INTERNATIONAL JOURNAL OF AGRICULTURE & BIOLOGY
ISSN Print: 1560–8530; ISSN Online: 1814–9596
22–0239/2022/28–1–59–66
DOI: 10.17957/IJAB/15.1952
http://www.fspublishers.org

Full Length Article

Laboratory and Field Efficiency of Lambda Cyhalothrin against Black Rat (*Rattus rattus*)

Randa A Kandil*, Heba Y Ahmed and Nema M El-Abd
Harmful Animals Research Department, Plant Protection Research Institute, Agriculture Research Center, Giza, Egypt
*For correspondence: mhassanein11@hotmail.com
Received 04 June 2022; Accepted 05 July 2022; Published 31 July 2022

Abstract

The present study aims to test the rodenticidal effect of lambda cyhalothrin (LCT) and its toxic effects on serum cholinesterase activity, brain malondialdhyde level, brain glutathione content and histological alterations of brain and stomach of black rat, *Rattus rattus.* To achieve this purpose, adequate concentration of lambda cyhalothrin bait (0.032%) was evaluated as treated bait *via* non- and free-choice feeding method. The data concluded that lambda cyhalothrin bait gave 80% mortality and the time of death ranged between 4–5 days in non-choice feeding test. Also, in the free-choice feeding test, it caused 80% mortality with 10–15 days' time of death, and achieved high acceptance percent 40.94%. Oral administration of 1/4 LD_{50} of lambda cyhalothrin caused suppression in serum cholinesterase activity, brain glutathione content and increase in malondialdhyde activity. In parallel with this toxic effect, marked histological alterations in brain and stomach tissues were observed. The effect of lambda cyhalothrin bait was assessed against *R. rattus* under crops stores conditions in Sids village, Beba district, Beni-Suef Governorate. The results elucidated that lambda cyhlothrin bait achieved 71.34% reduction in rat population. In conclusion, the findings of this study indicated that lambda cyhalothrin have rodenticidal efficacy under laboratory and field conditions. It could induce marked toxicity *via* oxidative stress and suppression of antioxidants as well as lesions occurred in brain and stomach tissues led to death of the treated rats. Therefore, it can be used in the integrated rodent control programs.

Keywords: Rodenticidal; Lambda cyhalothrin; *Rattus rattus*; Free-choice; Brain glutathione; Efficacy; Control program; Field conditions

Introduction

Rodents are considered the most destructive animal pests affecting the agricultural production in many countries (Jokić *et al*. 2010). In addition to their damaging to crops, rodents can cause severe damage to buildings and telecommunication equipments (Sinha 2014). Various techniques are applied to control rodents, inclusive manipulation of habitat, trapping and chemical control using rodenticide baiting (Witmer 2019). Rodenticide application is the quickest and cheap management mean obtainable (Baldwin *et al*. 2016). Zinc phosphide is a recorded toxicant for rodents control but it is susceptible to drooping in efficacy because of bait shyness (Horak *et al*. 2018). Anticoagulant-resistant rodents have been determined from various lands and pose a major problem for pest management (Hazra *et al*. 2017). Therefore, researchers should search for new compound to solve these problems.

Pyrethroids insecticides are considered axonic poisons acts on the nerve fiber by linking to a protein that controls the sodium channel. Comparing to Type I pyrethroids, that do their neurotoxicity *via* involvement with sodium channel function, Type II pyrethroids could influence chloride and calcium channels affecting on valid nerve function (Syed *et al*. 2018). Lambda cyhalothrin (LCT) is type II pyrethroids and used in agriculture, protection of food production and disease vector control (Fetoui *et al*. 2010; Lofty *et al*. 2013). Recorded oral LD_{50} of LCT is 79 mg/kg for male rat and 56 mg/kg for female rats (Kidd and James 1991; Mate *et al*. 2010). Treatment with k-cyhalothrin and cypermethrin caused decrease in the activity of acetylcholinesterase of *Channa punctatus* (Kumar *et al*. 2009). LCT induced marked inhibition in acetylcholine activity of *Oreochromis niloticus* (Piner and Uner 2014). LCT accumulates in tissue biological membranes initiating reactive oxygen species (ROS) changing the antioxidant systems and elevates lipids peroxidation (LPO) in mammals (Metwally *et al*. 2017). Huge quantities of ROS can oxidize lipids, proteins and nucleic acids leading to many health disorders (Snezhkina *et al*. 2019). Acute and chronic exposure to LCT caused decrease in mice brain antioxidants as well as histopathological alterations in brain (Pawar *et al*. 2016).

Searching for new rodent control techniques becomes a thrilling topic for researchers. So, the purpose of the current study is laboratory and field evaluation of synthetic pyrethroid, LCT, to control black rats, *Rattus rattus* and its biochemical and histological effects on the brain and stomach.

Materials and Methods

Tested compounds

Lambda cyhalothrin (LCT): Dolf-X 5% EC was obtained from Starchem Chemical Manufacturing Co., 6th of October city, Giza, Egypt. Oral LD_{50} for rats is 79 mg/kg (World Health Organization 1990; Mate *et al*. 2010). It was used as bait (LCT mixed with crushed maize and used sun flower oil). Used bait is composed of (0.63 mL LCT+ 2.52 mL used oil + 96.85 g crushed maize).

