

Regional vastus medialis and vastus lateralis activation in females with patellofemoral pain

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1 Title: **Regional vastus medialis and lateralis activation in females with**
2 **patellofemoral pain**

3

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26 **ABSTRACT:**

27 **Introduction:** To investigate whether regional activation patterns in the vasti muscles
28 differ between females with and without patellofemoral pain (PFP), and whether muscle
29 activation patterns correlate with knee extension strength.

30 **Methods:** Thirty-six females with PFP and 20 pain-free controls performed a
31 standardized knee flexion-extension task. Activation of vastus medialis (VM) and
32 lateralis (VL) was collected using high-density surface electromyography and analyzed
33 using Principal component (PC) analysis. Spatial locations and temporal coefficients of
34 the PCs, and percent variance they explain were compared between groups and
35 between the concentric and eccentric phases of the movement. Correlations were
36 assessed between PC features and knee extension strength.

37 **Results:** The spatial weights of PC1 (general vasti activation) and PC2 (reflecting
38 vastus-specific activation) were similar between groups ($R>0.95$). Activation patterns in
39 PFP were less complex than controls. Fewer PCs were necessary to reconstruct 90% of
40 the signal for PFP participants in the concentric phase ($p<0.05$), and the difference in
41 bias of activation to VM (concentric phase) or VL (eccentric phase) was less between
42 phases for PFP participants ($p<0.05$). Smaller difference in vastus-specific activation in
43 concentric and eccentric phases (less task specificity of VM/VL coordination) was
44 related to greater maximal knee extension strength ($p<0.05$, $R<-0.43$).

45 **Conclusion:** These data suggest PFP involves a simpler control strategy of VM and
46 VL. The inverse association between task specificity and maximal knee extension
47 strength suggests different presentations of PFP: lower knee extension strength but

48 VM/VL coordination task specificity comparable to controls, or knee extension strength
49 comparable to controls but lower VM/VL coordination task specificity.

50

51 **KEYWORDS:** Patellofemoral pain; EMG; quadriceps; muscle strength; Principal
52 Component Analysis.

53

54 **INTRODUCTION:**

55 Patellofemoral pain (PFP) is common in young individuals engaged in sports. It is
56 a complex, multifactorial syndrome with a pathogenesis that has not been fully
57 elucidated. Historically, poor patellar tracking due to unbalanced activation of the vastus
58 medialis (VM) and lateralis (VL) muscles has been considered to contribute to PFP (1).
59 Although some studies support this hypothesis (2–4), others have not identified
60 differences in timing or amplitude of activation of vastii muscles between symptomatic
61 individuals and painfree controls (5, 6). A systematic review of the literature highlighted
62 ‘substantial and unexplained heterogeneity’ (7). This variability is likely to be explained
63 by both physiological and methodological factors.

64 A possible contributor to this variability is the potential differences in muscle
65 activation between clinical presentations. Common clinical findings of PFP include lower
66 knee extension strength (8), lower hip muscle strength (9), and higher dynamic foot
67 mobility (10). In addition, interventions focused on different sites such as knee muscle
68 strengthening (11), hip muscle strengthening (12) and foot orthoses (13) have all been
69 shown to improve PFP symptoms in the short term. One possibility is that altered
70 quadriceps activation is more common in people with PFP who also have weak knee
71 extensors. To our knowledge, no studies have tested whether altered quadriceps
72 activation is associated with features of PFP clinical presentation.

