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Assessment of fipronil toxicity on wistar rats: A hepatotoxic perspective

R.M. Kartheek, M. David*

Environmental Toxicology and Molecular Biology Laboratory, Department of PG Studies and Research in Zoology, Karnatak University, Dharwad- 580003, India

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ABSTRACT

Extensive pesticide application has contributed to environmental contamination globally, imposing adverse health effects on non-target organisms. Need for an understanding of cellular response following pesticide exposure is, therefore, paradigmatic for elucidating perturbations occurring within biological systems. The present investigation was aimed to examine safe and toxic dose level of a persistent, synthetic, phenylpyrazole based insecticide, Fipronil (FPN) on rat liver. Experimental animals were divided into four groups and gavaged with 0.0 (control), 32.33 (high), 12.12 (medium) and 6.46 mg/kg body weight/day (low dose) of FPN for 90 days. While results for liver catalase and glutathione *S*-transferase indicated significant changes in high and medium dose groups, the superoxide dismutase and glutathione peroxidase activity suggested significant changes in all exposed groups as compared to control. Elevated levels of liver malondialdehyde reflected oxidative damage potential under the exposed groups but remained insignificant for low dose. Histologically, structural irregularities with findings like impaired portal vein and hypertrophy of hepatocytes were prominent under all the exposed groups. The FT-IR based spectral investigation further revealed changes in absorption patterns and peak intensities in rats exposed to FPN. Significant elevation was also noticed in liver enzymes; alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase in rat serum suggesting the toxicity in dose -dependent pattern. Based on the outcome, it could be ascertained that the toxicity of FPN is certain at high and medium dose levels but remains ambiguous at a low dose of 6.46 mg/kg body weight/day. The current upshots serve as a preliminary report thereby advising the farming community against the usage of FPN insecticide.

1. Introduction

Large -scale anthropogenic activities under chemical scale have predominantly contributed to environmental contamination globally [1]. Increased chemical persistence, mainly pesticides, in soil and water have found their direct correlation to deteriorating health conditions of non-target species ranging from amphibians to mammals [2,3]. Pesticides belong to the only group of chemical toxicants that are intentionally applied to the environment with an objective of enhancing food production through countering insect pests and controlling disease vectors additionally [4]. Even though the consequences of pesticide toxicity are known to be many, establishment of oxidative stress is high paradigm due to the occurrence of reactive oxygen species (ROS) in all aerobic individuals [5]. Upshots in the imbalance of oxidant and antioxidant magnitudes, basically due to elevated proportions of ROS in vivo is therefore considered as an imperative indicator of chemical stress [6]. ROS, as known, is an inevitable and obligatory composite of aerobic mechanism, often produced paradoxically in hepatocytic mitochondria and endoplasmic reticulum, channelized through cytochrome P450 cluster [7]. Although its role in signaling pathways is highly critical, its existence in surplus amounts could impart catastrophic damage to cellular proteins, with the processes often being irreversible [8].

Besides, the mammalian liver is of exceptional concern for studying pesticide -induced toxicity in various experimental models [9]. Its ability to carry out detoxification contributes to homeostasis [10]. Hepatocytic proteins, lipids, and DNA on other hand are among the cellular structures that are primarily affected by ROS that basically arises through exposure to toxic chemicals [11]. The process has been found to result in structural and functional discrepancies of hepatic tissue, ultimately resulting in the impaired health. Attrition of liver as a consequence of incessant detoxification is often reflected by an upsurge in liver enzymes [12]. Basic tools for determining the damage to the liver include the assay of liver enzymes; alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) levels from blood serum. These components and the end products of the metabolic pathways are highly sensitive for determining the incurred abnormality and have been considered as biochemical markers of liver dysfunction [13].

Fipronil (5- amino- 1- (2, 6-dichloro- α, α, α- trifluoro- *p*- tolyl)- 4-

* Corresponding author.
E-mail addresses: karthikraobiotech@gmail.com (R.M. Kartheek), mdavid.kud@gmail.com (M. David).

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