ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 05, pp.50994-50996, May, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

EFFECT OF PESTICIDE LAMBDA CYAHALOTHRIN ON HISTOLOGICAL ALTERATIONS IN THE GILL TISSUES OF FRESH WATER FISH, LABEO ROHITA UNDER SHORT TERM AND LONG TERM EXPOSURE PERIODS

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ARTICLE INFO

Article History:
Received 19th February, 2017
Received in revised form
21st March, 2017
Accepted 24th April, 2017
Published online 31st May, 2017

Key words:
Pesticide, Lambda cyhalothrin,
Histopathological study.

ABSTRACT

The exposure of the fresh water fish, *Labeo rohita* to lambda cyahalothrin water, for short term (24, 48, 72, 96 hours) and long term duration (10, 20, 30 days) lead to the formation of histopathological lesions of varying intensities on the gill tissues. In the present investigation, the histopathological changes in gill of Labeo rohita in normal condition and exposed to condition for short and long term period of lamda cyahalothrin water at selected periods have been observed

Citation: Dhanya, P.Y. and Dr. Sushama, S. 2017. "Effect of pesticide lambda Cyahalothrin on histological alterations in the gill tissues of fresh water fish, Labeo Rohita under short term and long term exposure periods", *International Journal of Current Research*, 9, (05), 50994-50996.

INTRODUCTION

Fish are widely used to evaluate the health of aquatic ecosystems because pollutants build up in the food chain and are responsible for adverse effects and death in the aquatic systems (Farkas *et al.,* 2002). Toxicity evaluation is an important and cost effective tool in water quality monitoring as it provides the complete response of test organisms to all the compounds in a cumulative way (Tisler and Koncan, 1999). Histopathological analysis has been tested and proposed as an efficient and sensitive tool to monitor the fish health and environmental pollution in natural water bodies (Costa *et al*., 2009). Aquatic pollutants directly or indirectly enter into the body of aquatic animals and affect different parts of their body and affect vital physiological mechanism (Kharat *et al*., 2011).

MATERIALS AND METHODS

Histopathological analysis of tissue sample

After exposure of 24hrs -30 days at the subethal concentration, the gill tissue of the fish *Labeo rohita* were collected and used for the histopathological study.

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The gills of fish once removed from fish were preserved in 10% formalin for nearly six hours. The well preserved gill were processed through dehydration (removing water from tissues) with dehydrating agents for 4 to 6 hours (Alcohol or Acetone) in three changes and the dehydrating agent within the tissue was cleared by clearing agent like xylene for 2-3 hours. After that the tissues were treated in melted paraffin wax of having melting points and 58-60 impregnated in the melted wax for 4 hours. Now the gill were well processed and impregnated, were embedded, within the wax. The blocks with embedded processed tissues were submitted for taking thin sections of 3 to5 micron size by using Rotary microtome. These sections taken on the glass slide were stained with haematoxylene and eosin stain. The stained sections were taken for microscopical study. The gill tissue excised from fishes of the control and experimental groups were fixed in 10 per cent formalin solution. After proper dehydration by graded alcohols, paraffin blocks were prepared and 4-5 μ thick ribbons were cut in rotary microtome and were stained with Eosin and Haematoxylin. The histopathological changes observed were photographed.

RESULTS AND DISCUSSION

The exposure of the fresh water fish, *Labeo rohita* to lambda cyahalothrin water, for short term (24, 48, 72, 96 hours) and

long term duration (10, 20, 30 days) lead to the formation of histopathological lesions of varying intensities on the gill tissues. In the present investigation, the histopathological changes in gill of *Labeo rohita* in normal condition and exposed to condition for short and long term period of lambda cyahalothrin water at selected periods have been observed.

Gill Histology

Control

Gill histology of the control fish revealed the intact nature of both primary and secondary gill lamellae. The secondary lamellar surface was covered with simple squamous epithelial cells and capillaries separated by mucous cells. Each primary gill lamellae was flat leaf like in structure. It consisted of double rows of secondary (respiratory) lamellae with the central supporting axis. They were situated laterally on either side of the interbranchial septum. The secondary lamellae on both sides were highly vascularised and covered by a layer of epithelial cells with uniform interlamellar spaces (Plate A).

