Exercise downregulates mTORC1 pathway through REDD1 expression in rat skeletal muscle

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An acute bout of exercise induces repression of protein synthesis in skeletal muscle due in part to reduced signaling through the mammalian target of rapamycin complex 1 (mTORC1). Previous studies have shown that upregulated expression of regulated in DNA damage and development (REDD) 1 and 2 is an important mechanism in the regulation of mTORC1 activity in response to a variety of stresses. This study investigated whether induction of REDD1/2 expression occurs in rat skeletal muscle in response to a burst of endurance exercise. Rats ran on a motor-driven treadmill at a speed of 28 $m \cdot min^{-1}$ for 90 min, and then the gastrocnemius muscle was removed and analyzed for phosphorylation of the eukaryotic initiation factor (eIF) 4E binding protein 1 (4E-BP1) and expression of REDD1/2. Exercise repressed the mTORC1-signaling pathway as shown by dephosphorylation of 4E-BP1. In addition, exercise induced the expression of REDD1 mRNA (~8-fold) and protein (~3-fold). Expression of REDD2 mRNA was not altered by exercise. These findings indicated that enhanced expression of REDD1 may be an important mechanism that could partially explain the downregulation of mTORC1 signaling, and subsequent inhibition of protein synthesis in skeletal muscle during exercise.