New target may slow disease progression in Duchenne muscular dystrophy

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Duchenne muscular dystrophy is a chronic disease causing severe muscle degeneration that is ultimately fatal. As the disease progresses, muscle precursor cells lose the ability to create new musclar tissue, leading to faster muscle deterioration.

This month in the *JCI*, work led by Ming-Jer Tsai at Baylor College of Medicine has identified a protein involved in coordinating the function of muscle precursor cells. The protein, COUP-TFII, may be a promising target for treating progressive muscle wasting in Duchenne muscular dystrophy.

In a mouse model of Duchenne muscular dystrophy, abnormally high levels of COUP-TFII exacerbated symptoms of muscle deterioration and diminished recovery after muscle injuries. These symptoms were linked to decreases in muscle precursor cell function. In contrast, reducing COUP-TFII activity decreased muscle degeneration in the mouse model.

These results indicate that blocking COUP-TFII may have beneficial therapeutic effects for delaying the progression of Duchenne muscular dystrophy.

More information: Xin Xie et al, COUP-TFII regulates satellite cell function and muscular dystrophy, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI87414

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