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How skeletal muscle cells die after chemical treatments

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Apoptosis deregulation is pathogenetic in several skeletal muscle disorders characterised by muscle mass loss, myofiber number decrease, apoptotic myonuclei increase and an elevated DNA fragmentation (Dupont-Versteegden, 2006). The aim of this work is to study in vitro, in C2C12 myoblasts and myotubes, the apoptotic behavior induced byetoposide, staurosporine and H_2O_2. Cell response was investigated by means of cytofluorimetric and morphological analyses, as well as by confocal microscopy of TUNEL reaction. Myotubes appeared more resistant than myoblasts to apoptotic induction in all experimental conditions. In particular, in myoblasts treated with etoposide and staurosporine apoptotic nuclei with chromatin margination and condensation were observed, in the presence of a diffuse DNA fragmentation evidenced after TUNEL reaction. The latter was observed also in myotubes, where apoptotic and normal nuclei inside the same syncytium appeared (D'Emilio et al., 2010). After H_2O_2 exposure, myotubes, differently from myoblasts, showed a poor cell sensitivity to cell death even if a certain DNA cleavage was observed. Intriguingly, autophagic granules diffusely appeared in myotubes after each treatment. In myoblasts all chemicals induced ROS increase. When their production exceeds cellular antioxidant capability, oxidative stress results and apoptosis is triggered. On the other hand, in myotubes mitochondria appeared better preserved than myoblasts, and, if damaged, they are probably degradated by autophagic processes. Finally, myotube resistance to apoptotic stimuli could be correlated to the fact that myogenic cells acquire an apoptosis-resistant phenotype during differentiation (Xiao et al., 2011).

References

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