Research Paper

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Imidacloprid Induced Neurotoxic and Histological Changes in Female Albino Rats

Zoology

KEYWORDS : Imidacloprid, Histopathology, Female albino rats, AChE

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ABSTRACT

Imidacloprid was given orally to female albino rats for 60 days. Rats were divided into three groups. Group 1 served as Control and was given corn oil through oral intubation. Group 2 served as Treated 1 given 1/45th LD50 of imidacloprid. Group 3 served as Treated 2 and given 1/22th LD50 of imidacloprid for 60 days to assess the acetylcholine esterase activity (AChE) in brain andhistopathological changes in brain and kidney of female albino rats.Imidaclopridat 1/22th LD50 of imidacloprid exposure leads to marked inhibitionin AChE activity in brain and histopathological changes in brain and kidney of imidacloprid (1/22th LD50) treated rats.

Introduction

Pesticides are a broad group of heterogeneous chemicals that have a significant public health benefit by increasing food production and decreasing food-borne and vector-borne diseases. However they are found to affect non-target organisms, including humans depending on the agent and the exposure (Chaudhary et al .1999). Imidacloprid is a neonicotinoid insecticide and classified under toxicity class II /III agents by United States Environmental Protection Agency (USEPA, 1994). Imidacloprid and its analogs are remarkably potent neurotoxic insecticides, which act as nicotinic acetylcholine receptor agonists (nAChRs) (Matsuda 2005). nAChRs play a central role in rapid cholinergic synaptic transmission and are important targets of insecticides (Sattelle 1990).It is one of the fastest sold insecticide across the world because of its high selective toxicity in insects and apparent safety in humans (Tomizawa*et al*., 2005). Its selective toxicity results from its high affinity to insect's nicotinic acetylcholine receptors compared to mammals (Tomizawa and Casida, 2003). It acts on nervous system by blocking post-synaptic acetylcholine receptors, which kills the insect (Tomizawa*et al*., 2005). Recently imidacloprid has raised concern because of its ability to cause egg shell thinning, reduced egg production and hatching time, which are considered as signs of possible endocrine disrupters (Berny et al., 1999; Matsuda et al., 2001) and honey bee colony collapse disorder (Faucon et al., 2005, Chauzat et al., 2006, 2009). Theneonicotinoids act as agonist at the acetylcholinestrase receptors(nAChRs) of the insect and mammals (Tomizawa and Casida,2003). Toxicity correlates with agonist cation and binding affinityat the vertebrate a4b2nAChR, the primary target in the brain(Tomizawa et al., 2001). Since imidacloprid is now being considered for replacement of other existing pesticides therefore the relative risk and benefits of this insecticide must be compared to the existing pesticide.The histopathological studies can be especially useful because they are a relatively sensitive indicator of damage and they provide information on toxicity from a variety of protocols.One of the most important processes in kidneys is excretion of metabolic waste products by glomerular and tubular filtration so we examined kidney for histopathological changes.

Materials and methods

Commercial product of imidacloprid (Confidor, 17.8%, SL) used in this study was purchased from the local market in Ludhiana, India.The study was conducted on sexually mature female albino rats of Wistar strain, 3 months of age, weighing 100–150 g obtained from Guru AngadDev Veterinary and Animal Sciences University (GADVASU), Ludhiana. The animals were housed in groups of two rats per cage. The rats were acclimatized for one week before using them for experimentation. The rats were maintained under controlled conditions of temperature (22 ± 2^0C) and humidity (30–70%) with 12 h light and dark cycle. The animals were given standard diet containing pelleted food and water ad libitum. The experimental protocol met the National guidelines on the proper care and use of animals in the laboratory research. The Institutional Animal Ethics Committee (IAEC) approved this experimental protocol. Adult females were divided into three groups. Group 1 served as Control and given corn oil through oral intubation. Group 2 served as Treated 1 given $1/45^{th}$ LD_{50}(lower dose) of imidacloprid. Group 3 served as Treated 2 given $1/22^{th}$ LD_{50}(higher dose) of imidacloprid for 60 days. Acetylcholine esterase activity was estimated by the method of Vass and Sachsseas modified by Morai*et al* (1946)

Histological Studies

After 60 days rats were anaesthetized by chloroform and dissected. Brain tissue was homogenized in phosphate buffer for the estimation of acetyl cholinesterase. Brain and kidneysamples were cleared from adhering tissues, fixed in 10% neutral formalin, dehydrated through ascending grades of alcohol and cleared in xylene. The wax-impregnated tissues were embedded in paraffin blocks and 5 µm thick sections were cut serially using microtome. Dewaxed slide sections were then rehydrated in descending grades of alcohol and stained with haematoxylin and eosin. Haematoxylin-eosin stained slides were studied under light microscope.

