with sodium channels. Chesterman et al. noted vomiting, liquid faeces, uncoordinated movements, tremors, and abnormal reflexes in dogs following the oral administration of deltamethrin dissolved in polyethylene glycol within gelatine capsules, attributing these manifestations to dose-dependent dysfunction of the autonomic nervous system [16]. In rats, toxicity indications following deltamethrin injection included salivation and choreoathetosis, a writhing form of toxicity referred to as choreoathetosis/salivation syndrome toxicity [17-20].

Clark and Brooks reported that Type-II syndrome toxicity, notably choreoathetotic writhing, is linked to abnormally elevated plasma noradrenaline and adrenaline concentrations [21]. Parkin, Carbaral, Bateman, Narahashi, and Soderlund observed comparable results in rodents following deltamethrin administration [22-23, 4-6]. Wu et al. administered deltamethrin in corn oil intraperitoneally to male Sprague Dawley rats and proposed that the degeneration and apoptotic cellular death in the rat brain are induced by deltamethrin's DNA fragmentation [24]. This underscores the role of apoptosis in deltamethrin's neurotoxicity. Sayim and Mokhtar attributed these symptoms to cerebral stroke signs and symptoms, which were the result of reduced brain acetylcholinesterase (AChE) activity and brain tissue ischemia [25-26]. Manna attributed these effects to the considerably low concentrations of gamma-aminobutyric acid (GABA) in brain tissue [27].

Chen proposed that these toxic effects are the result of mitochondria-mediated apoptosis of nerve cells in the rat brain [28].

Deltamethrin-treated rats demonstrated a statistically significant reduction in body weight, presumably attributable to hyperactivity, excessive salivation, anorexia, diarrhoea, and intermittent vomiting. This weight loss corresponds with Kavlock et al., who documented a 20% decrease in the body weight of female rats [29]. Madsen et al. and Elbetieha et al. similarly noted decreases in male Fisher and Sprague-Dawley rats following oral injection of deltamethrin [30-31]. Patro et al. observed similar outcomes when deltamethrin was delivered intraperitoneally in propylene glycol to Wistar albino rats [32]. Our findings, however, diverge from those of Sayim et al. and Varshneya et al., who reported no significant alterations in body weight in mice following the administration of the Type-II pyrethroid, cypermethrin [25, 33]. The disparities may result from varying delivery routes and dosages employed in the research.

Locomotion frequency, which is quantified by the distance travelled, immobility or resting time, and rearing in the open field, is a measure of both emotionality and arousal. A reduction in arousal or an increase in emotionality is typically indicated by a decrease or absence of movement within the apparatus [34-37]. Reduced locomotor activity was observed in the current study, but motor coordination was not affected. In this context, the primary response to increased emotionality in the open field is frozen behavior, which results in increased immobility and decreased locomotion frequency. Consequently, the observed decrease in locomotion frequency and increase in immobility may be a consequence of the elevated levels of emotionality that pyrethroid exposure induces. Bhattacharya and Mitra suggested that the reduced open field activity observed after deltamethrin administration may be as a result of elevated anxiety levels rather than general motor activities [38-39]. As a result, protracted immobility was observed in conjunction with decreased locomotor and rearing frequencies. Nevertheless, the hypothesis that deltamethrin influences affective parameters rather than motor function is substantiated by the absence of an impact on motor coordination.

The social interaction test indicated that deltamethrin reduced the duration of social contacts, implying it elicits anxiety-like behavior. The conclusion is based on findings from tests involving anxiolytic and anxiogenic drugs: a decrease in social contact time signifies an anxiogenic impact, whereas an increase implies an anxiolytic effect [40-42].

It is essential to evaluate the toxic effects of pyrethroids by examining the thermoregulation of rodents. Hyperthermia is frequently the result of type-I pyrethroid exposure, which is likely attributed to an increase in muscular activity [43]. Conversely, hypothermia may result from Type-II pyrethroids, such as deltamethrin. The absence of a change in rectal temperature suggests that the current dosages do not induce severe toxicity [44].

The Landing Foot Splay Analysis Test assessed coordination. The experimental rat displayed a greater foot angle than the control rat, indicating that the control rat experienced less stress and ambulated normally. Conversely, the experimental rats exhibited heightened agitation and ambulated slowly along the walkway, resulting in atypical foot placement and an elevated foot angle, signifying diminished and unnatural locomotion [45].

The experimental rats required a greater number of days to develop a sharp hanging grip, whereas the control rats took longer to hold and developed a sharp grip on alternate days. Furthermore, the negative geotaxis and palmer grasp of control rats were more acute than those of treated rats [46]. Significant deficits in motor abilities, coordination, and overall activity were observed in rodents treated with deltamethrin. Deltamethrin exposure can result in long-term cognitive and motor consequences, as evidenced by footprint analysis and open field tests.