Laboratory experiments

Experimental animals: Adult black rats, *R. rattus*, were collected live from stores, factories and houses located at Abu-Roash, Giza, Egypt by hundred rat traps (30 × 15 × 20 cm) submitted with fresh bait (tomato, cucumber or falafel). Animals were caught and transferred to Harmful Animals Research laboratory, Agriculture Research Center (ARC), Dokki, Giza, Egypt. Rats were acclimatized separately in cages of size 50 × 30 × 30 cm at 20–25°C and 12 h daily light dark cycles for 15 days before the commencement of experiment. Crushed maize and water were provided *ad libitum*. Pregnant and unhealthy rats were excluded. ninety rats (180–200 g) were partitioned into nine groups (each group of ten rats), six groups for treatments and three as control.

Non-choice feeding method: Serial concentrations of LCT bait (0.016, 0.024, 0.032 and 0.048%) were tested with constant factor (1.5%). It was used as bait (LCT mixed with crushed maize and used sun flower oil). Each rat was offered to treated bait 50 g for four successive days and the consumed bait quantity was weighted once a day. Then treated bait was excluded and ordinary crushed maize was introduced to live animals and monitored up to 28 days. The mortality percentages were registered over this period (Shefte *et al*. 1982).

Free-choice feeding method: This method is substantial to evaluate the acceptance of LCT bait by comparing its consuming with challenge diet (Palmateer 1974). Each animal was supplied with 50 g of LCT bait and 50 g of challenge diet (65% crushed maize + 25% ground wheat + 5% sugar + 5% corn oil) in different small bowls. Their positions were daily transformed to avoid location predilection through four consecutive days. Bait consumption and percent of mortality were recorded daily. As well as, percent of acceptance was deemed according to Mason *et al*. (1989).

$$\text{Acceptance (\%)} = \frac{\text{Treated bait consumption}}{\text{Treated bait consumption} + \text{challenge diet consumption}} \times 100$$

Biochemical studies

Samples preparation: A group of rats were administered orally with 1/4 LD_{50} of LCT (19.75 mg/kg b.wt). After seven days of treatment, animals were sacrificed and blood samples were collected from cervical vein and left to coagulant at room temperature. The brain was separated, placed in NaCl (0.9%) and was homogenized by Teflon homogenizer under cooling. Then, these specimens were centrifuged at 3000 rpm for 30 min. The clear supernatant serum was removed and it was put in deep freezer at 30°C until used. The same process was occurred with control rats.

Determination of serum cholinesterase activity, brain glutathione (GSH) content and brain Malondialdhyde (MDA) level

Serum cholinesterase activity was determined using reagent kit obtained from Quimica Clinica Aplicada S.A. Company (Spain) according to the method of (Kendel and Bottger 1967). Brain GSH content and MDA level were assessed utilizing reagent kit bought from Biodiagnostic Company (Egypt) according to (Beulter *et al*. 1963) and (Ohkawa *et al*. 1979) methods, respectively.

Histological studies

Brain and stomach were immediately removed from untreated and treated animals, put in 10% buffered formalin, washed in tap water and dehydrated using alcohol. Then were cleared in xylene and embedded in paraffin. Paraffin blocks were prepared and sectioned at 4 microns thickness by microtome. The prepared sections were deparaffinized and stained with hematoxylin and eosin (H&E) stain for detection *via* the light microscopy (Banchroft *et al*. 1996).

Field experiment

Field estimation of LCT bait (0.032%) was carried out under field crops stores condition of Sids Village, Beba, Beni-suef Governorate and infected with *R. rattus*. The area 1250 m^2 was splitted into three depots for treatment as well as three as control. Rats' intensity was determined pre and post treatment using food consuming method (Dubock 1984). This technique occurred with free crushed maize weighting 3000 g, divided into small black plastic sacks containing 50 g of each sack and were put inside bait station (plastic tube of 50 cm in length and 12 cm in diameter) distributed inside and outside stores. The consumed bait amount were weighted daily for five days and ejected, then the average consumption was estimated on the fourth and fifth days. After that, treated bait was applied in each bait station weekly for three weeks and the rest bait was

weighted once a week. For another week, the bait stations were left empty in the place. Then for one more week, untreated crushed maize was placed inside each bait stations, as mentioned above. The consumed amount was recorded and the population reduction percent was calculated after three weeks as follows:

$$\text{Population reduction (\%)} = \frac{\text{Pre-treatment consumption} - \text{Post-treatment consumption}}{\text{Pre} - \text{treatment consumption}} \times 100$$

Statistical Analysis

Experimental design was completely randomized with different replicate. The data were statistically analyzed using one-way ANOVA and also least significant difference (LSD) at ($P \leq 0.05$) *via* costal program (Cohort Software 2005).

