beta-Actin shows limited mobility and is required only for supraphysiological insulin-stimulated glucose transport in young adult soleus muscle

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Abstract

Studies in skeletal muscle cell cultures suggest that the cortical actin cytoskeleton is a major requirement for insulin-stimulated glucose transport, implicating the beta-actin isoform, which in many cell types is the main actin isoform. However, it is not clear that beta-actin plays such a role in mature skeletal muscle. Neither dependency of glucose transport on beta-actin nor actin reorganization upon glucose transport have been tested in mature muscle. To investigate the role of beta-actin in fully differentiated muscle, we performed a detailed characterization of wild type and muscle-specific beta-actin knockout (KO) mice. The effects of the beta-actin KO were subtle; however, we confirmed the previously reported decline in running performance of beta-actin KO mice compared with wild type during repeated maximal running tests. We also found insulin-stimulated glucose transport into incubated muscles reduced in soleus but not in extensor digitorum longus muscle of young adult mice. Contraction-stimulated glucose transport trended toward the same pattern, but the glucose transport phenotype disappeared in soleus muscles from mature adult mice. No genotype-related differences were found in body composition or glucose tolerance or by indirect calorimetry measurements. To evaluate beta-actin mobility in mature muscle, we electroporated green fluorescent protein (GFP)-beta-actin into flexor digitorum brevis muscle fibers and measured fluorescence recovery after photobleaching. GFP-beta-actin showed limited unstimulated mobility and no changes after insulin stimulation. In conclusion, beta-actin is not required for glucose transport regulation in mature mouse muscle under the majority of the tested conditions. Thus, our work reveals fundamental differences in the role of the cortical beta-actin cytoskeleton in mature muscle compared with cell culture.

Palabras clave
Palabras clave de autor: actin cytoskeleton; beta-actin; glucose transport; insulin; skeletal muscle

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