Conclusion:

Integrated methodologies that integrate neurophysiological and behavioural assays with biochemical markers may offer a valuable methodology for evaluating human neurotoxicity, despite some controversies in the reported findings and existing literature. By establishing pathways for prevention and treatment, the comprehension of the behavioural outcomes and cellular mechanisms of deltamethrin exposure will contribute to the reduction of morbidity and dysfunction.

Acknowledgement:

I wish to convey my deep appreciation to Dr. Mahindra Nagar, Dr. Kamlesh Khatri, and Dr. Veena Bharihoke for their priceless counsel, unwavering motivation, and illuminating support during my work. I express my gratitude to Mr. Laxman Singh for his assistance and technical support.

Conflict of interest

The author has no conflicts of interest to disclose. No grants are available for funding.

References

1. McGregor DB. Pesticide residues in food: deltamethrin. International agency for research on cancer: Lyon, France; 2000.
2. Dorman DC, Beasley VR. Neurotoxicology of pyrethrin and pyrethroid insecticides. *Vet Hum Toxicol*. 1991;33(3):238–243.

3. Clark JM. Effects and mechanism of action of pyrethrin and pyrethroid insecticides. In: Chang LW, Dyer RS, editors. *Handbook of neurotoxicol.* New York: Marcel Dekker; 1995:511–546.
4. Bateman DN. Management of pyrethroid exposure. *J Toxicol Clin Toxicol*. 2000;38(2):107–109.
5. Narahashi T. Neuroreceptors and ion channels as the basis for drug action: past, present, and future. *J Pharmacol Exp Ther*. 2000;294(1):1–26.
6. Soderlund DM, Clark JM, Sheets LP, et al. Mechanisms of pyrethroid neurotoxicity: implications for cumulative risk assessment. *Toxicology.* 2002;17(1):30–59.
7. Bhaumik A, Gupta PK. Teratogenicity of decamethrin in rats. *Indian Vet J*. 1990;2:213–219.
8. O'Malley M. Clinical evaluation of pesticide exposure and poisonings. *Lancet.* 1997;349(9059):1161–1166.
9. McLachlan JA. Environmental signaling: what embryos and evolution teach us about endocrine disrupting chemicals? *Endo Rev.* 2001;22(3):319–341.
10. Elunwood EH. Amphetamine psychosis: description of the individuals and processes. *J Nerv Ment Dis.* 1967;144(4):273–283.
11. Norton S. Amphetamine as a model for hyperactivity in rat. *Physiol Behav*. 1973;11(2):181–186.
12. Broadhurst PL. Experiments in psychogenetics. In: Eisenk HJ, editor. *Experiments in personality*. London: Routledge and Kegan paul; 1960:31–71.
13. Kulig BM, Lammers JHCM. Assessment of neurotoxicant induced effects on motor function. In: Tilson HA, Mitchell C, editors. *Neurotoxicology. Target organ series in toxicology*. New York: Raven Press; 1992:147– 179.
14. Schallert T, Whishaw IQ. Two types of aphagia and two types of sensorimotor impairment after lateral hypothalamic lesions: Observations in normal weight, dieted, and fattened rats. *J Comp Physiol Psychol*. 1978;92(4):720–741.
15. Meyer OA, Tilson HA, Byrd WC, et al. A method for the routine assessment of fore-and hindlimb grip strength of rats and mice. *Neurobehav Toxicol*. 1979;1(3):233–236.
16. Chesterman H, Heywood R, Perkin CJ, et al. Oral toxicity study in Beagle dogs. 1977.
17. Barnes JM, Verschoyle RD. Toxicity of new pyrethroids insecticide. *Nature.* 1974;248(5450):711.
18. Ray DE. An EEG investigation of decamethrin-induced choreathetosis in the rat. *Exp Brain Res*. 1980;38(2):221–227.
19. Ray DE, Cremer JE. The action of decamethrin (a synthetic pyrethroid) on the rat. *Pestic Biochem. Physiol*. 1979;10(3):333–340.
20. Verschoyle RD, Aldridge WN. Structure–activity relationship of some pyrethroids in rats. *Arch Toxicol.* 1980;45(4):325–329.
21. Clark JM, Brooks MW. Role of ion channels and intraterminal calcium homeostasis in the action of deltamethrin at presynaptic nerve terminals. *Biochem Pharmacol*. 1989;38(14):2233–2245.
22. Parkin PJ, Quesne Le PM. Effect of a synthetic pyrethroid deltamethrin on excitability changes following a nerve impulse. *J Neurol Neurosurg Psychiatry*. 1982;45(4):337–342.
23. Cabral JRP, Galendo D. Carcinogenicity studies with deltamethrin in mice and rats. *Cancer Lett*. 1986;49(2):147–152.
24. Wu A, Liu Y. Apoptotic cell death in rat brain following deltamethrin treatment. *Neurosci Lett*. 2000;279(2):85–88.
25. Sayim F, Yavasoglu NUK, Uyanikgil Y, et al. Neurotoxic effects of cypermethrin in Wistar rats: A haematological, biochemical and histopathological study. *Journal of Health Science*. 2005;51(3):300–307.
26. Mokhtar I, Yousef A, Talaat I, et al. Deltamethrin–induced oxidative damage and biochemical alterations in rat and its attenuation by vitamin E. *Toxicology*. 2006;227(3):240–247.
27. Manna S, Bhattacharyya D, Mandal TK, et al. Neuropharmacological effects of deltamethrin in rats. *Vet Sci*. 2006;7(2):133–136.
28. Chen YL, Casida JE. Photodecomposition of pyrethrin I, phthalthrin, and dimethrin: Modification in the acid moiety. *J Agric Food Chem*. 1969;17(2):208–21.
29. Kavlock RJ, Daston GP, DeRosa C, et al. Research needs for the risk assessment of health and environmental effects of endocrine disruptors: a report of the U.S. EPA– sponsored workshop. *Environ Health Perspect*. 1969;104 Suppl 4:715–740.
30. Madsen C, Claesson MH, Ropke C. Institute of toxicology, national food agency, soeberg, denmark. immunotoxicity of the pyrethroid insecticides deltametrin and alpha-cypermetrin. *Toxicology*. 1996;107(3):219–227.
31. Elbetieha A, Da'as SI, Khamas W. Evaluation of the toxic potentials of cypermethrin pesticide on some reproductive and fertility parameters in the male rats. *Arch Environ Contam Toxicol*. 2001;41(4):522–528.
32. Patro N, Mishra SK, Chattopadhyay M. Neurotoxicological effects of deltamethrin on the postnatal development of cerebellum of rat. *J Biosci*. 1997;22(2):117–130.
33. Varshneya C, Singh T, Sharma LD. Immunotoxic responses of cypermethrin, a synthetic pyrethroid insecticide in rats. *Indian J Physiol Pharmacol*. 1992;36(2): 123–126.
34. Ivinskis A. A study of validity of open-field measures. *Aust J Psychol*. 1970;22(2):175–183.
35. Kelley A. Locomotor activity and exploration. Methods in behavioral pharmacology, chapter 5, 1993. p. 499–518.
36. Walsh RN, Cummins RA. The open field test: a critical review. *Psychol Bull*. 1976;83(3):481–504.
37. Whimbey AE, Denemberg VH. Two independent behavioral dimensions in open-field performance. *J Comp Physiol Psychol*. 1967;63(3):500– 504.

