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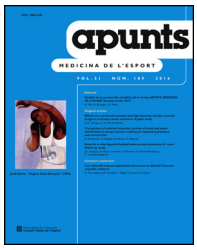

REVIEW

Potential risk factors leading to tendinopathy

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Abstract Tendinopathy has a multifactorial etiology that is not well understood. Risk factors are often separated into extrinsic (those acting on the body) and intrinsic groups (those acting from within the body). In this narrative review, we will separate potential risk factors into 1) load-related (extrinsic); 2) biomechanical factors (intrinsic); and 3) other individual factors such as systemic factors (intrinsic). Too much load is clearly linked to tendinopathy, but there appears to be large variation in how much load individuals can endure before developing tendinopathy. Less active people also suffer tendinopathy, suggesting that the effect of load is likely to be moderated by intrinsic factors. These individual intrinsic factors are likely to reduce tolerance or capacity to withstand load. This narrative review will provide a brief overview of key potential risk factors and mechanisms, as well as limitations in the current literature.

PALABRAS CLAVE
Tendinopatía;
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Biomecánica;
Prevención

Factores potenciales de riesgo que conducen a la tendinopatía

Resumen La tendinopatía tiene una etiología multifactorial que no es bien conocida. Los factores de riesgo a menudo se dividen en extrínsecos (los que actúan sobre el cuerpo) e intrínsecos (los que actúan desde dentro del cuerpo). En esta revisión descriptiva clasificamos los factores de riesgo potenciales en: 1) factores relacionados con la carga (extrínsecos); 2) factores biomecánicos (intrínsecos), y 3) otros factores individuales, como los factores sistémicos (intrínsecos). Una carga excesiva está claramente relacionada con la tendinopatía, pero parece que existe una gran diferencia en la cantidad de carga que los individuos pueden soportar antes de desarrollar una tendinopatía. Las personas menos activas también sufren tendinopatía, lo que sugiere que es probable que el efecto de la carga esté mediado por factores intrínsecos. Es

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probable que estos factores intrínsecos individuales reduzcan la tolerancia o la capacidad de soportar la carga. Esta revisión descriptiva ofrece un breve resumen de los principales factores de riesgo y mecanismos, así como las limitaciones en la literatura actual.

Load

Stretch shorten cycle loads

In the lower limb, repetitive stretch-shortening cycles (SSC) of the muscle-tendon unit (e.g. walking and running for Achilles tendinopathy or jumping for patellar tendinopathy)[1,2] is associated with tendinopathy. During SSC there is energy storage through elastic lengthening of the tendon and subsequent release of some of the stored energy to reduce the energy cost of locomotion.[3,4] In the Achilles-calf there is energy storage in terminal midstance and subsequently this energy contributes to positive work in toe off of walking and running.[2]

The magnitude of tendon load can be quite high during SSC activities. For example, Achilles tendon load is reported to be 6–8 times bodyweight in running[5,6] and as high as 8–10 times bodyweight in submaximal hopping.[7] The magnitude of tendon load is often lower with slow and heavy contractions that are commonly used in rehabilitation (e.g. heavy slow resistance training or eccentric training). Even during a maximal isometric planterflexion contraction load is about of a third to half (3.5 times bodyweight) of that during hopping.[8] Although it seems that higher load is not always a distinguishing feature between SSC and slow and heavy loads. Patellar tendon load during loaded squatting (4.78 bodyweights) is similar to load during a stop jump such as a spike jump take off (5.17 bodyweights).[9,10] However, the tendon strain rate is much lower during the squatting task (1–2 bodyweights/second) than during the stop jump task (almost 40 bodyweights per second). The tendon strain rate may explain why tendinopathy is associated with repetitive SSC rather than slow and heavy loads.[11]

Compression

Compression has been suggested to play a part in most insertional tendinopathies, or enthesopathies.[12] Benjamin et al.[13] described a specialized tendon–bone junction, 'the enthesis organ', that functions to reduce load concentration at the enthesis. A bony prominence, and at some sites bursa adjacent to the enthesis, have a role in absorbing and dispersing enthesis loads thereby limiting stress concentration at the tendon–bone junction. Tendons adapt to the increased compressive loads at the enthesis with increased fibrocartilage, larger water-binding proteoglycans and type II collagen.[14] Ground substance accumulation is a feature of insertional tendinopathy which has been suggested to be a response to compressive load.[12] The pathology has been localized to the side of the tendon adjacent to the bone, which also suggests compressive loads may be implicated.[14]

Soslowsky et al.[15] investigated the effect of compressive and tensile loads in isolation or combination (downhill running) on rat suprasinatus tendons. Their conclusions were that a compressive load alone did not lead to reduced mechanical properties, but a combination of compressive and tensile load was more damaging than tensile load alone. For insertional tendinopathies reducing enthesis compression is suggested to be an important aspect of prevention and management.[12] For example, in the case of the Achilles this can be achieved by using a heel wedge/lift.

Intratendinous loading patterns

Tensile stress may not be uniform throughout a tendon. Studies investigating cadavers,[16] optic fibers in vivo[17] and mathematical modeling[18] have found greater tensile strain in the posterior compared with the anterior side of the patellar tendon. This contrasts to Almekinders et al.[19] who found reduced strain in the posterior tendon in a cadaver study. Despite inconsistencies, these studies demonstrate different strain gradients or 'stress shielding' of part of the tendon may have a role in the development of patellar tendinopathy.[20] Similarly, in the Achilles tendon, there is emerging evidence from imaging studies using speckle tracking that the tendon does not strain uniformly under load.[21] The implications for the development of tendinopathy and rehabilitation (e.g. specific loading for certain parts of the tendon) are so far unknown.

Change in load

The most common cause for tendinopathy is described as ''training errors''. This is an ambiguous term, but is normally considered to encompass any alterations in physical load on the tendon.[22] Primarily, this involves fluctuations in intensity, frequency or duration of exercise, although it may be components of all three. Returning to training after a short break, e.g. after a holiday, is an example of a training error involving sudden change in load. The break in exercise is thought to lead to deconditioning, resulting in tendinopathy on a return to normal load. This is often evidenced in patients who misguidedly go through periods of rest to settle the tendon and then return to normal loading immediately, inevitably re-triggering symptoms.

Load management has recently been extensively investigated by Gabbett and colleagues.[23] They have demonstrated a relationship between chronic workload rates (exercise levels over the proceeding 4 week period) and the acute rates (that week, although, to date, this ratio has not been extensively investigated in relation to tendinopathy).[24] Clinically, it is important to ask about change in energy storage type