

Effect of aging on extracellular matrix and collagen turnover related pathways in human tendons and cultured human tenocytes

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The aging process involves different organs, including the musculoskeletal system. Age-related modifications affecting the tendon such as reduced cell proliferation and decreased glycosaminoglycan and proteoglycan content were previously described, but data are incomplete and discordant. Therefore, aim of our study was to characterize the effect of aging on tendons, with particular attention to collagen turnover. For this purpose, tendons and cultured tenocytes were obtained by healthy young (age <65 years) and aging subjects (≥65 years), and analyzed by morphological and molecular methods.

Our data show that aging tenocytes have a reduced proliferation rate. Haematoxylin and eosin, Sirius red and Alcian blue staining, respectively, revealed that tendon structure is maintained, and that collagen and proteoglycan content is similar in young and aging tendons. However, decreased lysyl hydroxylase 2b gene expression was observed in aging tenocytes, suggesting that differences in collagen maturation could be responsible for a decreased ability to resist mechanical loading.

By fluorescence microscopy, actin cytoskeleton modifications such as fewer and shorter stress fibers were observed in some aging tenocytes, consistent with a decreased ability to form focal adhesions and, therefore, a reduced migration potential. Intermediate filaments and microtubules were not modified by aging.

Considered as a whole, our results suggest that the structure of aged tendons is preserved, but the biomechanical properties could be impaired by reduced collagen cross-linking. Moreover, modifications of actin filaments could affect the mechanotransduction system allowing tenocytes to adapt their ability for extracellular matrix remodeling in response to mechanical loading. Therefore, aged tendons could be likely more prone to develop tendinopathies.

Keywords

Tendon; aging.