Results

Effect of LCT bait against *R. rattus* in laboratory

Data demonstrated the non-choice feeding test of LCT bait at different concentrations against black rat, *R. rattus*, is represented in Table 1. The average bait consumption was (10.26, 10.32, 7.36 and 0 g) for (0.016, 0.024, 0.0.32 and 0.048%) concentrations of LCT bait which gave 50, 50, 80 and 0% mortality respectively. The most effective bait concentration was (0.032%) that achieved 80% mortality and the time of death ranged between 4–5 days with 2.25-day mean. There was significant difference between treated bait consumption compared to control feeding in non-choice feeding test. Concerning the free-choice feeding test (Table 2), the average consumption of bait was 7.93 g of challenge diet. But it was 5.83 g for treated bait with high acceptance percent 40.94% and it caused 80% mortality with time of death ranged between 10–15 days and 11.25 day mean. There is a significant difference between challenge diet and treated bait consumption compared to control.

Effects of LCT on serum cholinesterase activity, brain glutathione (GSH) content and brain MDA activity

Data in Table 3 illustrated the effect of 1/4 LD_{50} of LCT on cholinesterase activity, brain GSH content and brain MDA activity of *R. rattus*. LCT caused significant suppression in cholinesterase activity of treated animals with difference percent of (-52.21%). Regarding brain GSH content, LCT compound caused significant decrease (-35.43%). On the other hand, treatment with LCT induced marked significant elevation in MDA activity comparing with brain tissue of control with difference percent of (44.57%).

Histopathological effects

Histopathological check of the brain sections of untreated rats revealed normal histological architecture structure of subiculum hippocampus, fascia dentate, striatum and cerebellum in Fig. 1, 3, 5, 7, respectively. While, Fig. 2, 4 and 6 shows that LCT treatment caused several lesions including nuclear pyknosis and degeneration of cells of hippocampus, striatum and fascia dentate consecutively. Concerning the effect of LCT on cerebellum, there is no histological change in Fig. 8. In Fig. 9 the stomach of control rat shows normal structure of mucosa, submucosa, muscularis and circular mucosa. While the animals treated with LCT showed alterations in stomach submucosa including odema with inflammatory cells infiltration (Fig. 10).

Field evaluation

The efficiency of LCT (0.032%) bait evaluated against *R. rattus* under field conditions of crops stores. Results in Table 4 showed the average consuming of crushed maize in pre-treatment was 498,03g from 3000 g and treated bait consumption was 737.97 g while the consumption of post-treatment was 142.73 g. The data disclosed that LCT bait achieved 71.34% reduction in rat population. There is significant difference between consumption regions of LCT bait, pre-treatment and post-treatment compared to control region.

Discussion

LCT is non-systemic pyrethroid insecticide that can induce pernicious effects on the nervous tissue and other organs causing neurotoxicity, behavioral and biochemical dysfunctions (Basir *et al*. 2011; Waheed *et al*. 2011; Al-Jammas 2020). In the present study, increasing concentrations of LCT bait in non-choice test caused gradual increase in mortality percent of *R. rattus*. While LCT bait (0.048%) caused bait shyness, so the most appropriate concentration of LCT bait is 0.032% that gave 80% mortality.

The treatment of rats in our study with LCT bait (0.032%) in both choice and non-choice methods caused marked death and this may be due to the addition of used sun flower oil which enhanced the food consumption percent *via* eaten causing degeneration in brain and bleeding from stomach. These data are in parallel with previous researches (Kidd and James 1991; Mate *et al*. 2010). Treatment of *R. rattus* with abamectin mixed with used oil using non and free feeding choice methods caused mortality percent of 100 and 80% respectively due to repeated accumulation bait and high acceptance percent of palatability due to using used oil (Kandil *et al*. 2015).

In the present study, administration of 1/4 LD_{50} of LCT motivates high suppression in cholinesterase activity. These effects are in agreement with other authors who stated that treatment of *Rana cyanophlyctis* with LCT markedly decreased the value of cholinesterase (Khan *et al*. 2003). Previous studies detected adverse neurotoxic impact of

Table 1: Effect of different concentrations of LCT bait against black rat for four days *via* non- choice feeding technique

LCT bait concentration (%)	Average bait consumption (g) (Mean ± SE)		LSD	Mortality %	Time of death (day)	
	Control	Treated			Range	Mean
0.016	$10.19^{a} \pm 0.91$	$10.26^{a} \pm 0.23$	1.21	50	4-5	2.25
0.024		$10.32^{a} \pm 0.87$		50		
0.032		$7.36^{b} \pm 1.38$		80		
0.048		0.00^{c}		0		

Values are expressed as means (consumptions) ± standard errors ^{abc} values in column with different letters are significantly different at ($P \leq 0.05$). LSD: Least Significant Difference

Table 2: Effect of LCT bait (0.032%) against black rat *via* free- choice feeding technique

Average consumption (g) (Mean ± SE)			LSD	Acceptance %	Mortality %	Time of death (day)	
Control	Challenge diet	Treated bait				Range	Mean
$10.19^{a} \pm 0.91$	$7.93^{b} \pm 0.11$	$5.83^{c} \pm 0.44$	0.87	40.94	80	10-15	11.25

Values are expressed as means (consumptions) ± standard errors ^{abc} values in column with different letters are significantly different at ($P \leq 0.05$). LSD: Least Significant Difference

Table 3: Effect of 1/4 LD_{50} of LCT on cholinesterase activity, brain GSH content and brain MDA activity

