レジスタンストレーニングの継続に伴う筋肥大応答の減弱メカニズムの解明

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Mechanism for Attenuation of Muscle Hypertrophic Effect Per Exercise Sessions in Continuous Resistance Training

by

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ABSTRACT

Resistance exercise training is effective for muscle hypertrophy, but continuous training gradually attenuates the hypertrophic effect per exercise session. Inactivation of mechanistic target of rapamycin complex 1（mTORC1, which plays role in protein synthesis in skeletal muscle）after resistance exercise is involved in the mechanism. Meanwhile, mTORC1 accounts for not only muscle protein anabolic systems but also muscle protein catabolic systems. Here, we investigated the bouts dependent changes in the ubiquitin-proteasome system and autophagy-lysosome system related factors, which are main protein catabolic systems in skeletal muscle, after resistance exercise. Male Sprague-Dawley rats were resistance-exercised 10 bouts with interval of 48 hours between bouts. The resistance exercise consisted of 50 repetitions of maximal isometric contractions of the right gastrocnemius muscle, elicited by transcutaneous electrical

stimulation under anesthesia. Immediately（0h post-RE）and 3h（3h post-RE）after the 1st and 10th exercise bouts, muscle samples were collected. The left gastrocnemius muscles were served as internal control. At 0h post-RE, the expression of ubiquitinated proteins decreased despite the number of exercise bouts. At 3h post-RE, the expression of ubiquitinated proteins increased despite the number of exercise bouts. At 0h post-RE, the expression of LC3-II（an indicator of autophagosome formation）did not change at both number of exercise bouts. At 3h post-RE, the expression of LC3-II decreased despite the number of exercise bouts. These results suggest that the response of the ubiquitin-proteasome system and autophagy-lysosome system does not change in continuous resistance exercise training, and the changes in the response of protein catabolic systems is not involved in the blunting of muscle hypertrophic effect.

要　旨

レジスタンストレーニングは，骨格筋量を維持・増大させるが，トレーニングの継続に伴い，セッション当たりの筋肥大効果は減弱する．そのメカニズムとして，筋タンパク質合成に関わるmTOR複合体1（mTORC1）の運動応答の鈍化が関与するとされている．一方でmTORC1は筋タンパク質分解系にも関与するが，その応答変化は不明である．この点を明らかにするため，本研究では10週齢の雄性SDラットに対して48時間毎にレジスタンス運動を実施し，1Bout目と10Bout目の終了直後と3時間後に対象筋を摘出し，各種解析を行った．ユビキチン化タンパク質の発現量は，Bout数に関わらず運動直後に減少し，3時間後に増加していた．オートファゴソーム形成の指標であるLC3-IIの発現量は，Bout数に関わらず運動終了3時間後に減少していた．以上のことから，レジスタンストレーニングの継続に伴い筋タンパク質分解系の応答は顕著には変化せず，筋肥大応答の鈍化メカニズムに関与しない可能性が示唆された．