Results At 10% of MVC, CSAs were significantly smaller throughout the gastrocnemius tendon compared to those at rest, but there were no significant differences in the Achilles tendon CSAs (Figure 1A). At 20% and 30% of MVC, the gastrocnemius tendon CSAs remained significantly smaller throughout its length (Figure 1B and C). At 20% of MVC only the most proximal CSA of the Achilles tendon was significantly smaller compared to that at rest (Figure 1B). At 30% of MVC, CSAs in the proximal region (5 scans) of the Achilles tendon were significantly smaller compared to those at rest (Figure 1C).

Discussion Reductions in tendon CSA as a result of tensile loading applied by muscle contraction were assumed to represent regional-specific longitudinal elongations, i.e. as the tendon is stretched it becomes thinner. Contrary to our initial hypothesis, the largest deformations did not occur in the region of smallest tendon CSA, but were instead tendon-specific with greater deformations in the gastrocnemius compared to the Achilles tendon. This occurred despite presumably lower forces acting on the gastrocnemius tendon, which suggests a reduced stiffness and modulus in the gastrocnemius tendon compared to the Achilles.

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87 PATELLAR TENDINOPATHY: LOOKING OUTSIDE THE TENDON…

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Introduction Musculoskeletal pain (MSK) is known to cause neuroplasticity and changes to muscle representation and motor control.[1] Patellar tendinopathy (PT) is a MSK pain that can persist for many years and causes dysfunction in the patellar tendon. Local tissue changes fail to explain tendon pain because pathology may or may not cause pain. Importantly, it is not known how the central nervous system modulates or interprets tendon pain and whether it is similar to other chronic/persistent pain conditions.

Corticospinal excitability is an important determinant for muscle function and differences between healthy and tendinopathic participants may correlate with altered muscle function associated with tendinopathy. The corticospinal changes in people with tendon pathology and no pain may contribute to our understanding of the "chicken or the egg" in pathology and pain. This study investigated whether there were corticospinal excitability differences between people with and without tendon pain and tendon pathology.

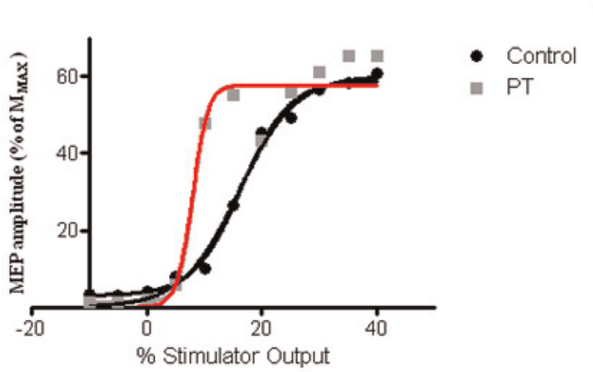
Methods Thirty jumping athletes (n = 6 women, n = 24 men) aged over 18 years with and without PT participated in the study. PT was diagnosed clinically. Ultrasound was used to assess for the presence of tendon abnormality.

Objective measures of SLDS pain (numerical rating score out of ten) and maximal voluntary isometric leg extension torque were recorded. The VISA-P questionnaire was administered. Single pulse transcranial magnetic stimulation (TMS) was applied over the motor area projecting to the quadriceps muscle group to obtain measures of corticospinal excitability. Surface EMG was placed on the rectus femoris muscle to record the motor evoke potentials (MEP).

Stimulus-response curves were obtained and the slope and peak values were used to establish the strength of projection. Results of tendinopathic and healthy individuals were compared. Within participant analysis was conducted to investigate the cortical changes associated with either unilateral or bilateral pain. People with tendon pathology were analysed separately to controls to determine the effect of pathology on corticospinal excitability.

Results Pilot data only (n = 11) is provided at this time with a full data set currently being analysed (n = 30). The mean VISA-P was 97.5 for healthy (n = 4) and 57.75 for people with PT (n = 7). The mean SLDS was 7.5/10 for people with PT.

PT increased cortical excitability, evidenced by a steeper slope in the stimulus response curve of people with PT compared with healthy people (p = 0.029, Cohen's d = 3.08). Figure 1 shows an example of a control participant and a participant with PT.



Abstract 87 Figure 1 Stimulus response curves shown for a control participant and a participant with pt. the y axis is the motor evoked potential as a percentage of Mmax obtained by direct stimulation of the femoral nerve

The x axis is the percentage stimulator output.

These changes were positively related to symptom duration (r^2 = 0.57) – the longer the duration of symptoms, the more pronounced the increase in cortical excitability.

Discussion People with PT have increased corticospinal excitability affecting motor control. Differences identified indicate that symptom duration impacts cortical excitability. It may be important to measure and consider these cortical changes in the rehabilitation of people with PT.

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88 EXERCISE REDUCES PAIN IMMEDIATELY AND AFFECTS CORTICAL INHIBITION IN PATELLAR TENDINOPATHY

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Introduction Patellar tendinopathy (PT) affects the ability to jump and land due to pain and associated corticospinal changes to motor patterning.

Whilst eccentric exercise is commonly used in rehabilitation, it can be painful to complete.[1] Tendinopathy is especially problematic in competitive season, during which there are constant time and performance pressures.[2] Where eccentric exercise has been completed in the competitive season, there has been poor adherence due to pain and either no benefit [Visnes, 2005] or worse outcomes [Fredberg, 2008]. There is a need for interventions that reduce pain

87 Patellar Tendinopathy: Looking Outside The Tendon ...

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Notes