

## Is There Evidence for Fascial Adhesions Caused by Crosslinks?

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**BACKGROUND:** In the Fascial Distortion Model (FDM) of Typaldos D.O., one of the described fascial distortions goes along with the formation of adhesions caused by mal attached cross-links in the banded fascia of tendons [1].

**LITERATURE REVIEW:** Stability of collagen is guaranteed by the development of hydrogen bridges and covalent bonds called cross-links. With aging non specific cross-links occur by accumulation of glucose [2]. A reaction between amino acids and deoxidized sugar forms a compound (Maillard reaction) which leads to an accumulation of “advanced glycation end products” (AGEs) [3]. Through aging and/or a higher level of glucose (e.g. diabetes) unspecific cross-links accumulate and the AGEs increase in the tissue [4, 5, 6]. Tendons fortified by this complex of sugar and amino acid can take a higher load but on the other hand are stiffer [5]. A characteristic of AGEs type cross-links is a decreased solubility and a higher resistance to proteolysis [7, 8]. Oxidative stress advances the formation of AGE pentosid cross-links [6, 9]. The AGEs thereby induce protein cross-links, in turn reducing elasticity. [10]. When a tendon is stretched its elongation is greater than the lengthening of its single collagen filaments, due to a gliding between the filaments [11]. This sliding movement is hampered by non-specific cross-links. By repeated, unaccustomed or excessive force the fibrils tear. In horses, fibrils with a smaller diameter were found in the tendon after prolonged loading. This causes a partial looseness of the fibril bond [12]. It seems to be proven that after smaller mechanical injuries the healing of sinew is accompanied by a higher level of collagen type III synthesis. The new fibrils are significantly thinner and accordingly more prone to rupture. The proceedings leading to this collagenic build-up at the ruptured area seem to be of much older origin than the acute tearing [11]. Riley observed the increase of collagen type III in cases of tendonitis of the supraspinatus sinew [13]. Bank et al conclude that the ECM, observed in cases of tendonitis of the supraspinatus sinew, is the result of an uncontrolled healing process in which a carefully built and highly functional matrix of the sinew is replaced by lesser and unorganized tissue [2].

**RESULT:** There is evidence for the existence of adhesions caused by cross-links in the connective tissue.

**DISCUSSION:** Although it seems likely that the collagen changes described in cases of AGEs increase and tendonitis can lead to alterations in the ECM matrix of the fascia it will need further studies to prove their existence in the fascia.

**CONCLUSION:** There is evidence for an increase of non specific cross-links in the connective tissue through an accumulation of AGEs. As it is known that they reduce elasticity, it is likely that they build up adhesions in the fascia.

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