Parameter Group	Cholinesterase (U/L)	Difference %	GSH (mg/g tissue)	Difference %	MDA (nm/g tissue)	Difference %
Treatment	15733.84 ± 3914.48^{b}	-52.21	$11.48^{b} \pm 1.91$	-35.43	$39.44^{a} \pm 3.94$	44.57
Control	32922.2 ± 3770.67^{a}		$17.78^{a} \pm 0.97$		$27.28^{b} \pm 0.55$	
LSD	8711.09		5.93		11.03	

Values are expressed as means ± standard errors ^{ab} values in column with different letters are significantly different at ($P \leq 0.05$). LSD: Least Significant Difference

Table 4: Efficiency of LCT bait (0.032%) against black rat under crops stores conditions

Treatment	Amount of crushed maize or bait distributed (g)	Average consumption of bait (g) Mean ± SD	Population reduction %
Control	3000	$790.82^{a} \pm 89.65$	71.34
Pre-treatment		$498.00^{b} \pm 14.60$	
Treatment		$737.97^{ab} \pm 32.53$	
Post-treatment		$142.73^{c} \pm 4.11$	
LSD	-	306.53	

Values are expressed as means (consumptions) ± standard deviation ^{abc} values in column with different letters are significantly different at ($P \leq 0.05$). LSD: Least Significant Difference

LCT. LCT blocks the closing of voltage-sensitive neuronal sodium ion channels and alters normal nerve function in both insects and mammals leading to paralysis or death (Schleier and Peterson 2012; Tomar *et al.* 2015). It has been reported that some actions directly returned to toxicity of pesticides can be as a result to disturbance in membrane fluidity as well as in lipid composition and also enzyme activities inhibition (Mossa *et al*. 2013). Due to its lipophilic nature, LCT can cause reverse effects on many tissues (Fetoui *et al.* 2010; Saleem *et al.* 2014). In our current study, treatment of *R. rattus* with 1/4 LD_{50} of LCT promotes significant elevation ($P < 0.05$) in brain MDA level compared with normal rats. These changes may be due to that LCT can induce ROS (Metwally *et al.* 2017). LCT can accumulate in biological tissue membranes initiating ROS which disrupt the antioxidant systems and elevate LPO in mammals. Metabolism of LCT occurs quickly in liver through ester hydrolytic cleavage and oxidative paths by CYP450 enzymes causing production of ROS (Sankar *et al.* 2012). MDA is considered the main secondary lipid peroxidation product of polyunsaturated fatty acids (Ayala *et al.* 2014). Different concentrations of LCT can induce oxidative stress and also damaging DNA (Piner and Üner 2012). It is well known that the non-enzymatic antioxidants simultaneously diminished in pesticides toxicity (Arab *et al.* 2018). In parallel with this a critical lowering in GSH level in LCT toxicity, in the present study, could lead to raised vulnerability of the brain tissue to free radical injury. The suppression of GSH level can be elucidated as a result to high GSH usage for conjugation and/or its role in balancing free-radicals (El-Demerdash 2012). LCT can cause oxidative damage due to their lipophilicity, so they could easily sneak the cell membrane (El-Saad and Abdel-Wahab 2020).

Our results are supported by the pathological lesions in brain and stomach of tested animals. Alterations in all parts of brain (hippocampus, fascia dentate and striatum) were observed including pyknosis and degeneration in cells. LCT could inhibit mitochondria in hippocampus of rat brain (Benaicha *et al*. 2021). Mitochondria permeability promotes mitochondria edema and also induces a release of various mitochondrial intermembrane proapoptotic proteins in the cytosol such as cyt-C leading to cell death (Webster 2012). It was stated that brain enzyme systems are able to generate autolytic characteristics for the cell, especially the lipolysis process, proteolysis and protein phosphorylation (Miller and Zachary 2017). Also, LCT treatment in the present study induced marked lesions in stomach submucosa including odema with inflammatory cells infiltration. Regarding the application in crops store, LCT caused 71.34% reduction in

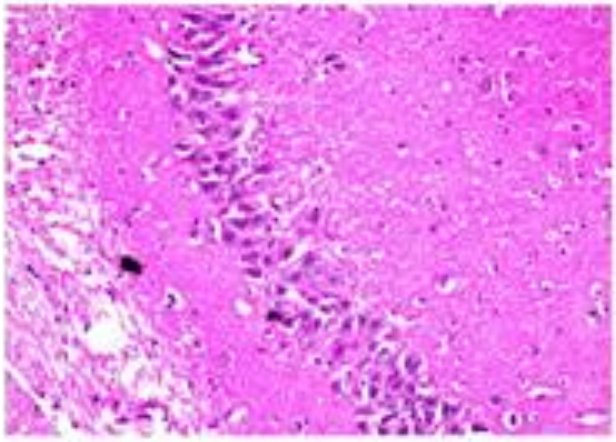

Fig. 1: Photomicrograph of H&E stained subiculum hippocampus of brain of untreated rats showing normal structure. × 400

