IJAE
Vol. 120, n. 1 (Supplement): 140, 2015
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Autophagic behavior in skeletal muscle cells

Francesco Maria Giordano, Sabrina Burattini, Elisabetta Falcieri, Sara Salucci, Michela Battistelli

Department of Earth, Life and Enviromental Sciences, Urbino University Carlo Bo, Urbino, Italy

Autophagy represents a physiological mechanism responsible for cell homeostasis and its deregulation is involved in several conditions related to muscle mass loss such as aging, anorexia, inflammatory diseases, cancer, disuse and immobilization (1). In our previous work, double membrane vesicles, suggestive of autophagy, appeared after chemotherapeutic treatments in C2C12 myotubes (2). Here, to better understand the autophagic behavior, skeletal muscle cells have been exposed to cisplatin, etoposide and staurosporine and their effects have been investigated by means of morphological, cytofluorimetric and functional analyses. Ultrastructural observations evidenced the presence of autophagic vacuoles containing abnormal mitochondria, nuclear materials and membranes. Flow cytometry evaluation of lysosomal compartment stability revealed an autophagic pattern increase, particularly after cisplatin and staurosporine exposure, suggesting the presence of a cell death mechanism. Since autophagic deregulation is involved in different muscle diseases, further studies are in progress to evaluate possible strategies, such as protein ipo/iper supplementation, to prevent the abnormal autophagic activation induced by chemical triggers, which lead to muscle atrophy (3).

References

[1] Sandri et al. (2013) Misregulation of autophagy and protein degradation systems in myopathies and muscular dystrophies. J Cell Sci. 126(23):5325-33. doi: 10.1242/jcs.114041.
[2] Salucci et al. (2013) The peculiar apoptotic behavior of skeletal muscle cells. Histol Histopathol. 28(8):1073-87.
[3] Fanzani et al. (2011) Cisplatin triggers atrophy of skeletal C2C12 myotubes via impairment of Akt signalling pathway and subsequent increment activity of proteasome and autophagy systems. Toxicol Appl Pharmacol. 250(3):312-21. doi: 10.1016/j.taap.2010.11.003.

Keywords

C2C12 cells; chemical drugs; autophagy.