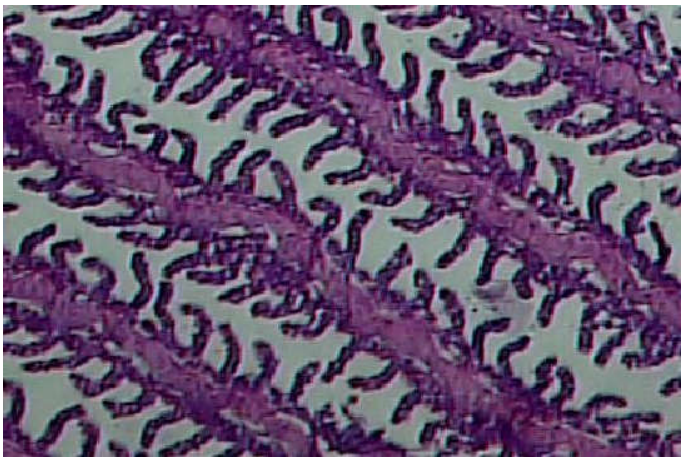

Plate A

When the fish was exposed for 24 hours to the short term exposure of Lambda cyahalothrin water, there was degeneration of epithelial lining (Plate. B). After 48 hours of exposure, degenerative changes in the secondary lamellae of gill was noted(Plate C) After 72 hours of exposure, there was fusion of secondary lamellae with irregular lamellar spaces (Plate D) After 96 hours of exposure, structural alterations such as epithelial proliferation, lamellar fusion and necrosis were observed (Plate.E).

