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Long term oral administration of a mixture of pyrethroids affects reproductive function in rats

Anandha Rao Ravula, Suresh Yenugu*

Department of Animal Biology, School of Life Sciences, University of Hyderabad, Hyderabad, 500046, India

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ABSTRACT

Pyrethroid toxicity using dosages that are relevant to the human settings are not reported. Male Wistar rats were treated for 9 or 12 months daily with a mixture of pyrethroids equivalent to a fifth or a twenty-fifth of that is present in cereals and vegetables consumed by an average Indian adult. Altered oxidant and antioxidant status, severe anatomical damage in the testis, caput, cauda, prostate, liver, lung and kidney and increased serum SGPT, SGOT and ALP activity was evident in all the treatment groups. Decreased levels of 3β- and 17β-HSD activity, litter size and impaired acrosome reaction was observed in all the treatment groups. To the best of our knowledge, for the first time, we demonstrate that exposure to even very low levels of pyrethroids (relevant to human consumption) for longer durations could cause damage to tissues that are important in general and male reproductive physiology.

1. Introduction

Pyrethroid usage to control pests in agricultural and domestic set ups continues to increase over a period of time. Synthetic pyrethroids are preferentially used because of their high insecticidal activity and biodegradability and thus accounts for almost one fourth of the world's insecticide market [1]. In general, a mixture of insecticides are used for improving crop production which contaminates the agricultural land and water [2]. An impending result of this is that they enter the food chain, which is evident from recent studies that many pyrethroids can be detected in food items such as rice, wheat, vegetables, fruits and nuts [3,4]. This is also true in the Indian context [5,6]. Thus, humans get constantly exposed to more than one pesticide at a time. Though many studies reported the effects of individual pesticides (including pyrethroids) on different organ systems, active investigations on the toxic effects of mixed pesticides have gained importance in the recent past because of the potential of human exposure via the food chain. In humans poisoned with a mixture of cypermethrin and chlorpyrifos, the ventilator free days were shorter when compared with humans exposed to one of the compounds [7].

Mixed pesticides are known to cause synergistic toxicity in animal models [8–10]. A large number of studies reported that exposure to pesticides and insecticides alters biochemical, histological and reproductive parameters. In a recent study, it is observed that oxidant and antioxidant status is altered in cypermethrin exposed pregnant rats and their new borns [11]. Similarly, we previously demonstrated alterations in oxidant and antioxidant status in adult rats and in off spring born to female rats exposed to allethrin [12–15]. A mixture of propoxur (PR) and permethrin (PE) when administered to rats contribute to disrupted redox status in the liver of rats [16]. Biochemical profiling related to liver and kidney function tests are reported in animal studies to evaluate pyrethroid toxicity. Measuring aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALKP), total bilirubin (TBIL), total proteins (TPs), cholesterol (CHOL), urea, and creatinine provide vital information on the damage caused to vital organs during pyrethroid exposure. Aberrations in ALKP, AST, ALT, TBIL, TPs, cholesterol, urea and creatinine was observed in mice treated with deltamethrin [17]. Pyrethroid toxicity also causes disruption in the steroidogenesis by influencing the activities of the steroidogenic enzymes, especially 3β- and 17β-hydroxy steroid dehydrogenase (HSD) [14,18–21]. Histopathological changes induced by a combination of pesticides is reported [16,22,23]). A recent study indicates that malformations in the reproductive tract is evident in rats exposed to mixed pesticides [24]. A combination of chloropyrifos and cypermethrin contributes to reproductive toxicity in male albino mice [23]. Further, a combined low dose of cyromazine, 4-(2-methyl-4-chlorophenoxy) butyric acid (MCPB), pirimicarb, quinoclamine, thiram, and ziram caused decreased birth weight in rats [25]. A mixture of pesticides at dose equivalent to the no-observed-effect-level (NOEL) caused impairment in spermatogenesis in rats [26].

* Corresponding author.
E-mail addresses: sureshsl@uohyd.ernet.in, ysnaidu@yahoo.com (S. Yenugu).

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