Matrix loaded and unloaded: can tendons grow when exercised?

THIS QUESTION IS PRETTY NAIVE, in that we all know that during overall growth and development connective tissue structures such as bone, ligaments, cartilage, and tendons will enhance their size to match the overall growth of the body (6). Several animal studies have documented that intense loading will enlarge tendon structures (1, 3), but they all required long periods of time before adaptation was observed and were done in animals that were constantly growing or were in their “childhood/adolescence.” In adult humans, it has been much harder to document any increase in tendon structures, and whereas cross-sectional studies predominantly have shown differences in tendon size in men (10), longitudinal studies have only been able to demonstrate moderate increases in tendon cross-sectional areas of the patella tendon (11) and not of the Achilles tendon (4). Because it is shown that acute exercise increases collagen synthesis (8), it suggests that collagen degradation increases simultaneously and/or that increased collagen turnover does not result in any major new formation of assembled nonsoluble collagen in tendon structures.

The study in this issue of the Journal of Applied Physiology by Legerlotz et al. (7) attempts to study the influence of different training types and vibration on tendon structures in animals. One of the strengths in the present study is its comprehensiveness in regard to determining both tendon morphological, biomechanical, and biochemical parameters simultaneously in a setup with many exercise perturbation. The combination of evaluating changes in protein expression and protein content of growth-modifying factors, together with the determination of mechanical properties of the tendon tissue, is important for the full understanding of tendon tissue adaptation. The study is not without limitations, in that the training protocols did not always reflect what humans would follow in their training regimens. Furthermore, the overall question one can be left with after this study is whether the so-called control group of animals was actually “forced” from training and thus represents a relative inactive group (2). If so, this would fit with the view that training as such only has small impact on tendon structures, whereas inactivity has a collagen synthesis inhibiting effect and thus that is what we see. If so, maybe tendons have the size they should have in adults, and what is needed is continuous activity throughout life to keep the tendons fit. Furthermore, the lesson to be learned is that there are many more aspects of connective tissue biology than the outer diameter of the tendon (5, 9) and that we are still far from understanding how mechanical loading is sensed by tendon structures and subsequently converted into chemical signaling and matrix protein formation.

REFERENCES


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