Supplementary file

Method

Preliminary data contained in this supplementary file were collected using the method described below. Participants were recruited from sub-elite and elite basketball and volleyball populations and were aged over 18 years and all playing / training three times per week. If they were not able to participate in games and trainings three times per week for any reason including PT, they were not included because of the potential for activity to modify the primary motor cortex (M1) and motor control as demonstrated in pilot testing.

While habitual activity has been shown to effect the M1, no study was identified that specifically considered the type of activity and the influence on the M1 and motor drive. Thirteen physically active, healthy participants were recruited (Table 1.) All participants completed at least three sessions per week of structured activity and included a mix of elite, sub-elite and recreational athletes. Athletes nominated their dominant leg for testing and the contralateral corticospinal excitability was tested

Table 1 Characteristics of participants in the pilot study that investigated the influence of jumping on corticospinal excitability

Characteristics	Description
N (men)	13 (9)
Age years (median+range)	26 (21-37)

BMI (mean±SD)	23.87±2.60
Weekly activity	Australian football, swimming, touch football, running, martial arts, volleyball and badminton

BMI, body mass index

Following testing, participants were grouped according to activity type (jumping yes/no). Data were coded and all data were analysed blinded to group activity type (Table 2). Stimulus response curves were constructed (normalised to M_{MAX}) and the slope was calculated using GraphPad Prism. Data for M_{MAX}, active motor threshold (AMT) and maximal voluntary isometric contraction (MVIC) are presented as median and range. In order to be conservative, non-parametric analyses (Mann Whitney U) were conducted on all comparisons, as the sample size was small and groups were an uneven number. Physically active people who did not participate in sports that have a jumping requirement exhibited lower corticospinal excitability, evidenced by a decrease in the slope of the stimulus response curve (higher number indicates decrease in slope) (Table 2).

Table 2 Corticospinal responses, peripheral measures and descriptions of each group

Group by activity (jumping yes / no)	Characteristics	M_{MAX}	AMT	MVIC	Slope (AU)
N=8 jumping participants (median+range)	6 men Australian football Martial arts Volleyball Badminton	24.61 (21.84-27.68)	42 (31-50)	172.5 (152-274)	5.91 (4.08-7.90)
N=5 non-jumping participants (median+range)	3 men Swimming Touch football Running	21.51 (18.7-27.46)	35 (31-50)	168 (105-183)	14.04* (9.66-17.69)

AMT, active motor threshold; MVIC, Maximum voluntary isometric contraction; AU, arbitrary units * denotes p=0.002

These data suggest that jumping has a profound effect on the CSE and that it is therefore important that any comparisons made between controls, those with other AKP or PT participate in jumping sports. Data were checked for the potential effect of gender and found to be non-significant ($p = 0.26$). There were no differences between groups for M_{MAX}, AMT or MVIC ($p < 0.05$).

Based upon these data, the decision was made to recruit both men and women but only include athletes that played / trained at least three times per week in jumping sports in all studies.

Note that the larger randomised clinical trial (RCT) (Rio et al., unpublished data and van Ark et al., unpublished data) included participants aged over 16 years however, only participants aged over 18 years were offered transcranial magnetic stimulation (TMS) testing.

Athletes were asked to complete a VISA-P, a questionnaire about patellar tendon pain and function that is scored between 0 and 100 with 100 being maximal pain free function [1]. Height in centimetres (cm) using a stadiometer and weight in kilograms (kg) without foot ware were recorded and this has been described previously [2].

Clinical diagnosis of patellar tendinopathy

Patellar tendinopathy (PT) in all studies was diagnosed as localised pain at the inferior pole of the patella during jumping and landing as well as during the single leg decline squat (SLDS), a reliable patellar tendon pain provocation test [3]. Grey scale ultrasound (US) was used to confirm the diagnosis by applying the following where at least one criterion had to be satisfied; presence of a hypoechoic area, increased thickness of the anterior/posterior diameter greater than six millimetres (mm) or the presence of vessels. Athletes with bilateral symptoms were asked to nominate their most painful knee on the SLDS using a numerical rating scale (NMR) 0-10 and measures of quadriceps torque were taken from this side only and the contralateral hemisphere was tested with TMS.

Inter-person data

Jumping athletes were recruited and offered bilateral TMS testing. Following testing, they were assessed clinically and using US to determine the presence of tendon pain (NRS pain on SLDS) and tendon pathology (US) for sub-grouping into unilateral or bilateral tendon pain or pathology.

