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The Functional Organization of Corticomotor Neurons Within the Motor Cortex Differs Among Basketball and Volleyball Athletes With Patellar Tendinopathy Compared to Asymptomatic Controls

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ABSTRACT

Patellar tendinopathy (PT) typically affects jumping-sport athletes with functional impairments frequently observed. Alterations to the functional organization of corticomotor neurons within the motor cortex that project to working muscles are evident in some musculoskeletal conditions and linked to functional impairments. We aimed to determine if functional organization of corticomotor neuron projections differs between athletes with PT and asymptomatic controls, and if organization is associated with neuromuscular control. We used a cross-sectional design, and the setting was Monash Biomedical Imaging. Basketball and volleyball athletes with ($n = 8$) and without PT ($n = 8$) completed knee extension and ankle dorsiflexion force matching tasks while undergoing fMRI. We determined functional organization via identification of the location of peak corticomotor neuron activation during respective tasks (expressed in X, Y, and Z coordinates) and calculated force matching accuracy for both tasks to quantify neuromuscular control. We observed significant interactions between group and coordinate plane for functional organization of corticomotor projections to knee extensors ($p < 0.001$) and ankle dorsiflexors ($p = 0.016$). Compared to controls, PT group peak corticomotor activation during the knee extension task was 9.6 mm medial ($p < 0.001$) and 5.2 mm posterior ($p = 0.036$), and during the ankle dorsiflexion task 8.2 mm inferior ($p = 0.024$). In the PT group, more posterior Y coordinate peak activation location during the knee extension task was associated with greater task accuracy ($r = 0.749$, $p = 0.034$). Functional organization of corticomotor neurons differed in jumping athletes with PT compared to controls. Links between functional organization and neuromuscular control in the PT group suggest organizational differences may be relevant to knee extension neuromuscular control preservation.

1 | Introduction

Patellar tendinopathy (PT) is highly prevalent in people who participate in volleyball and basketball, affecting 45% and 32% of elite athletes, respectively [1]. The condition is defined by hallmark symptoms of pain localized to the inferior pole of the patella [2] and functional impairments [3–5]. Lower maximal voluntary isometric force (MVIF) has been observed at the knee (extension and flexion) and hip (extension, abduction, and external rotation) [3], although it remains unclear if ankle MVIF is also affected [3, 4]. Impairments in neuromuscular control might also exist as one study supports altered postural control of the lower limb [5], although joint-isolated neuromuscular control has not yet been investigated (e.g., at the knee or ankle).

There is a paucity of investigations for potential mechanisms that could underlie functional impairments in PT. Neurophysiological studies have mostly focused on explaining knee extension impairments [6–8] and have established that there is lower efferent drive to the knee extensor motoneuron pool in populations with PT compared to asymptomatic controls [7, 8]. One recent study observed changes to subcortical excitability which were associated knee extension MVIF impairments, in addition to subtle alterations to excitability along the corticospinal tract [8]. It is possible that changes to the functional organization (location, distribution) of corticomotor neurons within the primary motor cortex (M1) that project to working muscles could also contribute to functional impairments in this population [9], either in parallel to aforementioned mechanisms or as an alternate mechanism. The M1 initiates voluntary movement and has been demonstrated to show considerable functional plasticity in response to pathology and trauma, or activity and motor skill learning [10]. For example, Shanahan et al. [9] identified different functional organization (more posterior location) of corticomotor neurons that project to the knee extensors in people with knee osteoarthritis and, notably, a greater degree of functional reorganization was linked to larger impairments in knee extension neuromuscular control.

Functional magnetic resonance imaging (fMRI) can be used to assess the functional organization of corticomotor neurons within the M1 by quantifying the hemodynamic response within this region if collected while actively performing a motor task [11]. The location of peak activation within the M1 is interpreted as representing corticomotor neurons that project to the corresponding working muscle. To date, the functional organization of corticomotor neurons within the M1 has not been studied in jumping athletes with PT. This could identify novel mechanisms that improve our understanding of functional impairments among people with this condition. Our primary aim was to determine if the functional organization of corticomotor neurons within the M1 that project to the knee extensors and ankle dorsiflexors during neuromuscular control tasks is different among basketball and volleyball athletes with PT compared to asymptomatic control athletes who also participate in basketball or volleyball. The secondary aim was to determine if functional impairments (neuromuscular control, MVIF) are present in jumping athletes with PT compared to asymptomatic controls, and if the functional organization of corticomotor neurons in jumping athletes with PT is associated with knee extension or ankle dorsiflexion neuromuscular control.

2 | Methods

2.1 | Study Design

We used a cross-sectional design and report the study in accordance with Strengthening the Reporting of Observational studies in Epidemiology (STROBE) recommendations for cross-sectional and case–control studies, and the International Scientific Tendinopathy Symposium Consensus (ICON) recommended standards for reporting participant characteristics in tendinopathy research [12]. The study was approved by the Monash University Human Ethics Committee (MUHEC project ID: 19892), and we collected written informed consent for all participants.

