Department of Rehabilitation Medicine Division of Physical Fitness

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General Summary

The research of our division has been focused on skeletal muscle plasticity, neuroscience, and exercise physiology.

Research Activities

The availability of heat stress is reported in the regeneration of damaged skeletal muscle. We examined mechanisms underlying the regeneration-promoting effect of heat stress (warm bath of 42°C for 30 minutes). Heat stress before damage accelerated the regeneration of myofiber size 6 days after the damage. We found HSP72 expression and Akt activity tended to be higher 6 days after damage. Therefore, activation of Akt and higher expression levels of stress proteins may be associated with the promotion by heat stress of muscle fiber regeneration.

We studied muscle fiber atrophy and regeneration with hindlimb unloading in adulthood and old age. Muscle atrophy due to unloading was greater in old age than in adulthood. We found deranged intramuscular structures and inclusion bodies with unloading in type I-predominant regions in both age groups. Although we observed muscular fibers with vacuoles of tubular aggregates in type II-predominant regions, denaturation was less than in type I-predominant regions. Such degeneration was more marked in old age than in adulthood.

Myostatin, a member of the transforming growth factor β superfamily, is a negative regulator of myogenesis and muscle hypertrophy. We examined the effects of hindlimb unloading with or without intermittent reloading on soleus muscle mass and expression levels of myostatin protein in rat skeletal muscles. We found that myostatin protein levels increased with hindlimb unloading. Intermittent reloading restrained expression changes. We conclude that myostatin plays important roles in the regulation of muscle mass.

Numerous studies have examined changes in blood adipocytokine density after exercise and diet therapy, but less is known about the rebound period after weight reduction. We examined adipocyte size and blood adipocytokine density immediately after and 2 days after exercise and diet therapy in Otsuka Long-Evans Tokushima fatty rats. Our findings suggest that blood leptin density in the rebound period may change independently of fat volume or the adipocyte size.

Exercise that is prolonged or of high intensity or both suppresses immune responses. However, the relationship between exercise intensity and the suppression of lipopolysaccharide (LPS)-induced tumor necrosis factor (TNF)- α is not fully understood. We studied the effects of different exercise intensities on LPS-induced TNF- α production in rats. We concluded that the suppression of LPS-induced TNF- α production depends on exercise intensity in rats.

Publications

Kitamura H¹, Minato K¹, Kimura M², Yamauchi H, Yano H³ (¹Wayo Women's Univ, ²Keio Univ, ³Kawasaki Welfare Univ). Lipopolysaccharideinduced tumor necrosis factor (TNF)- α production depends on exercise intensity in rats. Jpn J Phys Fitness Sports Med 2009; **58**: 405–8.