TACSM Abstract

Effects of Endurance Training on Skeletal Muscle Mass and Inflammation in a Rat Model of Heart Failure

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ABSTRACT

Chronic disease states including heart failure (HF) often lead to reduced physical activity, resulting in disuse and low-level systemic inflammation, which contribute to the decline in muscle mass and function. An important goal of therapy in HF patients is to counteract or prevent the development of skeletal muscle alterations in order to restore a normal functional capacity. Exercise is an established means of improving exercise capacity and quality of life in patients with HF. Therefore, we aimed to evaluate the impact of an endurance program on skeletal muscle mass and inflammation in a rat model of pulmonary arterial hypertension (PAH)-induced heart failure. **Methods:** 30 male Wistar rats (~250g) were randomly divided into 4 groups: control untrained (CU); control trained (CT), PAH-HF untrained (HFU), and PAH-HF trained (HFT). PAH-HF was induced by a single dose (60 mg/kg) of monocrotaline (MCT), a pharmacological agent typically used to aid in replicating experimental models of right ventricle HF. Control groups received an equivalent volume of saline solution. Trained groups were subjected to a 4-week endurance training program, which consisted of running on a treadmill 5 days/week at 60% of maximal endurance capacity. The concentrations of inflammatory cytokines (TNF-α, interleukin(IL)-1β and IL-6) in the tibialis anterior muscle were detected using a multiplex assay. Statistical analysis: Two-way ANOVA and Tukey's post-hoc test. Data is expressed as mean ± SD with significance level set at 0.05. **Results:** Both HFU and HFT showed a reduction in body mass compared to their respective controls, and the endurance training was unable to mitigate the loss of body mass (CU: 416±31, CT: 392± 28, HFU: 344±40, HFT: 344±33g; $p < 0.05$). Similar to the body mass, we found a decrease in muscle mass in both HFU and HFT compared to their control groups (CU: .73±0.04, CT:0.73±0.06, HFU: 0.60±0.11, HFT: 0.61±0.10g $p < 0.05$). Looking at the muscle concentration of the inflammatory cytokines TNF-α, IL)-1β, and IL-6, we found no statical difference (TNF-a CU: 639±87, CT: 651±92, HFU: 587±145, HFT: 576±107; IL-1b CU: 1879±294, CT: 1654±508, HFU: 1616±205, HFT: 1607±375; IL-6 CU: 421±26, CT: 381±29, HFU: 393±17, HFT: 384±40pg/ml $p > 0.05$). **Conclusion:** Pulmonary arterial hypertension- induced heart failure led to a reduction in body weight and skeletal muscle mass without the development of local inflammation in rats, regardless of training status.