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A potential role of stretch-activated channels in anabolic mechanotransduction in the atrophied rat soleus muscle

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Objective Prolonged immobilization or unloading of skeletal muscle causes muscle disuse atrophy, which is characterized by a reduction in muscle cross-sectional area and compromised contractile function. To date, the mechanisms of anabolic mechanotransduction in the atrophied mammalian skeletal muscle remain poorly understood. The aim of the present study was to assess a possible role of stretch-activated ion channels (SAC) in the propagation of a mechanical signal to anabolic signaling and protein synthesis (PS) in an isolated rat soleus muscle following mechanical unloading.
Methods The mechanical unloading was performed via hindlimb suspension (HS). Twenty-eight male Wistar rats weighing were randomly assigned to the following 4 groups (n=7/group): 1) vivarium control (C), 2) control + SAC inhibitor (gadolinium) (C+Gd^{3+}), 3) 7-day HS (HS), 4) 7-day HS + SAC inhibitor (HS+Gd^{3+}). Following unloading, an isolated rat soleus was placed in an organ culture medium and subjected to a bout of eccentric contractions (EC). Upon completion of the EC, muscles were collected for Western blot analyses to determine the content of the key anabolic markers. The rate of PS was measured by SUnSET technique.
Results EC-induced increase in PS was significantly less in the HS and HS+ Gd^{3+} groups vs. the C group. There was no statistically significant difference between the HS and HS+ Gd^{3+} groups in terms of EC-induced increase in muscle PS. A decrease in EC-induced phosphorylation of p70S6K, 4E-BP1, RPS6 and GSK-3beta in the 7-day unloaded soleus treated with SAC inhibitor did not differ from that of the 7-day unloaded soleus without SAC blockade. Thus, the inhibition of SAC with gadolinium did not lead to further decline in EC-induced phosphorylation of the key anabolic markers and muscle PS.
Conclusions The results of the study suggest that attenuation of mTORC1-signaling and PS in response to EC in unloaded soleus muscle may be associated with inactivation of SAC. The study was supported by the RFBR grant # 16-34-60055.