

Expression of Glycogen Metabolism Genes Are Regulated by Hydrogen Peroxide in BME26 Tick Embryonic Cells

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Hydrogen peroxide (H_2O_2) is a chemical commonly used to induce cell damage by oxidative stress, via ROS production. In the last decade, evidences has pointed that ROS are able to perform crucial physiological functions, such as signal transduction and cell survival. Carbohydrate metabolism, like glycogen pathway, acts mainly to supply cellular energy demand, and also have an important participation in the maintenance of physiological homeostasis and prevention of oxidative damage caused by ROS. Glycogen via comprises glucose storage and it is an essential reserve in BME26 cells. Synthesis and breakdown of glycogen involves the activity of many enzymes and regulatory proteins, such as Glycogen Synthase(GS), Phosphoglucomutase(PMG) and Glycogen Debranting(GDE). In this sense, the present work has been investigating the glycogen metabolism in high concentrations of H₂O₂ in a *Rhipicephalus microplus* embryonic cells, BME26. BME26 cells were treated with H_2O_2 in non-lethal (2.2mM) and lethal (4.4mM) concentrations, during 2 and 24 hours, and cell viability was evaluated by MTT assay. After treatments, transcriptional analyses of key enzymes in glycogen metabolism (GS, PMG and GDE) were performed. Glycogen quantification was also determined. Additionally, in treated cells PAS staining was performed to identify the glycogen compartmentalization and the immunolocalization of GS, observed by bright field and confocal microscopy, respectively. Data suggests a differential adaptive control related to H₂O₂ concentration and to exposure time. Increased transcription of GS and PMG was observed in the early stages of H₂O₂ incubation. After 24 hours treatment, the transcription of glycogen metabolism enzymes was downregulated. It was also measured an increased glycogen content and stronger PAS staining in non-lethal H₂O₂ concentrations. Glycogen synthase was immunolocalized as strong perinuclear signal under H₂O₂ treatment. These results contribute to a better understanding how the redox-dependent pathways are linked to carbohydrate metabolism, showing that controlled stress might trigger hormesis phenomenon.

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