2.2 | Setting

We recruited participants and collected data between June 2020 and July 2023. Participants first attended an initial session at either a research laboratory at Monash University Peninsula Campus, Victoria, Australia, or a private physiotherapy clinic in Melbourne, Victoria, Australia, for physical examination screening and familiarization. Within 1 week of the initial session, participants attended a single 1.5-h testing session at Monash Biomedical Imaging, Melbourne, Victoria, Australia.

2.3 | Participants

We utilized social media and University Departmental advertisements to recruit participants from Melbourne-based community and sub-elite sporting organizations. We initially screened potential participants by telephone, then at the initial session via history questions and a physical examination performed by a researcher who was a trained physiotherapist (PV). Participant flow through the recruitment process is shown in Appendix S1. We included participants in the PT group if a diagnosis of PT could be confirmed. This diagnosis was based on self-reported pain localized at the inferior pole of the patella and on palpation of the corresponding site. Participants were also required to report the following features: (i) experiencing pain during loading activities such as running, jumping, and walking downstairs, localized to the inferior pole of the patella; (ii) gradual onset of symptoms lasting a minimum of 3 months; and (iii) heightened pain intensity after periods of inactivity, like sitting, or upon waking.

Participants in the PT group performed a single leg decline squat and required a minimum pain rating of 2/10 on an 11-point numerical rating scale (NRS; 0 = no pain, 10 = worst pain imaginable). A trained physiotherapist (PV) performed ultrasound imaging (Mindray M7, Shenzhen, China) on symptomatic participants to confirm the presence of typical signs of tendinopathy, including hypoechoic regions and thickening (>0.40 mm) at the proximal patellar tendon.

Participants in both groups were required to be actively participating in basketball or volleyball at the time of testing and aged

18 years or older. Participants in either group were excluded if they were experiencing a painful condition at any body region at the time of testing (except PT, in the PT group), or had experienced a painful condition in the previous 6 months that lasted longer than 1 week or required health care professional consultation. They were also excluded if they had a history of severe headaches or migraines, patellar tendon rupture or surgery, or injection with corticosteroid, platelet-rich plasma or other pharmaceutical agents to the patellar tendon or surrounding area within the previous 6 months, or if there was a presence of neurological conditions, inherited connective tissue disorders, or type I or II diabetes.

All participants completed an MRI Screening and Information Form to establish safety which was confirmed by a radiographer (RM) via interview at the testing session.

2.4 | Procedure

A summary of experimental procedure is displayed in Figure 1. At the initial screening session, following confirmation of eligibility, we collected participant demographic data, and participants completed a battery of questionnaires, including the 7-Day Physical Activity Recall survey (used to calculate metabolic equivalents [METs] in the week prior) [13], and psychometric properties [14–21]. Participants in the PT group completed the Victorian Institute of Sport Assessment—Patellar (VISA-P; 0–100, with 100 indicating no patellar tendon pain or functional impairments) [22], and standardized single leg decline squat (SLDS) [2], rating their worst pain experienced at 90° knee flexion on a numerical rating scale (NRS; 0 is no pain, 10 is worst pain imaginable).

We assessed knee extension and ankle dorsiflexion MVIF at this initial session using a protocol consistent with another study [8]. This was assessed to enable sub-maximal force targets to be calculated for the force matching trials, to be performed during the subsequent testing session while undergoing fMRI. Participant's affected/most affected limb was positioned on a custom design MRI-safe rig, in 45° hip flexion and 80° knee flexion for the knee extension MVIF, and 0° hip flexion, 0° knee flexion and the ankle in plantigrade for the ankle dorsiflexion MVIF (Figure 2). In the asymptomatic control group, the dominant-side lower limb was used. Both the MRI-safe rig and the apparatus used for MVIF measurement are identical to that described in a previous investigation [9], and MVIF was normalized to participant body mass (N/kg).

At the testing session, we acquired fMRI scans using a Skyra 3 T MRI scanner (Siemens Healthineers, Erlangen, Germany) with a 32-channel head coil. We collected structural T1-weighted images (192 slices, 1 mm thickness; 0.94 × 0.94 × 0.94 mm in-plane resolution; echo time = 2.07 ms; repetition time = 2300 ms; flip angle 9°), while participants rested in supine. Following this, we acquired echo planar images in a transaxial plane (40 slices; 3 mm thickness; 3.0 × 3.0 × 3.0 mm in-plane resolution; echo time = 35.0 ms, repetition time = 3500 ms; flip angle 90°) with blood oxygen level dependent (BOLD) contrast, while participants completed recorded knee extension then ankle dorsiflexion force matching tasks.

