Promoting muscle regeneration in a mouse model of muscular dystrophy

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Duchenne muscular dystrophy (DMD) is a degenerative skeletal muscle disease caused by mutations in the protein dystrophin. Dystrophin functions to protect muscle cells from injury and loss of functional dystrophin results in break down and loss of muscle cells. A cellular signal relay system, known as a MAP kinase cascade, regulates the function of muscle stem cells, serving as a source of the new cells that are required for muscle regeneration.

In this issue of the *Journal of Clinical Investigation*, researchers led by Anton Bennett at Yale University identified the protein MKP-5 as a negative regulator of MAP kinase cascades in muscle stem cells. Loss of MKP-5 in a mouse model of DMD enhanced the development of new muscle cells (myogenesis) and helped prevent muscle degeneration.

These results identify MKP-5 as an important suppressor of myogenesis and suggest that therapeutics that inhibit MKP-5 could be useful in the treatment of degenerative muscle diseases.

More information: Improved regenerative myogenesis and muscular dystrophy in mice lacking MKP-5, *J Clin Invest*. doi:10.1172/JCI64375

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