



## Fipronil induced modulations in biochemical and histopathological aspects of male Wistar albino rats: A subchronic study

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### ABSTRACT

Growing demand of pesticides and their indiscriminate application has led to environmental contamination globally. The present investigation was aimed to elucidate the toxic potentials of fipronil (FPN) on liver biochemistry and histology of male Wistar albino rats. Rats were orally exposed to sub lethal dose of FPN (24.2 mg/kg body weight) for 1 (E1), 45 (E2) and 90 days (E3). The results suggested significant variations ( $P \leq 0.05$ ) in catalase, superoxide dismutase and glutathione S-transferase activities under E2 and E3 groups unlike E1, when compared with control group (C). Significantly elevated levels of malondialdehyde in rat liver noticed under E2 and E3 groups indicated oxidative damage to hepatocytes. The tissue damage confirmed through histopathological examination revealed findings like degenerated portal vein and necrosis in liver of FPN exposed rats. The findings suggest oxidative stress potential of FPN resulting from long term exposures (E2 and E3) unlike for single dose administration (E1). The modulations in histoarchitecture nevertheless indicates the possibilities of structural damage to liver under all exposure durations. Based on the outcome it is inferred that FPN is toxic to rats under prolonged exposure. It is therefore suggested that necessary precautions should be taken whenever FPN is used or disposed in areas with close mammalian proximity.

**Keywords:** Fipronil, Hepatotoxicity, Oxidative damage, Wistar rats and Xenobiotic.

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### INTRODUCTION

Large scale anthropogenic activities and indiscriminate use of synthetic organic chemicals has contributed to environmental contamination globally (Rhind, 2009; David and Kartheek, 2014). The wide range existence of insect pests has prompted the agricultural community to use broad spectrum insecticides, resulting in increased crop yield and reduced post-harvest losses. However, toxicity due to occupational exposure and accidental poisoning toxicity studies has been always debated in scientific community (Eisenstein, 2015). Based on the previous reports it is convincing that negligence during handling and application of pesticides could result in the greatest possibilities of pesticide exposure to field applicators and agricultural practitioners (Rauhet *et al.*, 2012; Kessler *et al.*, 2015; Lerro *et al.*, 2015).

Pesticides belonging to different class are known to control insect pests through impairing their cellular integrity and vacillating their biochemical mechanisms *in vivo* (David and Kartheek, 2016; Alavanja *et al.*, 2013; Mnif *et al.*, 2011). This mechanism is generally carried out by imparting critical conditions like oxidative stress resulting from elevated levels of reactive oxygen species (ROS) within biological systems (Costa *et al.*, 2008). Assessment of pesticides for their potential to induce oxidative stress in the exposed organism is one of the crucial methods for indicating pesticide toxicity (Arnal *et al.*, 2011).

ROS which is the responsible factor for imparting oxidative stress is never the less considered to be

an inevitable and obligatory composite of mitochondrial oxidative phosphorylation in eukaryotes (Ray *et al.*, 2012). Although its role in signalling pathways is highly critical, its occurrence in additional amounts is thought to result in catastrophic damage to cellular proteins (David and Kartheek, 2015), with the processes often being irreversible (Ahn and Baker, 2016). Its availability in appropriate threshold is therefore necessary to regulate and maintain the health and survival ability of the organism (David and Kartheek, 2015; Schieber and Chandel, 2014). The mammalian liver is of exceptional concern for studying oxidative stress, as it is recognized to be the site of detoxification of almost all xenobiotic components (Jaeschke *et al.*, 2002). As a result of which it is known to be largely affected through the breakdown of abundantly available poly unsaturated fatty acids and could interdependently result in production of ROS production (Cichoż-Lach, and Michalak, 2014).

FPN belongs to a new class of phenylpyrazole group (Tingle *et al.*, 2003) and is the relatively new yet, most widely used insecticide, having acknowledged for addressing issues related to insect resistance and public health hazards that are commonly encountered with conventional group of pesticide families (Bonmatin *et al.*, 2015). Majority of pesticides are known for disrupting biochemical and even possess tissue damaging propensities (Raj *et al.*, 2013). The literature support for outcome of biochemical and histopathological disruption remains well established for organophosphates (Subramanyaana *et al.*, 2012), organochlorines (Lakroun *et al.*, 2015) and pyrethroids (David and Kartheek, 2016; Fetoui *et al.*, 2010). However, similar toxicity effects have been rarely addressed for FPN so far, which aggravated the need to gauge their impact on mammalian class (Murillo *et al.*, 2011). Furthermore, the studies suggesting the impeding effects on functioning of