Force matching tasks were performed while participants were positioned on the MRI-safe rig (consistent with the positions used for MVIF assessment). Both tasks involved four isometric contraction blocks lasting 48 s each, interspersed with five resting blocks of identical duration. The MRI scanner triggered the start of the sequence, allowing force and MRI data to be synchronized. During the contraction blocks, participants aimed to match their actual force to a target force, that ranged between 0% and 5% MVIF and followed a fixed sinusoidal pattern with four cycles lasting 12 s each, for each isometric contraction block. This MVIF range was used as higher intensities have resulted in movement of the head [9]. Real-time force was displayed overlaying target force to participants through a head coil-mounted mirror directed toward a projection screen at the rear of the scanner [9].

We calculated force matching task accuracy using the calculation for root mean square (RMS) $= \sqrt{\left(\frac{\text{error value}}{\text{MVIF}}\right)}$, where error value was the difference between target force and actual force averaged across the four contraction blocks. Accordingly, this accuracy is considered RMS %error and was interpreted as representing knee extension or ankle dorsiflexion neuromuscular control, respectively.

2.5 | Processing

All fMRI processing was performed using the software package FSL (v.6.0) [23]. We used a protocol consistent to that used

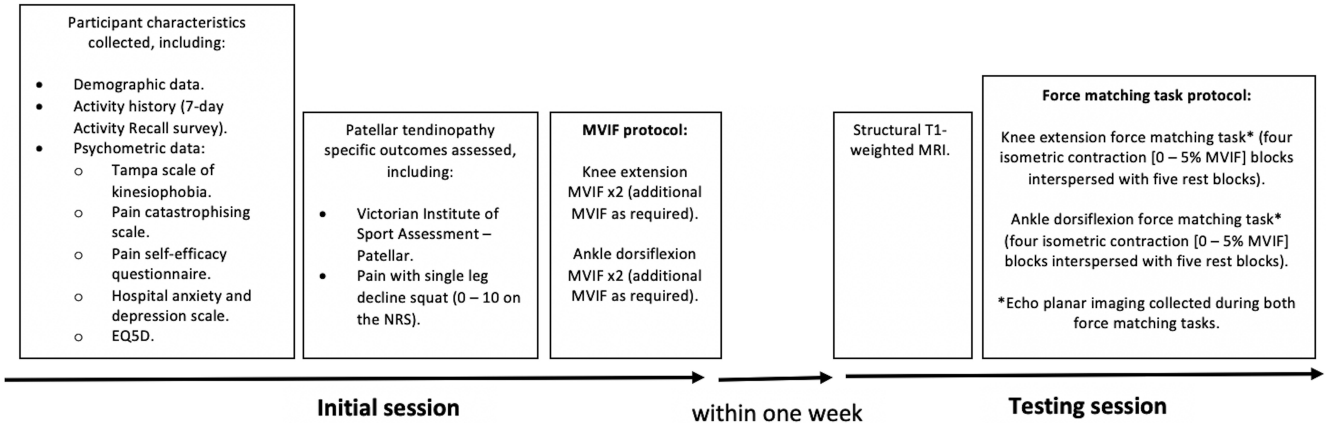

FIGURE 1 | Summary of experimental procedure.

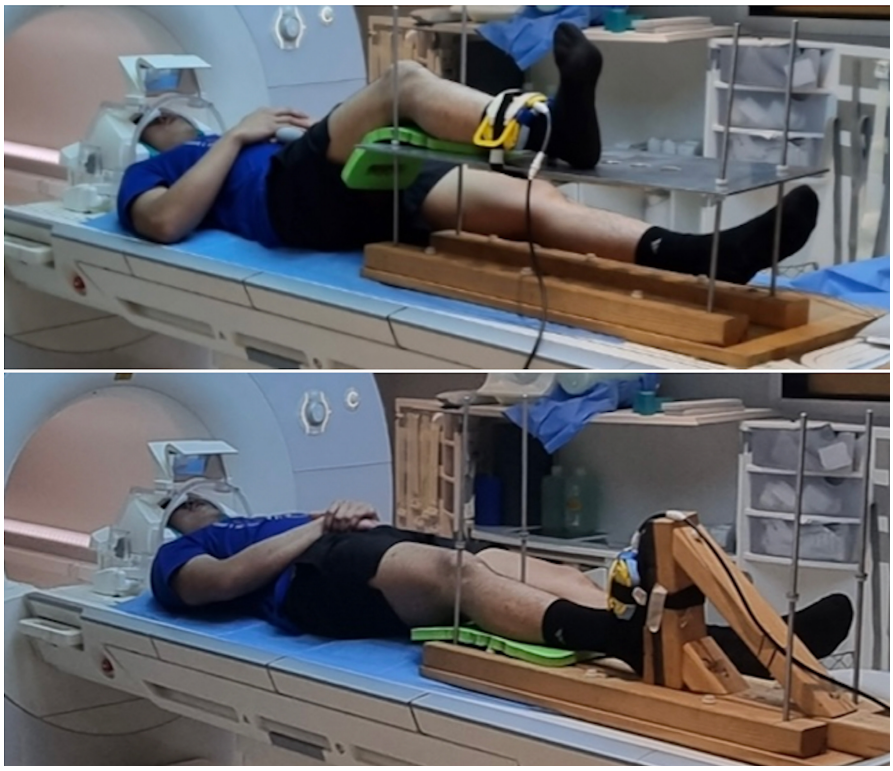

FIGURE 2 | Experimental setup.

