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

Myostatin, a member of the transforming growth factor-β super family, is a negative regulator of myogenesis and muscle hypertrophy. Peroxisomal proliferator-activated receptor (PPAR) γ co-activator 1α (PGC-1α) regulates muscular endurance by a shift in fiber type to type I and by mitochondrial biogenesis. We examined the effects of hindlimb unloading with or without intermittent reloading on muscle mass, fiber type, and expression levels of myostatin and PGC1α protein in rat skeletal muscles. We found that the soleus and medial gastrocnemius muscles atrophied by 47% and 31%, respectively, after unloading for 3 weeks. Also, changes in myosin heavy chain isoform composition from type I to IIb occurred in both muscles. The atrophy and shifts to a faster type were ameliorated by resistance exercise. Myostatin expression increased, and PGC-1α expression decreased in both muscles with hindlimb unloading. The resistance exercise restrained these expression changes. We conclude that myostatin and PGC-1α play important roles in the regulation of muscle mass and function.

Receptor activator of nuclear factor kappaB (RANK) ligand (RANKL) is involved in the differentiation and maturity of osteoclasts by coupling with RANK expressed on osteoclasts. We examined the effect of postmenopausal exercise on bone metabolism, including RANKL expression, in ovariectomized rats. The mineral density of the tibia decreased 3 months after ovariectomy. In addition, alkaline phosphatase activity, tartaric acid-resistant acid phosphatase activity, and RANKL protein expression increased with ovariectomy. However, these ovariectomy-induced changes were inhibited by habitual running exercise. We conclude that habitual postmenopausal exercise maintains bone mass by suppressing bone resorption through down-regulation of RANKL expression.

The treatment of obesity is important as an early measure to prevent metabolic syndrome. Adiponectin is secreted from smaller adipocytes and improves insulin resistance. Some studies have found that the effect of weight reduction to increase blood levels of adiponectin is weaker with exercise therapy than with diet therapy. Therefore, we examined the effect of a difference in speed of weight reduction on adipocyte size and blood adiponectin levels in Otsuka Long-Evans Tokushima fatty rats, which have overeating-related obesity.

The results obtained suggest that an exercise program with a low rate of weight reduction

over a longer period is better for maintaining high blood levels of adiponectin than is a program with a high rate of weight reduction over a shorter period.