High-strain tendons repair less frequently

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In a discovery that seems counterintuitive, a study appearing in the May 21st *Journal of Biological Chemistry* has found that tendons in high-stress and strain areas, like the Achilles tendon, actually repair themselves less frequently than low-stress tendons. This study sheds some light on the increased susceptibility of certain tendons to injury during aging.

Tendons, composed of collagen and other proteins, serve to connect muscle to bone and thus are vital for movement. Considering their strenuous activity, tendons need to be continually repairing collagen damage to avoid buildup of degraded proteins that could cause serious complications. Not all tendons are equal though; some tendons, like those in the hand, are primarily used to maintain proper limb placement while others, like the Achilles tendon in humans and the superficial digital flexor tendon (SDFT) in horses, have to bear a lot of weight and strain.

It would be expected that high-strain tendons would repair more frequently, yet Dr Helen Birch at University College London and colleagues examined protein turnover in the tendons of horses of various ages and found that the high-strain SDFT (located at the rear of the limb) repairs much less frequently than the low-strain common digital extensor tendon (CDET, located at the front of the limb). Birch and colleagues used an approach called amino acid racemization to measure protein age in the horse tendons. Amino acids are always incorporated into proteins in a specific orientation called the L-form, but afterwards can spontaneously convert into a mirror image called the D-form. Therefore, by measuring the ratio of L and D amino acids over time, one

can estimate the half-life of a protein.

Through this method, the researchers found that non-collagen proteins in tendon have an average half life of 2.2 years in SDFT and 3.5 years in CDET, which would be expected. However, SDFT collagen had a half-life of 198 years, compared to 34 years for CDET collagen. That means that every year, only 0.25% of the injury-prone collagen gets replaced in SDFT tissue. Over time, degraded protein and other mechanically-induced micro-damage could reduce the overall integrity of the tendon, which could lead to large-scale injuries. As to why the body would seemingly put its more important tendons at greater risk, the researchers suggest that it may be a trade off; too much repair may compromise the strength and stiffness of these tendons which are used heavily, so the body tries to preserve their structural integrity at the expense of increased injury risk later in life.

More information: "Aspartic Acid racemization and collagen degradation markers reveal an accumulation of damage in tendon collagen which is enhanced with aging" by Chavaunne T. Thorpe, Ian Streeter, Gina L. Pinchbeck, Allen E. Goodship, Peter D. Clegg and Helen L. Birch, *Journal of Biological Chemistry*

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