

AGRO ISN'T POP: NEUROENTERIC DYSFUNCTION INDUCED BY TEBUCONAZOLE IN WISTAR RATS

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INTRODUCTION: Considering the exponential growth of agribusiness and the intensification of agricultural practices, there is an increasing use of chemical pesticides, especially herbicides such as Tebuconazole (TEB). Although they are essential for pest control, indiscriminate use can lead to significant impacts, such as contamination of water and food, promoting bioaccumulation and biomagnification throughout the trophic chain. The growing exposure, especially through the food chain, highlights the gastrointestinal tract (GIT) as a potential biomarker due to its role in digestion and absorption of substances. This organ contains specific cells and an independent central nervous system, the enteric nervous system (ENS), which regulates the functions of GIT and is essential for maintaining homeostasis. Thus, the GIT and ENS may be affected by contaminants such as TEB. **OBJECTIVE:** Investigate the effects of subchronic exposure of Tebuconazole on the myenteric plexus of the duodenum in Wistar rats. **METHODS:** 36 male Wistar rats were kept under controlled conditions and randomly divided into four groups: a control group (CON) and three experimental groups receiving 10 mg/kg (E10), 20 mg/kg (E20), and 50 mg/kg (E50) of TEB. After 31 days, the animals were anesthetized, euthanized, and their duodenum was collected for histochemical analysis (NADH-dp and NADPH-dp) to evaluate the neuronal density. The results were statistically analyzed using One-Way ANOVA ($p < 0.05$). **RESULTS:** Exposure to TEB resulted in a significant reduction in the density of metabolically active neurons (NADH-dp) at E50 (14.61 ± 0.61) compared to the control group (CON) (23.66 ± 5.22). This reduction suggests a possible imbalance in other neuronal subpopulations, especially considering that many myenteric neurons are nitroergic, meaning they produce nitric oxide (NO). This result is evidenced by the reduction in NADPH-dp density at E50 (12.93 ± 0.79) and E10 (12.05 ± 1.53), compared to CON (15.51 ± 0.83). Although NO has a neuroprotective effect, it was insufficient to counteract TEB's harmful effects. Exposure may affect neuronal populations, including cholinergic neurons expressing choline acetyltransferase (ChAT), responsible for peristalsis. This could cause severe dysfunctions in individuals exposed to TEB. **CONCLUSIONS:** The results elucidate that TEB-exposure may impact on different populations of neurons, contributing to neuronal degeneration.

KEY WORDS: Herbicides; Gastrointestinal Tract; Gut Health; Myenteric Neurons; Agrotoxics.

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