73 Another factor that is likely to contribute to the unexplained variability of muscle
74 activation in PFP relates to the methods used to quantify muscle activity. The technique
75 most commonly used is surface electromyography (EMG), using one pair of electrodes
76 placed on the belly of each vastus (2, 4, 5). This straightforward measure has a number

77 of limitations: for instance, due to variation in the location of the innervation zone
78 relative to the electrodes, surface EMG amplitude differences up to 75% can be
79 observed in the VM for electrodes positioned only 15 mm apart (14). Although this effect
80 can be limited with normalization of EMG amplitude during isometric contractions (15),
81 the VM innervation zone has been shown to shift under the electrodes as a function of
82 knee angle (i.e. muscle length) (16), complicating the recording of representative
83 surface EMG in dynamic contractions. In addition, as VM motoneurons innervate
84 muscle fibres clustered within the muscle (17, 18), region-specific differences in VM
85 activation can be observed both in reflex (19) and voluntary (16, 20) contractions; as
86 regions within the vasti produce forces in different directions (21), differences in regional
87 activation may result in different distribution of the forces applied to the patella.

88 Recent advances in EMG technology allow for the placement of up several tens
89 of electrodes on single muscles (high-density EMG, HDsEMG). As signals are collected
90 from different locations of the muscle, HDsEMG helps to overcome some limitations of
91 conventional surface EMG. For instance, it is possible to take into account the effect of
92 the location of the innervation zone and other anatomical factors on the estimation of
93 neuromuscular activation (14), and to describe the activation of regions within a muscle
94 (16). Specifically, regional muscle activation can be identified using factorization
95 algorithms such as principal component analysis (22).

96 This exploratory study aimed to investigate whether VM and VL regional
97 activation patterns, identified using high-density surface electromyography, differ
98 between females with and without PFP. We hypothesized that, compared to painfree
99 participants, those with PFP would demonstrate different coordination between VM and

100 VL as defined by spatial and temporal features of principal components extracted from
101 VM and VL EMG activity during a standardized dynamic task. We also hypothesized
102 that coordination between VM and VL would differ most in participants with lower knee
103 extension strength. The research hypotheses were framed within contemporary theories
104 on neuromuscular adaptations to pain, which predict that altered neuromuscular control
105 is one factors that could sustain pain and function loss (23).

106

107 **METHODS:**

108 **Participants:**

109 Thirty-six females with symptomatic PFP and 20 healthy, sex-matched control
110 participants were recruited from the local community and physiotherapy clinics. To be
111 included in the PFP group, participants had to be: females, 19-35 years old, with retro-
112 or peri-patellar knee pain of intensity equal or greater than 3/10 (on an 11-point numeric
113 rating scale; 0 being 'no pain'; 10 being 'worst pain imaginable') for at least 1 month
114 aggravated by any of the following activities: sitting for long time periods, stairs,
115 squatting, running, kneeling or jumping. They also needed to report pain or discomfort
116 to at least one of the following tests: patellar palpation, patellar compression, resisted
117 knee extension with knee close to full extension, isometric knee extension while
118 applying pressure proximally to the patella. Control participants could not have had any
119 knee pain in the last 12 months. For both groups, exclusion criteria were: previous
120 lower-limb surgery, chronic neuromuscular disorders, or knee musculoskeletal
121 disorders. All participants provided written informed consent before the start of the

122 experimental session. The study that was approved by the institution's Clinical
123 Research Ethics Board.

124 Age, body mass, height, duration of pain (self-reported), average pain intensity in
125 the previous week (11-point numerical rating scale; 0 = 'no pain'; 10 = 'worst pain
126 imaginable') were obtained for each participant. Physical activity (General Physical
127 Activity Questionnaire, (24)) and functional limitation (Anterior Knee Pain score, (25))
128 were estimated using validated questionnaires. The test leg was the most painful knee
129 (if both were painful) or a random leg for controls.

130 **Clinical tests:**

131 Dynamic foot mobility was assessed using the 'foot mobility' test. Following a
132 validated and reliable procedure (26), foot arch height and midfoot width were
133 measured using a caliper twice; while sitting and standing. The difference between
134 measures taken in non-weightbearing and weightbearing positions was recorded and
135 used to describe dynamic foot mobility.