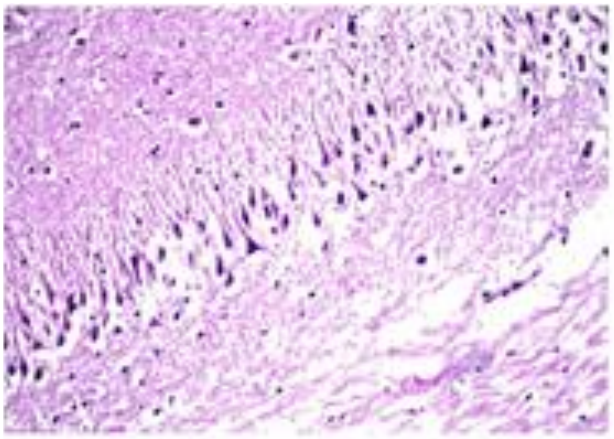

Fig. 2: Photomicrograph of H&E stained subiculum hippocampus of brain of LCT-treated rat showing nuclear pyknosis degeneration of hippocampus cells. × 400

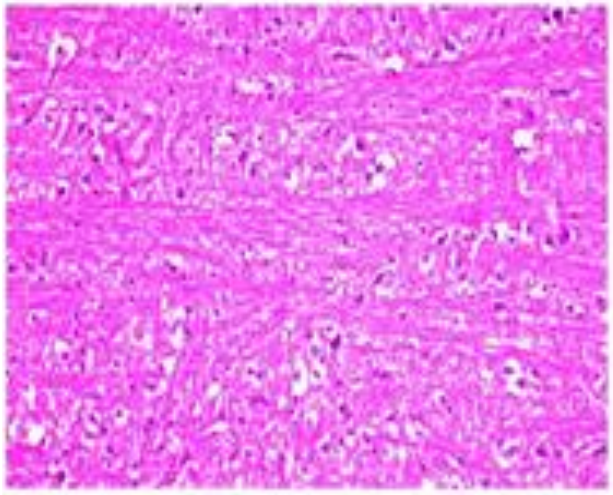

Fig. 3: Photomicrograph of H&E stained striatum of brain of untreated rat showing normal structure. × 400

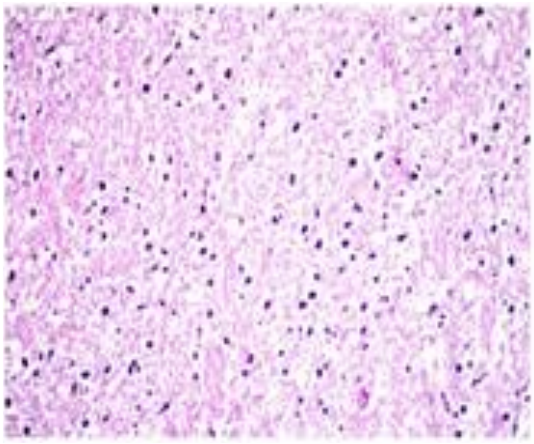

Fig. 4: Photomicrograph of H&E stained striatum of brain of LCT-treated rats showing nuclear pyknosis degeneration of striatum cells. × 400

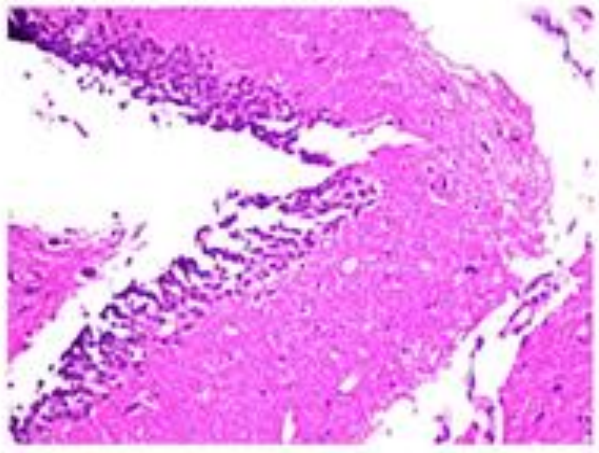

Fig. 5: Photomicrograph of H&E stained fascia dentate of brain of untreated rats showing normal structure. × 400

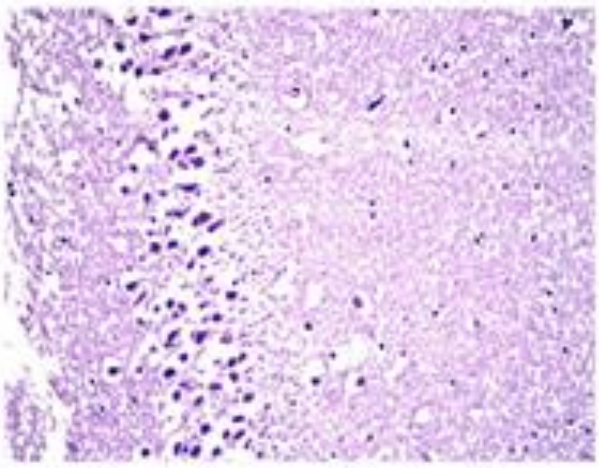

Fig. 6: Photomicrograph of H&E stained fascia dentate of brain of LCT-treated rats showing nuclear pyknosis degeneration of neuron cells. × 400

