



EFFECT OF UROLITHIN A AGAINST OXIDATIVE DAMAGE INDUCED BY MICROCISTYN-LR IN GLIA CELLS

8º Simpósio de Segurança Alimentar - Sistemas Alimentares e Alimentos Seguros, 8ª edição, de 03/10/2023 a 05/10/2023
ISBN dos Anais: 978-65-5465-068-7

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RESUMO

Introduction: The proliferation of cyanobacteria in water bodies is an important matter in public health discussion, as it produces cyanotoxins, such as Microcystin-LR (MIC-LR). Despite being a known hepatotoxic toxin, the toxic effects of MIC-LR in the nervous system have been recently reported. MIC-LR's intoxication happens through the consumption of water and food contaminated and reactive species (RS) generation, as well as, oxidative stress, are involved in the intoxication mechanisms of MIC-LR. Thus, antioxidants present in foods may be an alternative as potential chemoprotectants against the toxic effects of cyanotoxins. Ellagitannins (ET) are present in nuts, seeds, and fruits and, after gastrointestinal digestion, release ellagic acid (EA). The non-absorbable fraction of EA is converted by gut microbiota in urolithins (URO), which have been associated with benefits coming from ETs and EA rich foods. **Objectives:** The present study aimed to evaluate the putative protective effect of URO-A, the predominant isoform of URO, against MIC-LR toxicity in glia cells. **Methodology:** C6 cells were pre-treated with URO-A at 3 μ M for 24 h before being exposed to 10 μ M MIC-LR. After 24h of MIC-LR exposure, the treatments were removed and cells were washed and used in cell viability and reactive species (RS) generation assays. **Results and discussion:** C6 cells treated with 10 μ M MIC-LR presented increased RS generation along with reduced cell viability, when compared with the control group, corroborating the neurotoxic ability of this toxin. URO-A attenuated the RS generation induced by the MIC-LR ($p < 0.05$), although it has not been effective in mitigating cell death caused by the cyanotoxin ($p > 0.05$). Thus, it is possible that other mechanisms are involved in MIC-LR toxicity and they are not totally covered by the URO-A's antioxidant capacity. **Conclusion:** URO-A might be helpful against the damage produced by MIC-LR toxicity in glia cells by preventing RS generation. Nevertheless, further research is required before a final statement about the protective effect of this colonic metabolite against MIC-LR intoxications can be made.

PALAVRAS-CHAVE: cyanotoxins, oxidative stress, phenolic compounds, survival loss

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