In-season intervention study - Tendon neuroplastic training

Concealed randomisation was achieved by asking the athlete to draw an opaque sealed envelope with no external markings [4]. The envelope contained a number (either one = isometric or two = isotonic) produced by random number generation (Excel 2007©).

Data were collected as part of a RCT with two intervention arms completed over four weeks during a competitive season. The intervention was completed four times per week. There were two active intervention arms, either isometric or isotonic muscle contractions and no sham group. Exercises were completed on a leg extension machine (Figure 1).

Figure 1. An example of a leg extension machine used for the intervention of quadriceps muscle contractions

Both protocols were matched for time under load and rest between sets (set at two minutes to allow muscle recovery) (Table 3) [5]. Repetition maximum (for the isotonic group) and MVIC (for the isometric group) were determined for starting loads. As muscle work during isometric exercise and isotonic exercise cannot be directly measured, protocols were matched for rating of perceived exertion on the basis of pilot studies and to avoid delayed onset muscle soreness as this was an in season study and muscle pain may negatively affect compliance or sporting participation. Furthermore, the protocol were matched for time under tension and based upon data supporting the use of external pacing to modulate corticospinal excitability and inhibition.

Auditory cues have been shown to be beneficial on the induction of neuroplasticity [6-11]. Therefore both groups received an auditory file to play on their smart phone device during their exercise sessions that provided verbal instructions and paced the muscle contractions (Table 3). The aims of this were to try to ensure timing was adhered to, provide auditory stimulation and avoid self-pacing so that the only difference between groups was muscle contraction type.

Table 3. Protocol used in the randomised clinical trial

	Isometric group	Isotonic group
Time under tension	5 x 45 seconds = 225 seconds	4 x 8 3 seconds concentric and 4 seconds eccentric phase = 224 seconds
Starting load	80% MVIC	80%8RM
Progression	2.5% as able	2.5% as able
External pacing and cues	Metronome and verbal instructions	Metronome and verbal instructions
Equipment	Leg extension machine	Leg extension machine
Range	60 degrees	Comfortable range 10 degrees – 100 degrees

Note: the protocol was modified from [2] to prevent delayed onset muscle soreness.

Transcranial magnetic stimulation

Single pulse TMS was used to obtain stimulus response curves and paired pulse TMS technique was used to quantify SICI and these methods have been published elsewhere [2].

Outcome measures and statistical tests

For the inter-person study, outcome measures were active motor threshold, CSE and SICI. For the RCT, outcome measures were short interval cortical inhibition (SICI) and VISA-P. Non-parametric tests were chosen due to small sample size.

Results of the inter-person study

Bilateral data was obtained for 16 athletes (32 hemispheres). Of these, four were control participants recruited to compare SICI against the published physiological normal range for the quadriceps (50-70%) [6, 12] as it was unknown if SICI may be altered in jumping athletes. The median SICI ratio for both sides of control athletes was 56.81 (range 50.86-58.81) and there were no differences between hemispheres in control participants ($p=0.49$). There were no differences between sides in control participants for the slope of the stimulus response curve, left = 7.28, n=4, right = 9.55, n=4, $p=0.83$. This supported data obtained in previous studies of control participants (Rio et al., 2015 accepted) and previously published physiological ranges [12]. Two athletes for whom bilateral data was obtained were excluded (one swimmer and one rock climber). This was because significant differences were found in corticospinal excitability of athletes that regularly jumped and those that did not despite the same number of structured physical activity sessions in a week, in a pilot study of 13 athletes ($p=0.002$). Therefore, fourteen athletes were included in the data provided in the paper, n=4 controls and n=10 with tendinopathy.

The following sub-groups emerged: -

Unilateral tendinopathy (unilateral pain with unilateral pathology) n=3

Bilateral tendinopathy (bilateral pain and pathology) n=2

Unilateral tendinopathy (unilateral pain with bilateral pathology) n=5.

Therefore, the question posed was - what is the effect of unilateral pain on the other side (n=8)?

Results from randomised clinical trial – tendon neuroplastic training

Nine athletes (seven men and two women) with either unilateral (n=5) or bilateral (n=4) PT who were taking no medication were included in the study (Table 2). This was part of a larger trial of 29 people, who were all offered inclusion in the TMS component. Nine athletes completed the study, four in the isotonic group and five in the isometric group.

