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Tendon physiology and pathophysiology in exercise and physical training

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Human tendon consists predominantly of collagen that has a general dynamic turnover rate during growth, but after adolescence (~17 yrs) only a minor fraction of this is turned over regularly (around 5%) and most fibril and fascicular structures (around 95%) remain stable. Collagen synthesis can be stimulated by physical exercise as well as by growth hormone or insulin-like growth factor I, and conversely collagen synthesis is inhibited by physical inactivity (e.g. 14 days). The drop in collagen synthesis with inactivity cannot be counteracted by blocking inflammation.
The dynamic part of the tendon matrix seems to be under circadian control, and with mechanical overloading, an accumulation of water - predominantly between fascicles - is observed and this contributes to tendon swelling in tendinopathy. Along with this, heavy mechanical loading and overloading of tendon results in increased tendon blood flow, as well as in increased pain and nociceptive signaling over the first months with subjective symptoms. In the later and more chronic phase of tendinopathy, stimulation of collagen synthesis and new matrix formation is observed leading to structural changes of the matrix in tendon.
The regulation of tendon matrix quality control is poorly understood, but evidence that autophagy regulates tendon homeostasis by controlling procollagen is documented. Further it has been demonstrated that cell-cell communication in tendon tissue is facilitated by extracellular vesicles - including exosomes - that are released by tendon fibroblasts, and that this control of communication is under circadian control. Further, exosomes derived from tendon fibroblasts differ markedly in protein content from those derived from either myoblasts or muscle fibroblasts. This suggests a role for tendon fibroblast exosomes in tendon fibrosis.
Resistance training of a heavy or moderate nature is documented to be the preferable treatment towards tendinopathy, but exactly what molecular and cellular mechanisms are behind this is not described, but it is observed that individuals with chronic tendinopathy have had an increased matrix turnover for several years prior to the current tendinopathy, and this remains to be explained.

Topic: **Sports Medicine and Orthopedics**

Presentation **Invited**