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BASIC// DISCOVERY

A KEY to PREVENTING TENDINOSIS

RESEARCHERS HAVE LONG KNOWN THAT WITH AGE, BLOOD supply to tendon cells decreases, leaving them starved of oxygen. But knowing why this occurs and why it causes tendons to fray with age can be critically important to creating treatments that eliminate the need for surgery for tendinosis, a painful orthopedic condition.

An intriguing NIH GEMSSTAR-funded study by **Rowena McBeath, MD, PhD**, assistant professor of orthopedic surgery, found that the paired reduction of oxygen supply and the signaling molecule Rac1 causes aged tendon cells to change shape and flexibility. The findings have broad implications for both injury prevention and tissue repair.

In Dr. McBeath's study, donated human tendon cells were grown in an environment mimicking the low-oxygen environment common in older people. Those cells changed shape and became round and more similar to tough cartilage-like cells; in addition, older tendon cells reduced their production of the signaling molecule Rac1, which helps govern cell shape movement, and growth. With reduced Rac1, the tendon cells began to change shape—but only in low-oxygen conditions.

Now, Dr. McBeath is exploring whether increased Rac1 production might enable tendon cells to keep their shape and thus prevent tendinosis and the associated pain. She and her team are also working in the opposite direction: studying the possibility of using decreased levels of oxygen and Rac1 to spur cells to become fibrocartilage. Success in that effort could ultimately give clinicians the ability to grow patient-specific tissues to replace damaged tissue in the knee, hip or spine—in the long term, potentially obviating the need for joint replacement surgery. Dr. McBeath's new work has also received NIH support through a five-year K76 Beeson Career Development Award. ■