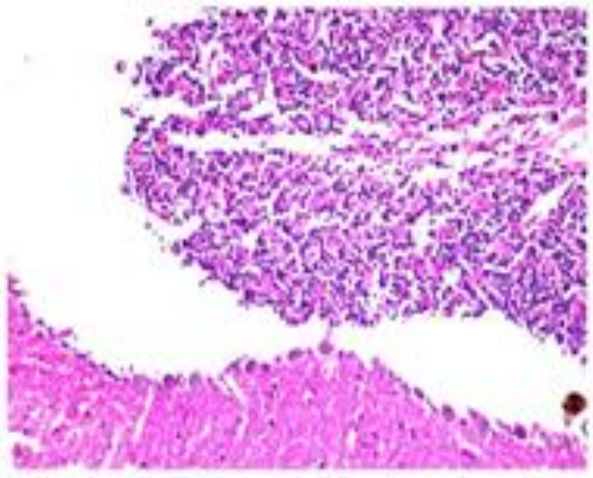

Fig. 7: Photomicrograph of H&E stained cerebellum of brain of untreated rats showing normal structure of cerebellum. × 400

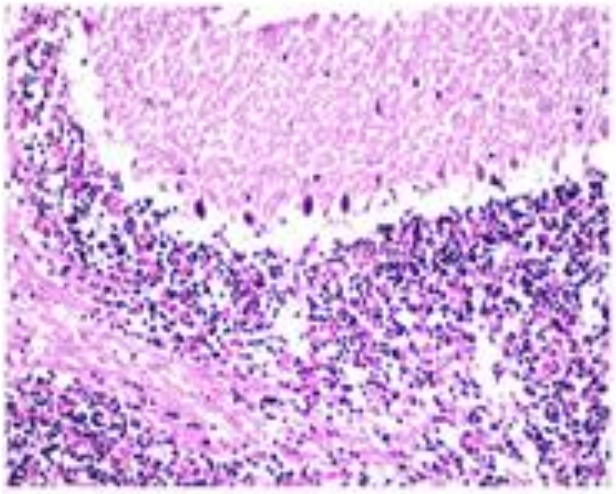

Fig. 8: Photomicrograph of H&E stained cerebellum of brain of LCT-treated rats showing normal structure of cerebellum. × 400

R. rattus population. The addition of the used oil may have raised LCT bait palatability causing rats to prefer feeding on bait rather than on stored crops inducing death. Mortality may be due to LCT increase reactive oxygen species causing toxicity in animal body *via* increasing MDA activity.

Conclusion

Treatment of *R. rattus* with LCT bait induced marked mortality in laboratory and field. This toxicity is evidenced by decreasing cholinesterase activity, GSH content and increasing MDA activity and inducing lesions in brain and stomach of animal's tissue which led to reduce the population of rats in the crops stores. Therefore, LCT can be used in the integrated rodent management in the concentration of 0.032%.

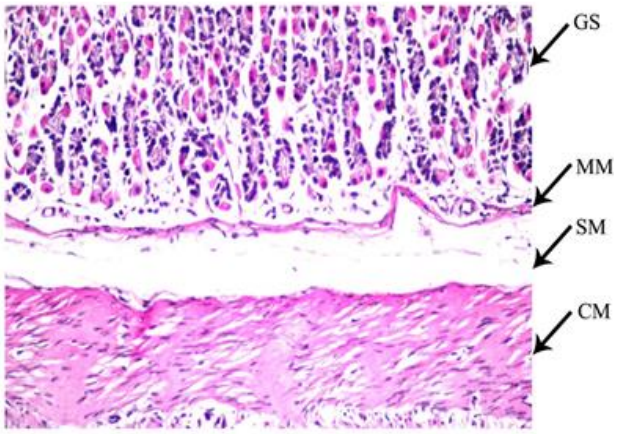

Fig. 9: Photomicrograph of H&E stained stomach section of untreated rat showing normal structure of gastric mucosa (GM), muscularis mucosa (MM), submucosa (SM) and circular muscle (CM). ×400

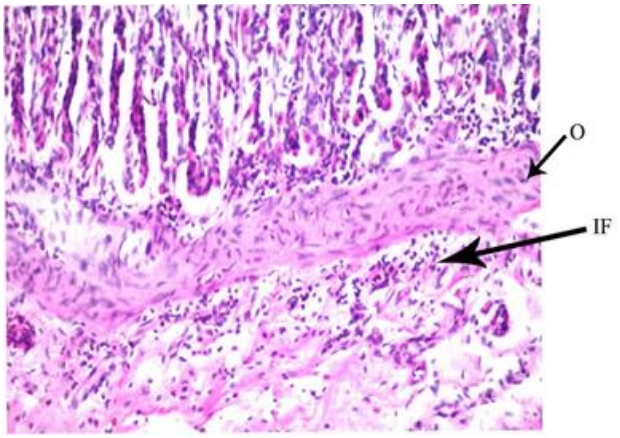

Fig. 10: Photomicrograph of H&E stained stomach section of LCT treated rat showing odema (O) and inflammatory cells infiltration (IF) in submucosal layer. × 400

Acknowledgment

The authors would like to express their sincere appreciation to Prof. Dr. Waheed Gabr, Professor at Harmful Animals Research Department, Plant Protection Research Institute, ARC, for correcting the manuscript scientifically. The authors are very grateful to Prof. Dr. Soha A. Mobarak, Assistant professor in Harmful Animals Department, Plant Protection Research Institute, ARC.