previously to analyze BOLD signals during force matching tasks in another persistent knee condition [9] for preprocessing, registration, and statistical analysis. Preprocessing included image reorientation to standard, removal of nonbrain structures using the Brain Extraction Tool (v.2.1) and, using fMRI Expert Analysis Tool (FEAT; v.6.0), motion and slice timing correction (interleaved), functional image brain extraction, high-pass temporal filtering, and spatial smoothing (5 mm full-width half-maximum). We also used FEAT for registration and statistical analysis. Participant functional images were registered to their structural images (linear, boundary-based registration) and their structural images nonlinearly registered to standard space (Montreal Neurologic Institute [MNI] 2 mm template).

We ran a general linear model that included regressors for the active and resting blocks of the force matching task, which we generated for each participant using their target force data. Additionally, we applied a temporal derivative. The fit of variance in BOLD signal to these regressors was calculated as parameter estimates, and contrasts of parameter estimates (COPE) generated to compare model fit to the null hypotheses (no fit). Contrasts of parameter estimates were expressed as a *z*-statistic for each voxel and filtered to generate individual participant maps of voxel clusters surpassing a threshold of $z = 3.1$, $p = 0.05$ that represented corticomotor neuron projections within the M1 that were active and correlated with target force throughout the sinusoidal pattern range of respective force matching tasks (as it is expected metabolic response to the task would also fluctuate in line with this sinusoidal pattern). Accordingly, one was generated for corticomotor neuron projections within the M1 active during the knee extension force matching task and another for corticomotor neuron projections within the M1 that were active during the ankle dorsiflexion force matching task.

We filtered each individual participant z-statistic map through a custom region of interest (ROI) mask using the FEATquery function to localize the search area to the expected region of activation for lower limb motor function [9, 24, 25]. The MNI coordinates for this custom ROI map limited the search area to the precentral sulcus in the hemisphere contralateral to the limb that performed the force matching task. This ROI mask was further adapted to match the search area used by Shanahan et al. [9] and based on the expected region of activation for lower limb motor function. In particular, the search area was limited to Y coordinate = −10 to −40 mm, and Z coordinate = 30 to 90 mm, and an additional limit was added to the search area for the X coordinate (0 to −30 mm when investigating the left hemisphere, and 0–30 mm when investigating the right hemisphere [which was flipped to left hemisphere coordinate range for statistical analysis]).

This search identified the position of the voxel with the highest z statistic (location of peak corticomotor neuron activation) within a cluster of voxels $z \geq 3.1$ ($p < 0.05$), expressed in MNI standard space (X, Y, and Z coordinates), for each individual and for both force matching tasks. We manually inspected coordinate locations to verify they were within a contiguous cluster of ≥20 voxels [9].

To determine the distance between the location of peak corticomotor neuron activation coordinates during the knee extension and ankle dorsiflexion tasks (henceforth referred to as ankle–knee coordinate distance), in each respective plane, we used the calculation = ankle dorsiflexion coordinate location–knee extension coordinate location.

2.6 | Sample Size Calculation

We calculated the required sample size using the software General Linear Mixed Model Power and Sample Size (GLIMMPSE; https://glimmpse.samplesizeshop.org), an open-source tool for calculating power and sample size for general linear multivariate models. Data from Shanahan et al. [9] were utilized to inform estimates in this calculation as we deemed that study to be the most analogous to ours in terms of case-population studied and methodology employed, across investigations with comparable protocols. In particular, we used point estimate and variability data for the location (X, Y, and Z coordinates) of peak corticomotor neuron activation during a knee extension force matching task. To detect a significant main effect of group, with power of 80% and the alpha set at 5%, we required $n = 15$ case and $n = 15$ control participants.

2.7 | Statistical Analysis

We examined the normality of variables of interest using the Shapiro–Wilk test in GraphPad Prism (Version 9.4.1, GraphPad Software, Boston, USA). Participant characteristics, force matching task accuracy, and MVIF data were investigated for between-group differences using independent samples t-tests for normally distributed data and the Mann–Whitney test for non-normally distributed data. Fisher's exact test was used to investigate whether sport played (volleyball or basketball), or sex (male or female), were significantly different between the groups [26].

Data were analyzed using linear mixed models (LMMs) in Statistical Package for the Social Sciences (SPSS; v.26.0; Chicago, USA). To investigate functional organization of corticomotor neuron projections in jumping athletes and controls, we included coordinates for the location of peak corticomotor neuron activation during the knee extensor force matching task as the dependent variable, and group and coordinate plane (X, Y, or Z) as fixed effects. We repeated this analysis with coordinates for the location of peak corticomotor neuron activation during the ankle dorsiflexion force matching task. We also used a LMM to investigate whether ankle–knee coordinate distance differed between groups, including ankle–knee coordinate distance as the dependent variable, and group and coordinate plane (X, Y, or Z) as fixed effects. In any of these LMMs, if significant interactions were identified, or significant main effects, we ran post hoc pairwise comparisons to determine if group differences were present for each level of the respective dependent variable.

