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Trigger finger, tendinosis, and intratendinous gene expression.

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Abstract

The pathogenesis of trigger finger has generally been ascribed to primary changes in the first annular ligament. In contrast, we recently found histological changes in the tendons, similar to the findings in Achilles tendinosis or tendinopathy. We therefore hypothesized that trigger finger tendons would show differences in gene expression in comparison to normal tendons in a pattern similar to what is published for Achilles tendinosis. We performed quantitative real-time polymerase chain reaction on biopsies from finger flexor tendons, 13 trigger fingers and 13 apparently healthy control tendons, to assess the expression of 10 genes which have been described to be differently expressed in tendinosis (collagen type 1a1, collagen 3a1, MMP-2, MMP-3, ADAMTS-5, TIMP-3, aggrecan, biglycan, decorin, and versican). In trigger finger tendons, collagen types 1a1 and 3a1, aggrecan and biglycan were all up-regulated, and MMP-3and TIMP-3 were down-regulated. These changes were statistically significant and have been previously described for Achilles tendinosis. The remaining four genes were not significantly altered. The changes in gene expression support the hypothesis that trigger finger is a form of tendinosis. Because trigger finger is a common condition, often treated surgically, it could provide opportunities for clinical research on tendinosis.

KEYWORDS: qPCR; quantitative real-time PCR; stenosing tendovaginitis; tendinopathy; tendinosis; tendovaginitis stenosans

PMID: 22882155 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

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