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Resistance Exercise as a Therapeutic Strategy for Cancer-Related Sarcopenia: Molecular Insights from a Preclinical Model

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Background. Cancer-related sarcopenia is a unique form of muscle atrophy. No other type (immobilization, aging, microgravity, etc.) results in such profound impairment of patient prognosis. Considering that and given the absence of a curative treatment, we aimed to investigate the molecular basis of cancer-related sarcopenia and the role of exercise in its management.
Methods. Two experiments were conducted. In the first, colon cancer–related sarcopenia was induced using the AOM/DSS preclinical model. A skeletal muscle panel was performed to identify differentially expressed genes (DEGs) associated with sarcopenia. In the second experiment, cancer-related sarcopenic mice were assigned to a 5-week resistance exercise (RE). Untrained sarcopenic mice served as controls. The RE protocol consisted of ladder climbing with progressive loads attached to the animals'tails. Gene and protein expression were compared between sarcopenic and exercise-trained sarcopenic mice. In both experiments, sarcopenia was determined by the reductions in muscle strength and impaired locomotor capacity, along with myofiber atrophy, assessed by in vitro cross-sectional area.
Results. Volcano plot analysis identified 10 differentially expressed genes (DEGs) related to skeletal muscle fibrosis, protein degradation, and inflammation. Hierarchical clustering analysis revealed distinct expression patterns between sarcopenic and non-sarcopenic mice. Receiver operating characteristic (ROC) analysis demonstrated that Atrogin-1 and MuRF-1, two key components of the ubiquitin–proteasome system, have potential diagnostic value for sarcopenia. RE prevented skeletal muscle atrophy and impaired motor performance, which are hallmark features of sarcopenia. In addition to normalizing the expression of several inflammation- and fibrosis-related genes, RE also prevented the exercise-induced increases in muscle Atrogin-1 and MuRF-1 protein levels, key regulators of muscle atrophy.
Conclusion: RE prevents cancer-induced sarcopenia by downregulating Atrogin-1 and MuRF-1, key components of skeletal muscle proteolysis signaling. These findings demonstrate the potential of RE to treat cancer-related sarcopenia and detect key targets for sarcopenia-specific pharmacological development.

Keywords

muscle atrophy, proteolysis, exercise, mice

Conflict of Interest & Ethical Approval

yes

Abstract submitters declaration

yes

Author: DEMINICE, Rafael (State University of Londrina)

Co-authors: Mr ALEXANDRE ESTIGARRIBIA PRIANTI, Diogo (State University of Londrina); Mrs AZEVEDO DA SILVA GUIMARÃES, Tatiana (State University of Londrina); Mrs SANCHES CELLA, Paola (Universidade Estadual do Norte do Paraná)

Presenter: DEMINICE, Rafael (State University of Londrina)

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