Results and Discussion

Significant AChE inhibition was observedat higher dose of imidacloprid as compared to control rats(Fig 1.)Imidacloprid produced similar inhibition in serum choline esterase activity in quails. (Zaahkooket al 2009). Exposure of high dose of imidacloprid for 60 days produced necrosedpurkinji cells with loss of dendrites in brain of female rats as compared to controls. There was degeneration of tubules and glomeruli of kidney of the female rats at higher dose as compared with controls. (Fig 2 and Fig 3).High dose of imidacloprid (20 mg/kg/day) resulted in inhibition in brain AChE activity and mild focal necrosis with swollen cellular nuclei, cytoplasmic lesions in rat liver and slight degeneration of tubules and glomeruli of kidney of the female rats (Bhardwaj et al. 2010). Imidacloprid produced similar histopathological lesions in liver, kidneys, and brain of Japanese quail exposed to chemical for 6 weeks (Omiama 2004) and in layer chickens exposed to 139 mg/kg imidacloprid (Kammon et al. 2010).Imidacloprid at oral doses of 15 mg/kg/day produced apparent histopathological changes in liver, kidneys, heart, and brain in male and female mice on 28th day of insecticide treatment. Vacuole degenerations, dilatation of sinusoids, dissociated remark cordons, pyknotic nuclei, and leukocyte infiltration were observed in livers of male and female mice. There were shrinkage of glomeruli and degeneration of epithelial cells in kidneys, degeneration in hearts, and focal gliosis in brains of male and female mice (Ince S 2013). Imidacloprid at $1/10^{th}$ of LD_{50} treatment resulted in dilatations of central vein and sinusoids between hepatocytes (Toor, Sangha, and Khera 2012).

Conclusion

Imidaclopridat $1/22^{th}$ LD_{50} of imidacloprid exposure leads to marked inhibition in AChE activity in brain and histopathological changes in kidney and brain of imidacloprid ($1/22^{th}$ LD_{50})

treated rats. So, imidacloprid at 1/22th LD_{50} was found to be potent toxic agent affecting histology in treated rats.

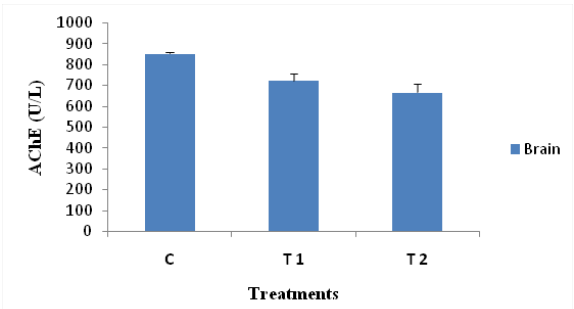

Fig 1.Acetylcholinesterase inhibition of brain of female rats after oral administration of imidacloprid Results are expressed as Mean ± S.E. *P<0.05, **P<0.01

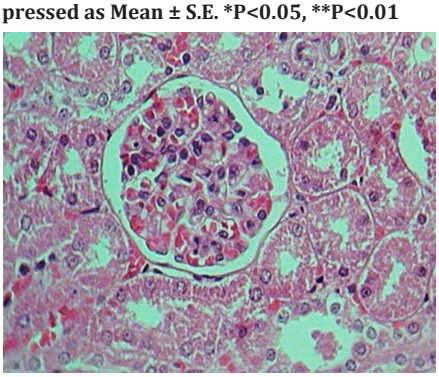

Fig 2. A. T.S of kidney of control female rats showing normal tubular and glomerular structures (H & E X 400)

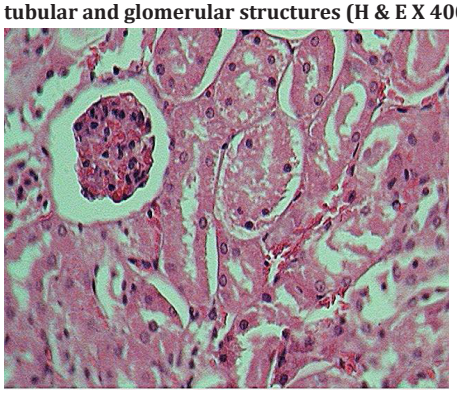

Fig 2. B. T.S of kidney of treated female rats (1/22 thLD$_{50}$ of imidacloprid) rats showing degeneration of tubular and glomerular structure

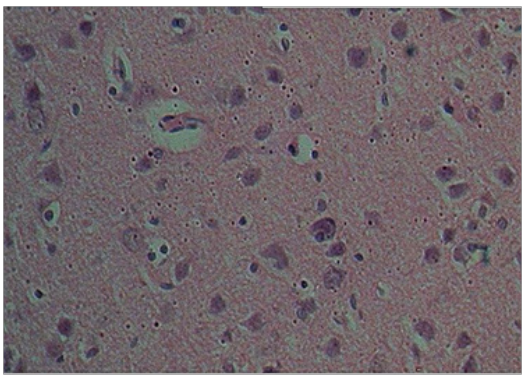

Fig 3. A. T.S of brain of control female rats showing normal structure of purkinji cells, molecular and granular layers (X 400)

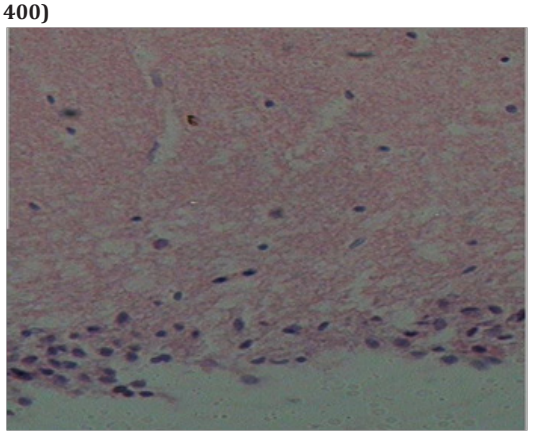

Fig 3. B. **T.S of brain of treated females rats (1/22th LD50 of imidacloprid) showing degenerative changes in purkinji cells and loss of granules in granular layer (H & E X 400)**

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