TACSM Abstract

Effects of Continuous Aerobic Exercise on Skeletal Muscle Atrophy Induced by Heart Failure

CAROLINA R. TONON[1], BERTHA F. POLEGATO[1], QUINTEN PIGG[2], DILLON HARRIS[2], AARON GARCIA[2], DANIELA INOUE YOSHIMURA[2], & MARIANA JANINI GOMES[2]

[1]Department of Internal Medicine; Botucatu Medical School, Sao Paulo State University (Unesp); Botucatu, Brazil
[2]Department of Kinesiology and Sport Management; Texas A&M University; College Station, TX

Category: Doctoral

Advisor / Mentor: Janini Gomes, Mariana (m.janinigomes@tamu.edu)

ABSTRACT

Heart failure is a common and severe disease that worsens morbidity, mortality, and quality of life. One of the clinical manifestations of heart failure is peripheral muscular atrophy, whose presence impacts the prognosis of heart failure patients. Inflammation is one of the main mechanisms related to muscle atrophy. We hypothesized that inflammation, through excessive activation of the NLRP3 inflammasome, would be tightly associated with skeletal muscle atrophy induced by heart failure. Additionally, aerobic exercise, well known for its anti-inflammatory action, would counteract this condition in a rat model of heart failure. **PURPOSE**: We aimed to evaluate the effects of continuous aerobic exercise training on gene expression of Nlrp3 and atrogin-1, a key gene involved in skeletal muscle atrophy, in rats with heart failure induced by monocrotaline. **METHODS:** Thirty male Wistar rats were randomly divided into four groups: Control (C), Exercised (Ex), Heart Failure (HF) and HF exercised (HFEx). Initially, rats in groups HF and HFEx received a single dose of monocrotaline 60 mg/kg, intraperitonially, while groups C and Ex received a saline solution injection of equivalent volume. Two days after the injection, rats in Ex and HFEx groups were submitted to 30 minutes of treadmill running, 5 days/week, for 4 weeks, at moderate-intensity, determined by maximal endurance test. Groups C and HF were not subjected to any exercise program. After 4 weeks, rats were euthanized and the medial head of the gastrocnemius muscle was collected. Gene expression of atrogin-1 and Nlrp3 was performed by real time PCR. Two-way ANOVA was used for statistical analysis with significance level of $p<0.05$. **RESULTS:** The rats in HF group had lower gastrocnemius muscle mass than rats in the C group (C: 1.53 ±0.05; Ex: 1.37 ± 0.13; HF: 0.98 ± 0.21; HFEx:1.20 ± 0.19 g; pHF<0.001, pEx=0.680. pHFxEx=0.03). The decrease in muscle mass was combined with increased atrogin-1 gene expression in the HF groups (C: 0.54 ± 0.60; HF: 6.52 ± 5.76; Ex: 0.33 ± 0.11; HFEX: 2.59 ± 0.89 au; pHF<0.001, pEx=0.449, pHFxEx=0.807). We did not observe changes in gene expression of Nlrp3 between the groups (C: 0.38 ± 0.15; HF: 0.45 ± 0.21; Ex: 0.46 ± 0.37; HFEx: 0.29 ± 0.25 au; pHF=0.618, pEx=0.787, pHFxEx=0.293). **CONCLUSION**: Our data showed that monocrotaline-induced heart failure caused skeletal muscle atrophy in rats, in a mechanism independent of the NLRP3 inflammasome. A 4-week continuous aerobic exercise protocol provided partial protection from skeletal muscle atrophy.