Using PT group data, we investigated whether knee extension or ankle dorsiflexion force matching task accuracy was associated with functional organization coordinate data (X, Y, and Z planes). For all correlation analyses, we used Pearson's r when data were normally distributed, and Spearman's rho when analyzing data that were non-normally distributed, and set thresholds to negligible ($r < 0.3$), low (0.3–0.5), moderate (0.5–0.7), high (0.7–0.9), and very high (>0.9) [27].

We set significance at $p < 0.05$ for all analyses. We present data as individual data plots, and group data as mean (standard deviation [SD]), or median (interquartile range [IQR]). Between-group differences are presented as mean (95% confidence intervals).

3 | Results

Eight athletes with PT and eight asymptomatic control athletes participated in this study. The PT group had a high mean symptom duration of 44 (SD 36) months, and predominately bilaterally symptoms (88%). PT specific data are presented in Table 1. There were no significant between-group differences for participant characteristics between the PT and control groups (Table 1).

Ankle force matching task accuracy was 0.17 (95% CI 0.03–0.31; t (14) $= 2.61$, $p = 0.021$) RMS %error lower in the PT group than the control group, while no significant differences were observed between groups for knee extension force matching task accuracy ($p = 0.616$) (Figure 3; Appendix S2). Ankle dorsiflexion MVIF was 0.35 (95% CI 0.10–0.60; t (14) $= 3.28$, $p = 0.010$) N/kg greater in the PT group than the control group. No significant differences were observed between groups for knee extension MVIF ($p = 0.294$) (Figure 4; Appendix S2).

3.1 | Location of Peak Corticomotor Neuron Activation Within the Motor Cortex

Individual coordinate data for the location of peak corticomotor neuron activation during the knee extension and ankle dorsiflexion force matching tasks are displayed in Figure 5, and group mean (SD) in Appendix S3. For the location of peak corticomotor neuron activation during the knee extension task, we observed a significant interaction between group and coordinate plane (f (48) $= 10.356$, $p < 0.001$). In the PT group, the X coordinate was located 9.63 (95% CI 4.80–14.45; $p < 0.001$) mm medially, and the Y coordinate was located 5.19 (95% CI 0.36–10.01; $p = 0.036$) mm posteriorly, compared to the control group. No significant difference was observed between groups for Z coordinate location ($p = 0.515$).

For the location of peak corticomotor neuron activation during the ankle dorsiflexion task, we observed a significant interaction between group and coordinate plane (f (48) $= 4.546$, $p = 0.016$). The Z coordinate was located 8.16 (95% CI 1.14–15.19; $p = 0.024$) mm inferiorly in the PT group compared to the control group. No significant differences were observed between groups for X ($p = 0.060$) or Y coordinates ($p = 0.876$).

For ankle–knee distance, no significant interaction was observed between group and coordinate plane (f (48) $= 1.929$, $p = 0.156$).

3.2 | Associations Between the Location of Peak Corticomotor Neuron Activation During Force Matching Tasks, and Force Matching Task Accuracy

In the PT group, we observed a strong positive association between Y coordinate location for peak corticomotor neuron activation during the knee extension force matching task and knee extension force matching task accuracy ($r = 0.749$, $p = 0.033$). No other significant associations were observed ($p > 0.05$) (Appendix S4).

TABLE 1 | Participant characteristics in patellar tendinopathy and control groups (mean [SD], unless stated otherwise).

	Patellar tendinopathy ($n = 8$)	Asymptomatic control ($n = 8$)
Age, years	29.1 (9.0)	22.4 (5.7)
BMI	26.2 (3.7)	23.8 (3.6)
Sex, male (%)	6 (75%)	7 (88%)
Sport, volleyball (%)	5 (63%)	7 (88%)
Activity, METs	299.9 (38.9)	306.2 (58.75)
TSK, /68	38.5 (4.7)	34.1 (6.7)
PCS, /52	19.6 (9.1)	17.0 (7.5)
PSEQ, /60	46.9 (10.5)	48.8 (10.5)
HADS anxiety subscale, /21	6.4 (4.2)	7.1 (4.3)
HADS depression subscale, /21	3.1 (3.0)	2.4 (2.0)
Quality of life rating, 0–100	77.6 (8.9)	81.8 (10.0)
Duration, months	44 (36)	—
Bilateral symptoms (%)	7 (88%)	—
SLDS pain, /10	5.6 (2.0)	—
VISA–P, /100	59.3 (14.8)	—

Abbreviations: BMI, body mass index; HADS, hospital anxiety and depression scale; MET, metabolic equivalents; PCS, pain catastrophizing scale; PSEQ, pain self-efficacy questionnaire; SLDS, single leg decline squat; TSK, Tampa scale of kinesiophobia; VISA-P, Victorian institute of sport assessment—Patellar.