38. Lazarini CA, Florio JC, Lemonica IP, et al. Effect of prenatal exposure to deltamethrin on forced swimming behavior, motor activity, and striatal dopamine levels in male and female rats. *Neurotoxicol Teratol*. 2001;23(6):665–673.
39. Bhattacharya SK, Mitra SK. Anxiogenic activity of quinine-an experimental study in rodents. *Indian J Exp Biol*. 1992;30(1):33–37.
40. File SE. The use of social interaction as a method for detecting anxiolytic activity of chlordiazepoxide- like drugs. *J Neurosci Methods*. 980;2(3):219–238.
41. File SE, Hyde JR. Can social interaction be used to measure anxiety? *Br J Pharmacol*. 1978;62(1):19–24.
42. Sanchez C, Arnt J, Costall B, et al. The selective sigma2-ligand Lu 28– 179 has potent anxiolytic– like effects in rodents. *J Pharmacol Exp Ther*. 1997;283(3):1323–1332.
43. Hudson PM, Tilson HA, Chen PH, et al. Neurobehavioral effects of permethrin are associated with alterations in regional levels of biogenic amine metabolites and amino acid neurotransmitters. *Neurotoxicology*. 1986;7(1):143–153.
44. Kavlock R, Chernoff N, Baron R, et al. Toxicity studies with decamethrin, a synthetic pyrethroid insecticide. *J Environ Pathol Toxicol*. 1979;2(3):751–765.
45. Gandhi DN, Dhull DK. Postnatal behavioural effects on the progeny of rat after prenatal exposure to methylmercury. *American Journal of Experimental Biology*. 2014;1(1):31–51.
46. Joya, Sangha GK. Development and behavioural toxicity of deltamethrin on Rattus norvegicus following gestational exposure. *Journal of Applied and Natural Science*. 2016;8(1):40–45.