Protective Effect of *Oreochromis niloticus* Lectin (OniL) Against Toxicity Caused by Tert-Butyl Hidroperoxide (t-BOOH) in B16-F10 murine melanoma cells

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Cell oxidative stress occurs when the antioxidant mechanisms are overwhelmed by ROS and the resulting damage can lead to cell death either by necrosis or apoptosis. t-BOOH is an organic hydroperoxide, broadly used to induce oxidative stress in a variety of cells. In this study we evaluate the protective action of *Oreochromis niloticus* lectin (OniL) against oxidative stress induced by t-BOOH as well as the viability, ROS and death of B16-F10 murine melanoma cells exposed to t-BOOH. Melanoma cells (10⁶ cells) were treated with OniL during 24h and then cell oxidative stress was induced with 400 µM t-BOOH, during 2 h. The cell viability was determined by trypan blue exclusion assay or by annexin V conjugated with fluorescein isothiocyanate and propidium iodide. Annexin-FITC-/PI+ cells were considered necrotic and annexin-FITC+/PI- represented cells in apoptosis. Mitochondrial production of ROS was estimated with the MitoSOX Red probe (5 µM) and monitored by flow cytometer. Nonparametric assays were used in statistical analysis. Although OniL did not decrease mitochondrial ROS production, it protected against toxicity induced in melanoma cells by t-BOOH. A 58% increase in cell viability and a decrease in the number of necrotic cells by 25.5% were observed. The mechanism of such a protection by OniL is under investigation.

Key Words: lectin, oxidative stress, melanoma cells

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