EVALUATION OF NEPHROTOXICITY IN RATS AFTER REPEATED DAILY EXPOSURE TO THE HERBICIDE CLOMAZONE

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Introduction: Brazil is one of the largest consumers of pesticides in the world, due to its diverse climate, wide range of agricultural crops, and prominent role as a global food producer. The herbicide clomazone (2-(2-chlorobenzyl)-4,4-dimethyl-1,2-oxazolidin-3-one) is widely used in soybean, rice, and sugarcane crops, primarily acting against grasses. Its mechanism of action involves the inhibition of carotenoid biosynthesis, leading to chlorophyll degradation. The kidneys, as key organs responsible for xenobiotic elimination, are potential targets of toxicity and warrant special attention in toxicological studies. **Objective:** To assess the tubular kidney damage through the quantification of the enzyme N-acetyl-β-D-glucosaminidase (NAG) in the urine of rats treated with clomazone. **Materials and Methods:** Rats were orally treated with clomazone at doses of 15, 30, and 60 mg/kg for 28 days. The control group received distilled water (n = 6 animals/group). Following euthanasia (CEUA-UFRGS 43478), urine was collected in Eppendorf tubes for spectrophotometric analysis of NAG enzymatic activity. **Results:** No statistically significant differences in NAG activity were observed between the clomazone-treated groups and the control group. **Conclusion:** The lack of changes in NAG activity suggests that renal tubular damage is not a primary mechanism of clomazone toxicity under the experimental conditions evaluated. However, further investigation using additional nephrotoxicity biomarkers is recommended.