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# $H_2O_2$ stress damage is reversed by melatonin in a spinal cord organotypic model

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Spinal cord injury (SCI) is characterized to be a two-step process: the primary lesion consisting of the initial trauma; the secondary damage, characterized by multiple processes including inflammation, oxidative stress and cell death that lead to a significant expansion of the original damage and to an increase of the functional deficit (1). Among the aforementioned processes, the oxidative stress plays a significant role in pathophysiology of SCI. In this study, we evaluated the role of the melatonin, an indoleamine recognized as a potent antioxidant and immunomodulator (2, 3 )Reiter et al., 1995, Favero et al., 2015), on the oxidative stress, the tissue vitality and the neuritic plasticity in an experimental model of organotypic cultures of Sprague Dawley rat spinal cord slice (SPS) treated with hydrogen peroxide ($H_2O_2$) and/or melatonin. Five experimental protocols were performed: 1) control; 2) $H_2O_2$ exposure (50 μM); 3) melatonin treatment ($5^{-10}$M for 24 hours); 4) $H_2O_2$ exposure and post-treatment with melatonin; 5) $H_2O_2$ exposure after pre-treatment with melatonin. Cellular death was investigated by propidium iodide (PI) assay and the vitality by MTT assay. The total thiols (SH) levels, contrasting the oxidative stress, the neuronal specific nuclear protein (NeuN) and the synaptophysin (Syp) immunopositivity were also evaluated. Melatonin significantly decreases the number of dead cells and increases slice vitality, mainly in slices treated before $H_2O_2$ exposure. Moreover, melatonin attenuates total thiols decrease and NeuN and Syp immunopositivity reduction. Overall, these findings suggest that melatonin may exert a potential beneficial effect upon the progression of SCI secondary damage, protecting the tissue from a further degeneration.

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Keywords

Stress damage; melatonin; spinal cord organotypic model.