## 2-A-08 Changes in protein contents of PGC-1α and RIP140 and metabolic adaptation after three weeks of clenbuterol administration

Daisuke HOSHINO, Hideo HATTA

*Department of Life Sciences (Sports Sciences), The University of Tokyo*

**Purpose:** It is known that chronic administration of clenbuterol, a β2-adrenergic agonist, increases muscle mass and induces transition of slow-to-fast muscle fibers with decreases in activities of muscle mitochondrial enzymes. We hypothesized that the administration of clenbuterol would decrease PGC-1α and increase RIP 140 protein in skeletal muscle because these are potential regulators of expressions of proteins involved in oxidative metabolism. **Methods:** To evaluate our hypothesis, rats were randomly divided into control or clenbuterol group. Rats in the clenbuterol group were administrated clenbuterol (30mg/L) with the drinking water. **Results and Discussion:** After 3 weeks, COXIV protein and citrate synthase activity, mitochondrial markers, decreased in red and white TA muscle in the clenbuterol-treated rats. PGC-1α protein was decreased and RIP140 protein was increased in red TA muscle. At the same muscle, lactate dehydrogenase, a glycolytic enzyme, activity increased after the 3 weeks of clenbuterol administration. Therefore, the 3 weeks of clenbuterol administration impaired oxidative capacity namely COXIV protein and citrate synthase activity and enhanced glycolytic enzyme activity, accompanied with the decrease in PGC-1α and the increase in RIP140 protein. These results suggest that clenbuterol-induced the decrease in mitochondrial activity would be associated with changes in PGC-1α and RIP140 protein contents.

**Key words:** mitochondria, skeletal muscle, β2-adrenergic activation

## 2-A-09 Single oral taurine supplementation activates muscleglycogen resynthesis during period of recovery from exhaustive exercise.

Yumiko Takahashi, Daisuke Hoshino, and Hideo Hatta (The university of Tokyo)

The aim of this study was to investigate effects of oral taurine (2-aminoethanesulfonic acid) supplementation in ICR mice on recovery of muscle glycogen and other metabolic substrates following exhaustive exercise. We orally ingested taurine (0.5 mg/g BW [body weight]) or physiological saline (water) immediately after treadmill running at 25 m/min for 90 min. Metabolite concentrations in blood and tissues were measured at 1, 2 and 3 hour of recovery periods with free access to food. Blood glucose concentration ($p<0.001$) and tibialis anterior muscle and liver glycogen contents ($p<0.05$) were significantly lower than pre-exercise level. At 1 hour after treadmill running, the concentrationsof plasma free fatty acids were significantly higherin taurine group than in water group ($p<0.05$). Muscle and liver glycogen contents recovered to pre-experiment level without significant difference between treatments at 1 hour of exercise. At 2 hour after exercise, glycogen contents of both red and white tibialis anterior muscle were significantly higher in taurine group ($p<0.05$). Plasma insulin concentrations were similar in both groups at each time point. GLUT4 protein contents in plantaris muscle were similar in both groups at any time point. These results suggest that single taurine supplementation after prolonged exercise has an effect on skeletal muscle glycogen resynthesis during glycogen supercompensation phase without altering GLUT4 protein expression and plasma insulin concentration.