



### Effects of exercise on muscle atrophy in simulated weightless rats

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**Objective** Insufficient physical activity, aerospace weight loss, and fixed treatment of fractures, tendons, and neuropathy, or the resulting muscle atrophy caused by reduced exercise, have become an urgent health problem. Exercise has received widespread attention as an effective means of preventing muscle atrophy. Through the literatures related to "sports and muscle atrophy" at home and abroad, the effects of exercise on muscle atrophy in simulated weightless rats were analyzed, in order to provide theoretical basis and guidance for exercise prevention and treatment of muscle atrophy.

**Methods** The electronic databases PubMed, EMBASE and SPORTDiscus were searched for relevant studies reporting on the effects of physical exercise on muscle therapy. The keywords are: "aerobic exercise or resistance exercise"; "muscle atrophy"; "weightless or weightlessness" and the limitation period is 2013-2018. At the same time, the relevant literatures of the Chinese Journal Full-text Database were searched. The search terms were: "aerobic exercise; resistance exercise; muscle atrophy", and the language of the article was limited to Chinese. Inclusion criteria: exercise and sarcopenia; effects of exercise on muscle atrophy; relationship between muscle atrophy and exercise. Exclusion criteria: repetitive studies. A total of 54 literature reviews were included in the literature.

**Results (1)** The simulated weightless model is mainly for unloading and tail suspension. After the tail suspension, the soleus muscle becomes thinner and thinner, the elasticity decreases, the volume and mass decrease, the cross-sectional area of the muscle fiber decreases, and the cross-sectional area of the muscle fiber increases after exercise intervention, suggesting that exercise can effectively slow down the quality of the soleus muscle caused by simulated weightlessness.

(2) After 4 weeks of tail suspension, IGF-1 was found to change significantly. Exercise can stimulate the secretion of IGF-1, TESTO and myogenin to some extent, and promote the synthesis of muscle protein. At the same time, myogenin showed different expression under different exercise forms, suggesting myogenin. It may not be used as a predictor of muscle atrophy change.

(3) Oxidative stress occurs after 4 weeks of tail suspension, aggravating muscle atrophy, and activity of SOD and GSH-Px is enhanced after exercise intervention. The possible mechanism is to exercise the body SOD and GSH-Px. Vitality, thereby reducing muscle atrophy caused by oxidative stress in the body. (4) Exercise to reduce muscle atrophy may be through down-regulation of atrogen-1 expression and reduction of Caspase-3 expression.

(5) Regardless of endurance exercise or resistance exercise, long-term low-intensity exercise or high-intensity exercise can down-regulate atrogen-1 expression in skeletal muscle. However, different exercise intensity and different exercise patterns have irregular expression of atrogen-1. The effects of different exercise patterns and exercise intensity on the expression of atrogen-1 under simulated weightlessness and its specific mechanism need to be further explored.

**Conclusions** Exercise improves muscle atrophy by promoting the secretion of anabolic hormones in the body, increasing the antioxidant capacity in the body and inhibiting the expression of atrogen-1 protein in the ubiquitin proteasome pathway, which promotes the synthesis and inhibition of skeletal muscle protein to a certain extent. Decomposition of skeletal muscle proteins to reduce muscle atrophy caused by tail suspension.