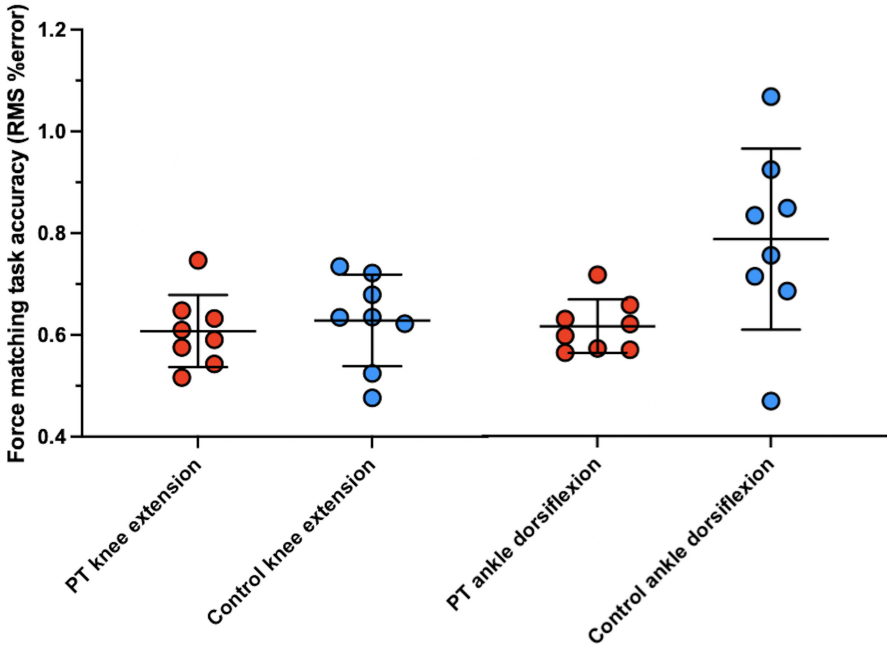

FIGURE 3 | Individual and mean (SD) knee extension and ankle dorsiflexion force matching task accuracy (RMS %error) in patellar tendinopathy and control groups.

4 | Discussion

In this study, we aimed to determine if the functional organization of corticomotor neurons within the M1 that project to the knee extensors and ankle dorsiflexors during neuromuscular control tasks differed among basketball and volleyball athletes with PT compared to asymptomatic controls. We found functional organization differed between the groups, with the location of peak corticomotor neuron activation during the knee extension force matching task being 9.6 mm more medial and 5.2 mm more posterior in our PT group compared to controls, while peak corticomotor neuron activation location during the

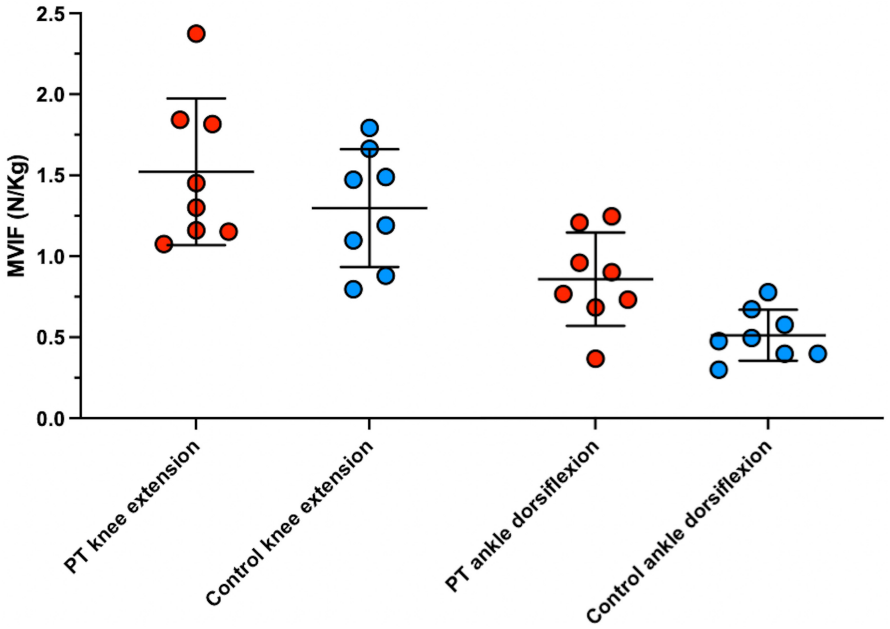

FIGURE 4 | Individual and mean (SD) knee extension and ankle dorsiflexion MVIF (*N*/kg) in patellar tendinopathy and control groups.