Author Contributions

Randa A. Kandil proposed the research plan, processed the laboratory and field experiments and shared in writing the manuscript. Heba Y. Ahmed shared in proposing the research plan, performed the laboratory and field

experiments and participated in writing the manuscript. Nema M. Abd participated in proposing the research plan, contributed in laboratory and field experiment and in writing the manuscript. All authors read and approved the final manuscript.

Conflict of Interest

The authors declare that they have no competing interests.

Ethics Approval

None.

References

Al-Jammas S (2020). The histological changes induced by cytarabine on rabbit's kidneys (with and without vitamin E administration). *Iraq J Vet Sci* 34:9–13

Arab SA, MR Nikravesh, M Jalali, AZ Faze (2018). Evaluation of oxidative stress indices after exposure to malathion and protective effects of ascorbic acid in ovarian tissue of adult female rats. *Electr Phys* 10:6789–6795

Ayala A, MF Muñoz, S Argüelles (2014). Lipid Peroxidation: Production, Metabolism, and Signaling Mechanisms of Malondialdehyde and 4-Hydroxy-2-Nonenal. *Oxid Med Cell Longev* 2014:1–31

Baldwin RA, R Meinerz, GW Witmer (2016). Cholecalciferol plus diphacinone baits for vole control: A novel approach to a historic problem. *J Pest Sci* 89:129–135

Banchroft JD, A Stevens, DR Turner (1996). *Theory and practice of histological techniques*, 4th Edition, p:766. Churchil Livingstone, New York, London, San Francisco, Tokyo

Basir A, A Khan, R Mustafa, M Zargham-Khan, F Rizvi, F Mahmood (2011). Toxicopathological effects of lambda-cyhalothrin in female rabbits (*Oryctolagus cuniculus*). *Hum Exp Toxicol* 30:591–602

Benaicha B, R Rouabhi, S Gasmi, F Menaceur, I Mennai, Z Lakroun, H Fetoui, M Kebieche, R Soulimani (2021). Triggering mitochondrial dysfunction and apoptosis under chronic low-dose lambda cyhalothrin exposure modulated by *Melissa Officinalis* L. methanol extract in rat hippocampus. *Mol Cttular Biochem* 2021:1–28

Beulter E, O Duron, MB Kelly (1963). Improved method for the determination of blood glutathione. *J Lab Clin Med* 16:882–888

Cohort Software (2005): Costat Program V. 6.311. (780 lighthouse), Ave. PMB 320. Monterey, California, USA

Dubock AC (1984). Pulsed baiting – a new technique for high potency, slow acting rodenticides. *In: Proceedings of Conference of the Organization and Practice of Vertebrate Pest Control*, pp:105–103, Hampshire, UK

El-Demerdash FM (2012). Cytotoxic effect of fenitrothion and lambda-cyhalothrin mixture on lipid peroxidation and antioxidant defense system in rat kidney. *J Environ Sci Health B* 47:262–268

El-Saad AMA, WM Abdel-Wahab (2020). Naringenin Attenuates Toxicity and Oxidative Stress Induced by Lambda-cyhalothrin in Liver of Male Rats. *Pak J Biol Sci* 23:510–517

Fetoui H, M Makni, M Garouiel, N Zeghal (2010). Toxic effects of Lambda-cyhalothrin, a synthetic pyrethroid pesticide, on the rat kidney: Involvement of oxidative stress and protective role of ascorbic acid. *Exp Toxicol Pathol* 62:593–599

Hazra DK, R Karmakar, R Poi, S Bhattacharya, S Mondal (2017). Rodent menace, their management and role of possible new rodenticide formulations to combat resistance. *J Entomol Zool Stud* 5:202–206

Horak K, NM Hofmann, BA Kimball (2018). Assessment of zinc phosphide bait shyness and tools for reducing flavor aversions. *Crop Prot* 112:1–22

Jokić G, P Vukša, M Vukša (2010). Comparative efficacy of conventional and new rodenticides against *Microtus arvalis* (Pallas 1778) in wheat and alfalfa crops. *Crop Prot* 29:487–791

Kandil RA, ME Mohallal, SA Mobark (2015). Rodenticidal efficiency of abamectin biocide on black rat, *Rattus rattus* under laboratory field condition in new reclaimed land. *Egypt J Agric Res* 93:657–664

Kendel M, R Bottger (1967). Kinetic method for Determination of pseudo. Cholinesterase (acetylcholine, acytylhdrolease) activity. *Klin Wschr* 45:325–327

Khan MZ, M Zaheer, F Fatima (2003). Effect of Lambda Cyhalothrin (Pyrethroid) and Monocrotophos (Organophosphate) on Cholinesterase Activity in Liver, Kidney and Brain of *Rana cyanophlyctis*. *Kor J Biol Sci* 7:165–168

Kidd H, DR James (1991). *The Agrochemicals Handbook*, 3rd edn., pp:27–33. *Royal Society of Chemistry Information Services, Cambridge*, UK