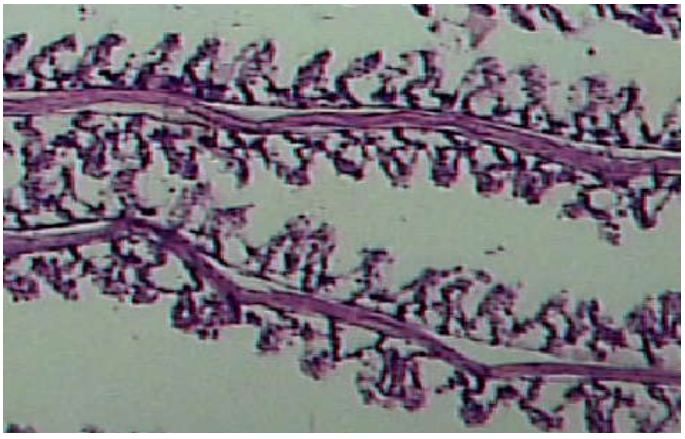

Plate B

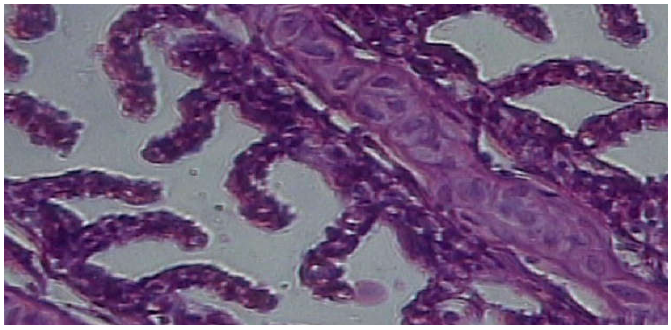

Plate C

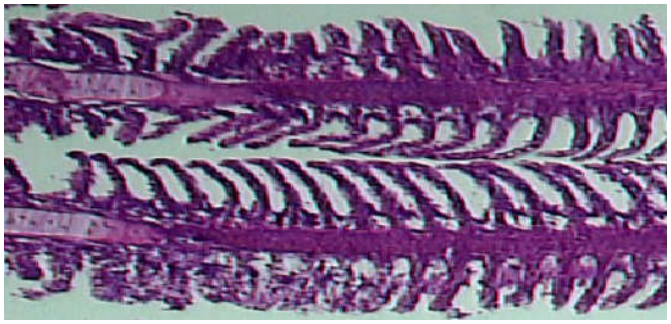

Plate D

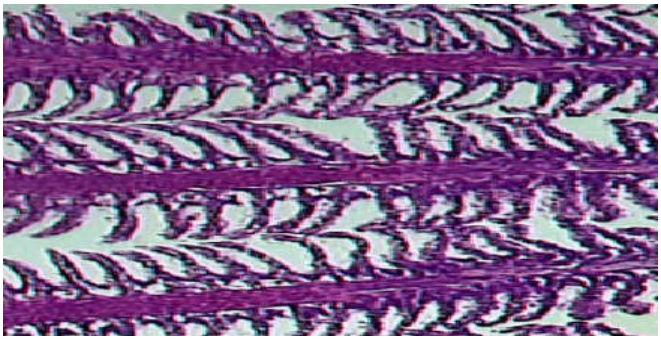

Plate E

When the fish was exposed for 10 days to long term exposure of lambda cyahalothrin water considerable degenerative changes were observed. The secondary lamellae showed necrosis at the basal region. It showed congestion with infiltration by the chronic infiltration of cellular exudates (Plate. F). After 20 days, papillary formation with dilated the vasculature. The infiltration was found with chronic inflammatory cells (Plate. G). After 30 days, the damage became more noticeable leading to collapsed secondary lamellae (Plate.H).

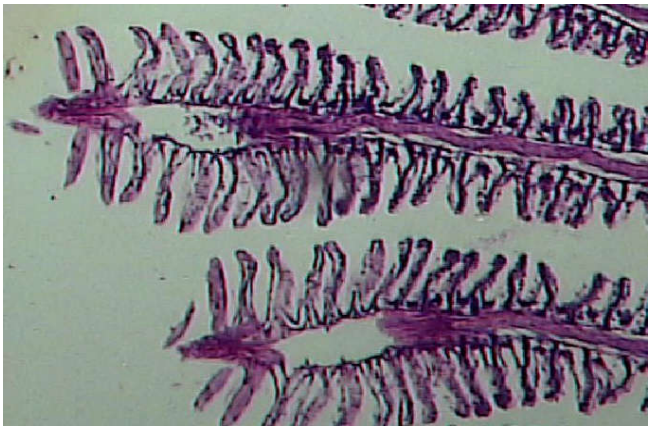

Plate. F

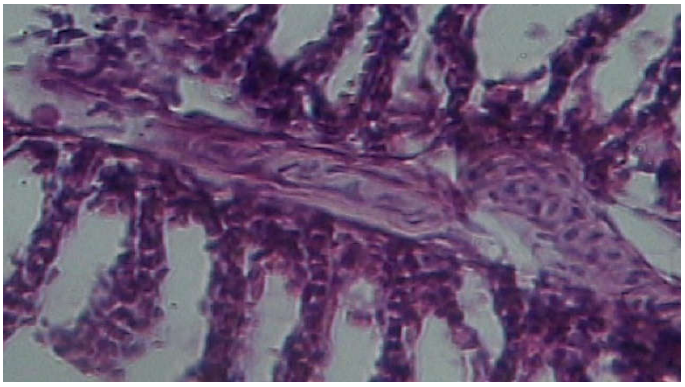

Plate G

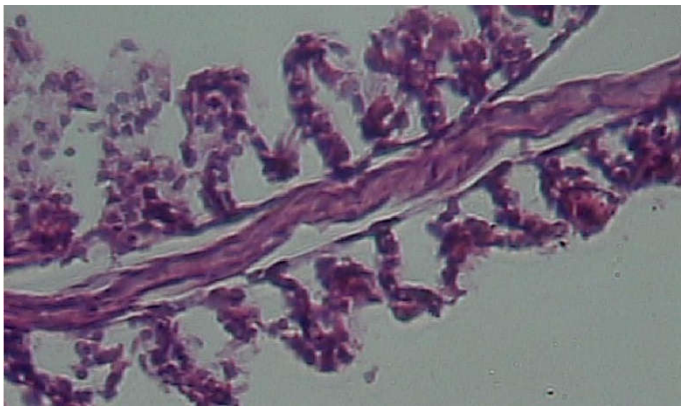

Plate H

Khidr *et al*. (2001) have observed the effects of changes in the gills of fish, *Cyprinus carpio* exposed to δ-methrin resulting in necrosis, vacuolar degeneration, desquamation, epithelial lifting, edema, shorting of secondary lamellae and lamellar fusion. Machado and Fanta, (2003) reported that organophorous methyl parathion intoxication in the fish, *Metynnis roosevelti* caused dropsy, vascular degeneration, cloudy swelling, necrosis and other degenerative changes in epithelial and pillar cells of the gills. Club shaped lamellae is an example of progressive degeneration in the gills. In the present study, the epithelial necrosis and desquamation of the gill epithelium are direct responses to the action of water pollutants. The defenses noticed are lifting up of the epithelium, hyperplasia and lamellar fusion.

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