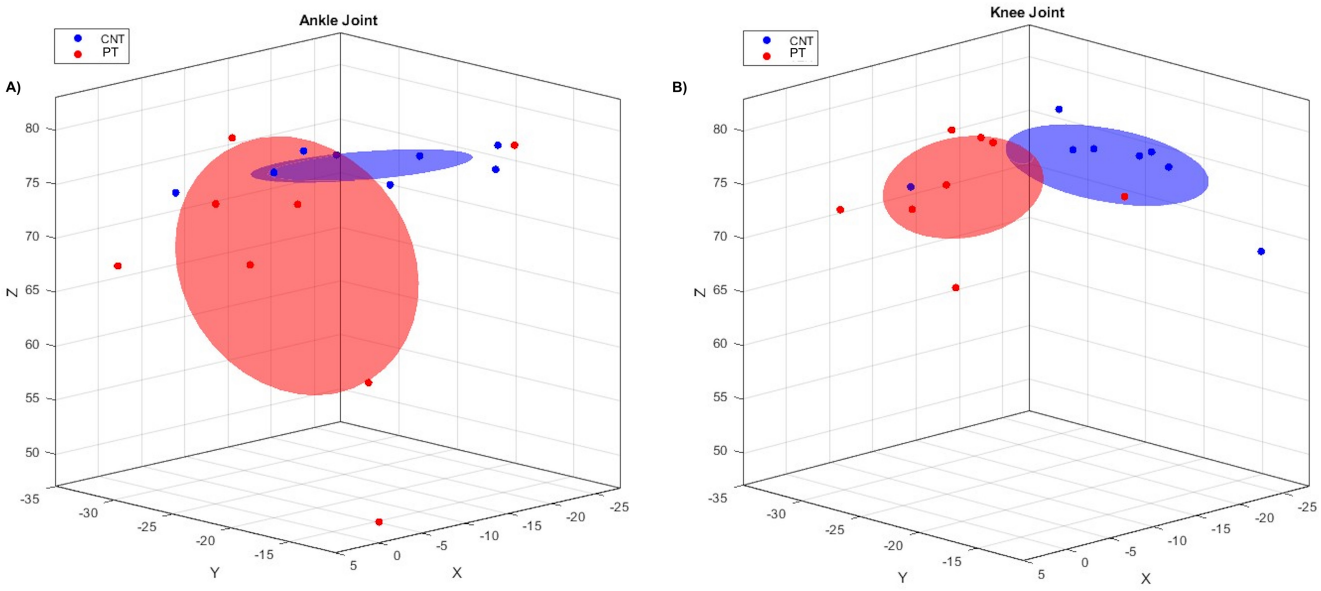

FIGURE 5 | Individual and group (ellipsoid) coordinate location for peak corticomotor neuron activation during the (A) ankle dorsiflexion force matching task and (B) knee extension force matching task, in patellar tendinopathy (PT; red) and control (CNT; blue) groups.

ankle dorsiflexion force matching task was located 8.2 mm more inferior in the PT group. In our PT group, we also observed a correlation between the Y coordinate location for peak corticomotor neuron activation during the knee extension force matching task and knee extension task accuracy (more posterior location of peak corticomotor neuron activation was associated with greater knee extension task accuracy). Together, our findings suggest functional organization of the M1 may be relevant to knee extension neuromuscular control in jumping athletes with PT.

Differences in functional organization have previously been demonstrated in a number of persistent musculoskeletal conditions [9, 28–33]. Notably, in some reports [9, 31], links have been established between the distance the peak activation coordinate location is shifted away from the respective control coordinate and worse neuromuscular control. For example, Shanahan et al. [9] observed the location of peak corticomotor neuron activation during a knee extension force matching task was shifted 4.1 mm more anteriorly in individuals with knee osteoarthritis compared to a control group, and greater anterior shift was associated with worse accuracy on the knee extension force matching task. Changes in functional organization can also result in temporal alterations to neuromuscular control [31]. Accordingly, functional organization (or, plausibly reorganization) might be implicated in functional impairments.

We observed an association between the PT group location of peak corticomotor neuron activation during the knee extension force matching task and knee extension force matching task accuracy, with a more posterior coordinate location associated with more accurate neuromuscular control. Remarkably, our findings diverge from previous investigations. Specifically, we observed a substantial displacement in the peak corticomotor activation location away from the mean of the respective control coordinates, which correlated with improved neuromuscular control. Our findings indicate that basketball and volleyball athletes with PT might undergo functional reorganization of the M1 [34], as a compensation to preserve motor function. This is as opposed to the functional organization observed in certain other persistent conditions that might be linked to functional impairments [9, 31]. A limitation to this hypothesis is our study design was cross-sectional so we cannot be certain of the temporal nature of the relationship.

Functional reorganization of M1 corticomotor projections has been documented at length in athletic populations and linked to skill acquisition and maintenance [34]. This reorganization is thought to result from repetitive and complex skills training [34]. Both groups included in our study were required to be actively participating in skilled sport (basketball or volleyball). As a result, some degree of functional reorganization would be expected compared to sedentary populations; however, this would not explain the between-group differences we observed. One recent study reported lower subcortical excitability in basketball and volleyball athletes with PT that was associated with lower knee extension MVIF [8]. It is possible our findings indicate functional reorganization of the M1 in jumping athletes with PT is an adaptive neural strategy that aims to compensate for lower subcortical excitability, to maintain drive to the knee extensor motoneuron pool and sustain motor function that is relevant to neuromuscular control.