Table 2. Baseline characteristics and testing

										Baseline						
	intervention	**LOT sx**	**height**	**weight**	**BMI**	**KTW L**	**KTW R**	**VISA**	**Leg**	**SLDS**	**MVC**	**AMT**	**M wave**	**curve**	**V50**	**SICI**
M	isotonic	84	181.5	74	22.464	11	7	69	R	8	202	26	26.519	0.1004	1.701	41.87
M	isometric	24	185	81	23.667	13	8	13	R	7	113	28	18.64	0.2165	1.454	33.82
F	isometric	1	178	65.3	20.61	12	11	65.5	R	5	177	33	15.482	0.2368	1.68	56.3
F	isotonic	24	170.5	100.9	34.709	12	9	46	L	7	220	29	18.381	0.1493	1.475	24.65
M	isometric	36	188	81.1	22.946	12	11	76	R	7	165	36	22.354	0.1283	1.414	41.12
M	isotonic	4	183	79.7	23.799	17	16	65	L	5	263	25	17.474	0.1242	1.389	79.74
M	isometric	36	182.6	84.1	25.223	7	8	63	L	7	152	35	24.29	0.1239	1.452	28.61

M	isotonic	120	194	96.5	25.64	17	17	65	R	9	221	38	22.824	0.1237	1.443	20.17
GROUP		30	182.8	81.05	23.733	12	10	65		7	189.5	31	18.64	0.1283	1.454	41.12

M, male. F, female. LOT sx, length of time of symptoms (months). BMI, body mass index. KTW, knee to wall (cm). SLDS, single leg decline squat. MVC, maximal voluntary isometric contraction. AMT, active motor threshold. Mwave, maximal compound wave. SICI, short interval cortical inhibition

The individual data is provided (Table 3,4) including calculated change scores for the outcome measures – VISA-P, SLDS, SICI. Median and mean are provided, though non-parametric tests were chosen due to small sample size.

Table 3. Individual post intervention data

	VISA	SLDS	MVC	AMT	M wave	curve	V50	SICI
Isotonic	67	3	268	23	25.39207	0.05295	1.791	91.36862
	59	4	228	27	21.98545	0.2336	1.74	71.2662
	84	1	244	25	19.03453	0.1124	1.445	72.98601
	73	3	280	30	24.72848	0.07267	1.532	82.97635
Median	70	3	256	26	23.35697	0.092535	1.636	77.98118
Mean	70.75	2.75	255	26.25	22.78513	0.117905	1.627	79.64929
Isometric	41	4	165	26	20.78287	0.2308	3.483	99.44911

	97.5	2	188	33	16.67	0.335	1.934	81.55782
	84	4	229	33	19.98445	0.1536	1.407	72.11755
	72	2	264	35	21.254	0.245	1.478	75.788
	78	3	210	33	20.42	0.247	1.71	52.44907
Median	78	3	208.5	33	20.38366	0.2379	1.706	75.788
Mean	73.625	3	211.5	31.75	19.67283	0.2411	2.0755	76.27231

MVC, maximal voluntary isometric contraction. AMT, active motor threshold. Mwave, maximal compound wave. SICI, short interval cortical inhibition

Table 4. Change scores pre and post intervention

	VISA	SLDS	MVC	% MVC change	AMT	M wave	curve	V50	SICI
Isotonic	-2	5	66	32.67327	-3	-1.12718	-0.04745	0.09	49.49833
	13	3	8	3.636364	-2	3.604896	0.0843	0.265	46.61606
	19	4	67.82	25.78707	0	1.56073	-0.0118	0.056	-4.32399
	8	6	59	26.69683	-8	1.904929	-0.05103	0.089	62.81073
Median	10.5	4.5	62.5	26.24195	-2.5	1.732829	-0.02963	0.0895	46.61606

Isometric	28	3	52	46.0177	-2	2.142729	0.0143	2.029	65.6265
	32	3	11	6.214689	0	1.18803	0.0982	0.254	25.25372
	8	3	64	38.78788	-3	-2.36908	0.0253	-0.007	30.9979
	9	5	28	13.7931	-2.5	1.665379	0.0198	1.1415	56.12128
	15	4	30	16.66667	0	-0.16	0.074	0.245	23.84107
Median	18.5	3	40	26.29049	-2.25	1.426704	0.02255	0.69775	43.55959

The individual SICI data following the RCT is provided (Table AA5).

Table AA5. Individual pre and post cortical inhibition

	Pre (%)	Post (%)	Change (%)
Isometric	33.82	99.45	65.63

2	56.30	81.56	25.25
3	41.12	72.12	31.00
4			
5	24.26	75.79	56.12
	28.61	52.45	23.84
Group median	33.82	75.79	43.56^
Isotonic	41.87	91.36862	49.49833
1	24.65	71.2662	46.61606
2			
3	77.31	72.98601	-4.32399
4	20.17	82.97635	62.81073
Group median	33.26	77.98	46.62#

^ p=0.06, # p=0.25

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