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Resistance Training prevents Skeletal Muscle Atrophy Induced by hypoxia through regulating Akt-FoxO1 pathway

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Objective Skeletal muscle atrophy induced by hypoxia on the plateau will lead to the decrease of muscle strength and the degeneration of athletic ability. Resistance training is an efficient method to stimulate the growth of muscle and improve protein synthesis. Akt-FoxO1 (Fork head box protein 1) pathway plays a significant role in the regulation of skeletal muscle protein degradation. However, it is not clear whether resistance training could prevent skeletal muscle atrophy induced by hypoxia and what is the regulation role of Akt-FoxO1 pathway. This study built a rat model that resistance training inhibited the skeletal muscle atrophy induced by hypoxia and explore the variation of Akt, FoxO1, Murf and Atrogin-1.

Methods 40 male 8-week-old Sprague-Dawley (SD) rats were divided into 4 groups randomly: control group (C), resistance training group (R), hypoxia group (H) and hypoxia resistance training group (HR). H and HR group were placed into simulated 4000m altitude (12.4%, O₂%) and R and HR group received ladder resistance training. Their incremental load is calculated by using average body weight. After 4 weeks intervention of hypoxia and resistance training, body composition, wet weight of skeletal muscle (soleus, musculus gastrocnemius, extensor digitorum longus and musculus biceps brachii) and skeletal muscle cross-sectional area (CSA) were measured. The expression of Akt, FoxO1, Murf and Atrogin-1 were detected by Western blot and RT-PCR. Moreover, immunofluorescence technique was used to locate the phosphorylation of FoxO1.

Results The lean body mass of HR group was significantly higher than H group ($P < 0.05$). The wet weight and CSA of musculus biceps brachii in HR group were also higher than H group obviously ($P < 0.05$). The results of real-time fluorescence quantitative PCR and western blot showed that the expression of FoxO1 and MuRF of hypoxia group (H group) were significantly higher than control group. However after the intervention of resistance training, the expression of Akt was significantly up-regulate and FoxO1, MuRF were significantly down-regulate. Immunofluorescence technique was used to observe the location of FoxO1 phosphorylation and the expression out of nucleus.

Conclusions Resistance training contribute to prevent the occurrence of skeletal muscle atrophy induced by hypoxia and the form of climbing ladder training can stimulate the hypertrophy of biceps in rats. The results revealed that FoxO1 phosphorylation out of nucleus became higher after resistance training. All above revealed that resistance training could inhibit skeletal muscle atrophy induced by hypoxia. Akt promoted FoxO1 phosphorylation may become the molecular mechanisms that resistance training can inhibit the atrophy of skeletal muscle induced by hypoxia.