

Endurance training induces apoptosis in the tumor mass in the C26-bearing mouse model

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Cachexia, sarcopenia and anorexia are characterised by muscle wasting. This condition is a weakening, shrinking, and loss of muscle caused by a disease or lack of use. The loss of muscle causes a decrease in strength and inability to move compromising the quality of life. Recently we demonstrated that the skeletal muscle of endurance trained Balb/c mice release IL-6 and Hsp60 (inside exosomes) in the blood stream.

We studied the expression of Hsp60 in the muscles of trained and untrained C26-bearing mice, to understand if Hsp60 was over-expression may improve muscle performance and reduce cachexia. Four different interleukins have been also studied in cachectic mice, to understand which was their effect on Hsp60 expression both in the tumor mass and the trained muscle.

In the present study we demonstrated that: 1) IL-6 is released by the trained muscle; 2) IL-6 is release also by the tumor mass, 3) in animals inoculated with the C26 tumor and trained after inoculation, IL-6 is synthesized mainly by the skeletal muscle and the tumor mass undergo apoptosis.

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References

Barone et al. (2016) Skeletal muscle Heat shock protein 60 increases after endurance training and induces peroxisome proliferator-activated receptor gamma coactivator1 α 1 expression. *Sci Rep.* 6 :19781.

Key words

Colon carcinoma, metastasis, cachexia, Hsp60, interleukin-6.