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Terbuthylazine toxicity through a 28-day study: Oxidative stress responses and primary DNA damage in the blood of adult male Wistar rats

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Terbuthylazine is a chlorotriazine herbicide that acts primarily as an inhibitor of photosynthesis. It is used mainly in the production of maize but is also applied in the cultivation of various fruits, and vegetables, as well as in forestry. We evaluated the *in vivo* effects of terbuthylazine following a 28-day oral administration in adult male Wistar rats at 2.300 mg/kg b.w./day, 0.400 mg/kg b.w./day, and 0.004 mg/kg b.w./day. After treatment, oxidative stress responses were measured in the blood of both the terbuthylazine-treated and the control rats using biomarkers of lipid peroxidation, activities of glutathione peroxidase (GSH-Px), superoxide dismutase (SOD) and catalase. To establish DNA damaging effects, we applied an alkaline comet assay on the leukocytes of the exposed and control animals. Exposure to terbuthylazine slightly disturbed lipid peroxidation, which was not dose-dependent. Treatment also led to increases of GSH-Px activity in blood, which was statistically significant at 0.400 mg/kg b.w./day. The observed increase of SOD activity in erythrocytes was most prominent at the highest dose tested. On the other hand, plasma SOD activity dropped significantly at two lower doses. Catalase activity in plasma decreased, which was statistically significant at the highest dose. Terbuthylazine produced measurable effects on leukocyte rat DNA. Based on the values measured for comet tail length and tail intensity, which were both lower at the two higher concentrations, it can be concluded that one of possible mechanisms of terbuthylazine's effect on the DNA molecule includes intercalation. This may in turn slow down the migration of DNA during electrophoresis. Taken together, our findings suggest that the 28-day exposure of rats to very low doses of terbuthylazine, which were within toxicology reference values, resulted in oxidative stress responses and low-level DNA instability. Our results call for further research using other sensitive biomarkers of effect, along with different exposure scenarios.

Biography

Anja Mikolić has graduated from the Faculty of Food Technology and Biotechnology, University of Zagreb in 2005. She acquired her PhD in Biomedicine and Health Sciences from the Faculty of Pharmacy and Biochemistry, University of Zagreb in 2015. She is employed in Analytical Toxicology and Mineral Metabolism Unit at Institute for Medical Research and Occupational Health (IMROH), Zagreb, Croatia since 2007. Her professional fields are designing and performing *in vivo* studies on experimental animals (rats), studying biomarkers of oxidative stress; assessment of human exposure to essential and toxic metals by bio-monitoring, interactions of metals and reproductive toxicity, development and application of analytical methods regarding hormone and antioxidant enzyme analysis. She has published 6 original scientific papers in the international peer-reviewed journals, participated in few national and international scientific conferences and is a member of few scientific associations.

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