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Cytoprotective effects of melatonin in C2C12 skeletal muscle cells: a multiple technical approach

Valentina Baldassarri[1], Sara Salucci[1], Davide Curzi[1], Sabrina Burattini[1] and Elisabetta Falcieri[1,2]

[1] Department of Earth, Life and Environmental Sciences, University of Urbino "Carlo Bo", Urbino, Italy
[2] Institute of Molecular Genetics, CNR and Istituti Ortopedici Rizzoli, Bologna, Italy

Melatonin has a wide range of physiological functions including protection against oxidative stress, which is carried out through its ability to act as a free radical scavenger and to stimulate antioxidant enzyme production (Allegra et al., 2003). Oxidative stress is a major player in initiating apoptosis in skeletal muscle, as well as in other tissues. Apoptosis is essential for skeletal muscle development and homeostasis; nevertheless, its misregulation has been frequently observed in various myopathies (Loro et al., 2010). Several authors demonstrated that melatonin exerts anti-apoptotic actions in various cell models (Hibaoui et al., 2009) and our previous studies evidenced that it prevents apoptosis induced by UV-B and H_2O_2 in U937 cells (Luchetti et al., 2006; Salucci et al., 2010). In this work, melatonin activity has been investigated in C2C12 cells, after apoptotic chemical treatments. Myoblasts and myotubes were pre-treated with melatonin and then exposed to H_2O_2, cisplatin, etoposide and staurosporine. Data, obtained by means of TEM and TUNEL-CLSM, show that melatonin prevents apoptosis induced by H_2O_2, cisplatin and etoposide. Differently, staurosporine-induced apoptosis is not inhibited, probably because this trigger has a mechanism of action different from free radical increase. These results confirm melatonin ability to act as an antioxidant and anti-apoptotic molecule, thus suggesting a possible therapeutic strategy for myophaties involving apoptosis misregulation.

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