Muscular dystrophy clue

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Patients with Duchenne muscular dystrophy (DMD) have a genetic mutation that causes incurable muscle degeneration. Research has shown that the immune system plays a complex, double-sided role in muscular dystrophy—promoting both muscle repair and muscle degradation.

In their recent article in *Frontiers in Pharmacology*, Jonathan H. Soslow, MD, MSCI, Sergey Ryzhov, MD, Ph.D., and colleagues studied blood samples from normal people and people with DMD, and found that the patients with DMD had more T cells expressing a surface protein called CD26 compared to normal patients.

Additionally, in the DMD patients, the level of CD26-expressing T cells correlated with increased muscle strength. This may be because CD26 can bind an enzyme called adenosine deaminase (ADA), which can increase the levels of inosine, an anti-inflammatory molecule, in muscle.

The researchers hypothesize that the increased CD26-expressing cells help slow down muscle degradation in DMD patients by bringing more ADA into muscles. Their work shines a light on potential new therapeutic targets for DMD patients.

More information: Jonathan H. Soslow et al. Increased Number of Circulating CD8/CD26 T Cells in the Blood of Duchenne Muscular Dystrophy Patients Is Associated with Augmented Binding of Adenosine Deaminase and Higher Muscular Strength Scores, *Frontiers in Pharmacology* (2017). DOI: 10.3389/fphar.2017.00914

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