1-B-08 Contraction-induced change in subcellular localization of carnitine transporter (OCTN2)

Yasuro FURUICHI, Hisashi TAKAKURA, Tatsuya YAMADA, Kazumi MASUDA

Faculty of Human Sciences, Kanazawa University

Purpose: Since carnitine plays an important role on fat oxidation, influx of carnitine could be crucial for muscle metabolism. The OCTN2, a sodium-dependent solute carrier, is assumed to transport carnitine into skeletal muscle cell. We have found that contractile activity facilitated carnitine uptake in skeletal muscles. However, the regulation mechanism of carnitine transport during muscle contraction remains unclear. The purpose of the present study was to clarify whether OCTN2 translocates to the plasma membrane due to muscle contraction. Methods: Isometric tetanic muscle contractions were elicited on the left hindlimb muscles of male Wistar rats. Immediately after electrical stimulation, hindlimb muscles were fixed by perfusion of phosphate buffer with paraformaldehyde and the muscle cross-sections were analyzed immunohistochemically. We also extracted the sarcolemma-enriched fraction from deep portion of m. gastrocnemius by centrifugation and measured the amount of OCTN2 protein by western blot. Results: analysis Immunohistochemical demonstrated OCTN2 was mostly located in the sarcolemma and also in the intracellular region. Colocalization of OCTN2 and dystrophin was increased in contracting muscle compared with resting muscle. Western blots showed that the sarcolemmal OCTN2 was increased by muscle contraction. Conclusion: The present study revealed that muscle contraction induced an increase in the amount of OCTN2 in the sarcolemmal membrane by histochemical and biochemical analysis. We conclude that carnitine uptake into myocyte is subject to short-term regulation by muscle contraction and involves the translocation of OCTN2 from intracellular stores to the sarcolemma.

Key words: OCTN2, carnitine transport, muscle contraction

1-B-09 Functional overload induces increases in monocarboxylate transporter (MCT)1 and MCT4 in muscle

Yu Kitaoka¹, Masanao Machida¹, Hideo Hatta², Tohru Takemasa¹

¹University of Tsukuba, ²The University of Tokyo

Purpose: A number of studies have shown that changes in muscle contractile activity regulate the expression of monocarboxylate transporters (MCTs) in the skeletal muscle. The aim of this study was to investigate the effect of functional overload on MCT1 and MCT4 protein expression.

Methods: Plantaris muscles from male ICR mice (8-week-old) were functionally overloaded for 15 days by ablation of the synergistic muscles. Blood samples are collected for measuring plasma testosterone and lactate concentration. MCT1, MCT4 and AMPK protein expression was determined by Western blotting.

Results and Discussion: The body weight was not altered thorough the experimental period. The plantaris muscle weight increased after I day of overload (p<0.05). MCT1 and MCT4 protein expression increased after 12 days after functional overload (p<0.05). AMP-activated protein kinase (AMPK) phosphorylation status [phospho-AMPK (Thr172)/total AMPK | was elevated after 3-9 days of functional overload (p<0.05). Plasma testosterone concentration was elevated after 12 days of functional overload (p<0.05), while lactate concentration was not altered. Thus, the current study demonstrated that heavy mechanical loading induces increase in MCT1 and MCT4 protein expression in the muscles with increase in AMPK phosphorylation status and plasma testosterone concentration.

Key words: functional overload, lactate, MCT