Compared to controls, our PT group were 22% more accurate during the ankle dorsiflexion force matching task, and their ankle dorsiflexion MVIF was 33% greater. Although the precise mechanisms behind these novel findings remain unclear, one plausible explanation is that they may be linked to adaptations along the corticospinal-motoneuronal pathway. One theory suggests that increased excitability of corticospinal tract neurons could compensate for lower spinal excitability, thereby maintaining efferent drive to the knee extensor motoneuron pool [8]. Additionally, these neural adaptations may also enhance ankle function, as evidence exists for task- and muscle-dependent connections in corticospinal excitability between knee extensors and ankle dorsiflexors during motor imagery tasks [35]. Furthermore, co-activation of these regions during locomotion could contribute to the observed effects [36, 37].

A loss of organization, or "smudging," of discrete corticomotor neuron projections to adjacent or similar muscles has been proposed as a possible mechanism underlying motor dysfunction [28, 29, 32, 33]. We did not identify any differences between our PT and control groups for ankle–knee coordinate distance, indicating smudging is not a feature of the functional organization of corticomotor neurons in jumping athletes with PT. This offers an alternate explanation for why knee extension neuromuscular control was not different in our PT group despite finding altered location of peak corticomotor neuron activation during knee extension and ankle dorsiflexion force matching tasks. It is interesting to consider if the corticomotor neuron projections we studied were sensitive to detect a presence of smudging; this might have been identified had we aimed to locate corticomotor neurons active while alternate muscles worked, for example, the location of corticomotor neurons projecting to adjacent muscles that contribute to knee extension [28]. We did not observe any between-group difference for knee extension neuromuscular control or MVIF, and it would be expected that either of these functions could be negatively affected if smudging was present. Accordingly, it is unlikely smudging would be identified in jumping athletes with PT even if alternate corticomotor neuron pathways were studied.

Consideration should also be applied to whether the force matching tasks we employed were appropriate to elicit the metabolic, and consequent hemodynamic, responses within the M1 that is representative of typical cortical demand. Our force matching tasks only ranged between 0% and 5% MVIF, while typical jumping sports activity would require much greater intensity. Motor cortex hemodynamic response has been shown to increase alongside isometric exercise intensity [38], so it is possible a different force matching task intensity would produce different results, or at the very least an expanded map of active corticomotor neurons. Corticomotor neuron projections have been demonstrated to be both task and movement specific [39], and likely utilize a pattern of neurons distributed across the M1 [39]. Consequently, it is possible different results might have been produced had we used a different task, for example, knee extension or ankle dorsiflexion MVIF.

5 | Limitations

In this study, we interpreted the fMRI BOLD response to the force matching tasks, and by extension the peak loci of this signal, to signify the activity of corticomotor projecting neurons. In addition to these excitatory neurons, building evidence suggests the BOLD response may also be influenced by the activity of other cells, including intrinsic cortico-cortical inhibitory neurons, astrocytes and vascular cells (e.g., endothelial cells, vascular smooth muscle, and pericytes) [40]. Accordingly, our results may have been influenced by the activity of these alternate cell types. However, it is important to recognize that they still predominantly reflect the activity of excitatory corticomotor projecting neurons, as these neurons are the primary consumers of tissue oxygen [40]. We only included athletes still actively participating in basketball or volleyball at the time of testing. As a result, it is possible our findings do not represent a general population with PT. A recent study highlighted differences could exist between people with PT who do and do not continue sports participation, such as psychological coping strategies, and severity of symptoms [8]. Given the small sample size included in our study, caution should be practiced when drawing conclusions from these findings, and they should be considered exploratory in nature. Using our data for peak corticomotor neuron location, we performed post hoc power analyses with the open-source

tool GLIMMPSE (https://glimmpse.samplesizeshop.org), and our results do not appear to be at elevated risk of type II error (power ≥ 0.9 to detect group x coordinate interaction).

6 | Perspectives

Our findings indicate that the functional organization of corticomotor neurons that project to the knee extensors and ankle dorsiflexors during force matching tasks is different among basketball and volleyball athletes with PT compared to controls. Associations in the PT group between functional organizational differences and force matching task accuracy indicate these differences may be relevant to compensations that aim to preserve knee extension neuromuscular control.

Author Contributions

P.V., D.J.K., A.K.F., and P.M. conceived the design and developed the methods. P.V. collected the data. P.V., D.J.K., B.V., and P.M. performed analysis, and P.V., D.J.K., B.V., A.K.F., A.G., and P.M. interpreted findings, prepared the manuscript, and approved the final submission.

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Ethics Statement

This study was approved by the Monash University Human Ethics Committee (MUHEC project ID: 19892), and we collected written informed consent for all participants.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.