Kumar A, DK Rai, B Sharma, RS Pandey (2009). λ-Cyhalothrin and cypermethrin induced *in vivo* alterations in the activity of acetylcholinesterase in a freshwater fish, *Channa punctatus* (Bloch). *Pest Biochem Physiol* 93:96–99

Lofty HM, AAA El-Aleem, HH Monir (2013). Determination of insecticides malathion and lambda-cyhalothrin residues in zucchini by gas chromatography. *J Bull Fac Pharm Cairo Univ* 51:255–560

Mason JR, ML Avery, DL Otis (1989). Standard protocol for evaluation of repellant effectiveness with bird. *Birds Sect Res* 20:1–20

Mate MS, RC Ghosh, S Mondal, DB Karmakar (2010). Effect of Lambda Cyhalothrin on Rats: An Acute Toxicity Study. *J Ind Soc Toxicol* 6:25–28

Metwally HG, HF Abd-Ellah, NEM Shaheen, MSH Afifi, NI Al-Zail (2017). Role of Mesenchymal stem cells in the treatment of testicular toxicity induced by Lambda-Cyhalothrin in Rats. *Wulfenia J* 24:108–138

Miller MA, JF Zachary (2017). Mechanisms and Morphology of Cellular Injury, Adaptation and Death. *Pathol Bas Vet Dis* 2017:2–43

Mossa AH, AA Refaie, A Ramadan, J Bouajila (2013). Amelioration of Prallethrin-Induced Oxidative Stress and Hepatotoxicity in Rat by the Administration of *Origanum majorana* Essential Oil. *Biomed Res Intl* 2013:1–11

Ohkawa H, W Ohishi, K Yagi (1979). Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Anal Biochem* 95:351–358

Palmateer SD (1974). Laboratory testing of albino rats with anticoagulant rodenticides. *In: Proceedings of the Vertebrate Pest Conference* Vol. 7 pp:63–72. University of Nebraska, Lincoln, Nebraska, USA

Pawar NN, PC Badgujar, LP Sharma, AG Telang, KP Singh (2016). Oxidative impairment and histopathological alterations in kidney and brain of mice following subacute lambda-cyhalothrin exposure. *Toxicol Ind Health* 33:277–286

Piner P, N Uner (2014). Neurotoxic effects of lambda-cyhalothrin modulated by piperonyl butoxide in the brain of *Oreochromis niloticus*. *Environ Toxicol* 29:1275–1282

Piner P, N Üner (2012). Oxidative and apoptotic effects of lambda-cyhalothrin modulated by piperonyl butoxide in the liver of *Oreochromis niloticus*. *Environ Toxicol Pharmacol* 33:414–420

Saleem U, S Ejaz, M Ashraf, M Ovais-Omer, I Altaf, Z Batool, R Fatima, M Afzal (2014). Mutagenic and cytotoxic potential of Endosulfan and Lambda-cyhalothrin – *in vitro* study describing individual and combined effects of pesticides. *J Environ Sci*26:1471–1479

Sankar P, AG Telang, A Manimaran (2012). Protective effect of curcumin on cypermethrin-induced oxidative stress in Wistar rats. *Exp Toxicol Pathol* 64:487–493

Schleier JJ, RKD Peterson (2012). The joint toxicity of type i, ii, and nonester pyrethroid insecticides. *J Econ Entomol* 105:85–91

Shefte N, LR Bruggers, EW Schafer (1982). Repellence and toxicity of three African grain eating birds. *J Wild Manage* 46:453–457

Sinha B (2014). Non-Empirical Validation of Indigenous Rodent Control Methods Practiced in Northeastern India. *In: Proceedings of Indian National Science Academy*, Vol. 80, pp:235–245, New Delhi, India

Snezhkina AV, AV Kudryavtseva, OL Kardymon, MV Savvateeva, NV Melnikova, GS Krasnov, AA Dmitriev (2019). ROS Generation and Antioxidant Defense Systems in Normal and Malignant Cells. *Oxid Med Cell Longev* 2019:1–17

Syed F, KK Awasthi, LP Chandravanshi, R Verma, NK Rajawat, VK Khanna, PJ John, I Soni (2018). Bifenthrin-induced neurotoxicity in rats: Involvement of oxidative stress. *Toxicol Res* 7:48–58

Tomar M, A Kumar, SK Kataria (2015). Evaluation of acute toxicity of Lambda cyhalothrin in *Mus musculus* L. *Ind J Exp Biol* 53:551–555

Waheed RM, HH Bakery, RM El-Shawarby, MEA Salem (2011). Toxicity of lambda cyhalothrine on erythrogram, liver and kidney with molorated by vitamine C. *BVMJ* 22:238–248

Webster KA (2012). Mitochondrial membrane permeabilization and cell death during myocardial infarction: Roles of calcium and reactive oxygen species. *Future Cardiol* 8:863–684

Witmer GW (2019). The changing role of rodenticides and their alternatives in the management of commensal rodents. *Human Wildlife Intl* 13:186–199

World Health Organization (1990). Cyhalothrin, Environmental Health, p:34. Criteria, 99; Geneva, Switzerland