136 Isometric knee extension strength was measured using a Biodex (System 4 Pro;
137 Biodex Medical Systems, Shirley, NY). The hip and knee angles were standardized at
138 85° and 45° (0° being full extension), respectively, and the participants were secured
139 firmly to the chair. The resistance was applied approximately 2 cm proximal to the
140 medial malleolus. The participants were asked to contract maximally the quadriceps
141 muscle of the leg tested, reaching a maximal contraction over 1-2 s to ensure a smooth
142 contraction and to maintain it for at least 3 s. This procedure was repeated 3 times with
143 at least 1 minute of rest in-between trials. Verbal encouragement was provided at each
144 trial. The highest peak torque of the three trials was used as maximal knee extension

145 strength (KES); analyses were also run on the KES value normalized to body mass
146 (nKES).

147 **Protocol:**

148 After a few repetitions to warm-up, participants performed 10 repetitive knee
149 flexion-extension movements on the dynamometer from ~100 to 5° of knee flexion
150 against a constant resistance set at 10% of their KES. A metronome standardized the
151 pace at 3 s for each concentric knee extension, eccentric knee extension and rest.

152 **Data collection:**

153 Similar to a previous study (16), the HDsEMG grids were placed according to
154 anatomical references (Fig.1). The medial and lateral edges of VM and VL were
155 identified using ultrasound imaging (LogicScan 64 LT-1T; Telemed, Vilnius, Lithuania)
156 and were marked on the skin. As thickness of the subcutaneous tissues between the
157 electrodes and the muscle may influence EMG recordings, a single ultrasound image
158 was also taken in the proximal and distal region of both muscles, approximately in
159 correspondence of the proximal and distal third of the grid array. VM and VL innervation
160 zones were located using a linear electrode array (16 silver bar electrodes, 10-mm
161 interelectrode distance; OTBioelettronica, Torino, Italy) and marked on the skin. Two
162 HDsEMG grids (semidisposable adhesive matrix; OTBioelettronica) were placed on the
163 skin so that the innervation zone aligned between the second and third column, and all
164 the electrodes were placed of the target muscle. Each grid comprised 64 electrodes
165 arranged in 5 columns and 13 rows with a single electrode missing in one of the
166 corners, 8 mm inter-electrode distance and was held in place using bi-adhesive foam.
167 With this electrode position, activation of different muscle regions can be observed

168 along the columns of the electrode grid, with negligible influence of changes in VM
169 muscle architecture associated with changes in knee angle joint (16). Reference
170 electrodes (2x3.5 cm; conductive hydrogel; Kendall, Covidien, Mansfield, MA) were
171 placed on the patella and on the medial and lateral epicondyles. HDsEMG signals were
172 collected in monopolar modality using an EMG amplifier (128-channel EMG-USB;
173 OTBioelettronica, Torino, Italy). Signals were amplified 500-1000 times, filtered (band-
174 pass 10-750 Hz) and digitized at 2048 Hz using a 12-bit A/D converter. The knee
175 position signal from the dynamometer was acquired simultaneously using the same
176 amplifier.

177 **Data analysis:**

178 Ultrasound images were analysed using ImageJ (National Institutes of Health,
179 Bethesda, Maryland, USA). The thickness of the subcutaneous tissues was measured
180 as the distance between the skin and the most superficial edge of each muscle. All
181 EMG analyses were run in Matlab 2016B (The MathWorks, Inc., Natick, MA, USA). A
182 Butterworth filter (4th order, 10-400 Hz) was applied to the EMG signals before
183 processing. Envelopes were calculated for each channel of both HDsEMG grids by full-
184 wave rectification and low-pass filtering at 8 Hz (Butterworth filter, 4th order). For each
185 participant, the EMG values corresponding to 10-90° of the knee flexion-extension
186 repetitions were extracted, and envelopes were normalized to the maximal envelope
187 value of all channels across VM and VL. EMG envelopes were concatenated in two
188 matrices of 128 EMG channels by N samples (N = 20 or N = 36 participants, multiplied
189 by time samples), one for the PFP group and one for the control group.

190 As the analysis aimed to identify regional activation within the vasti, Principal
191 Component Analysis (PCA, (27)) was applied to the HDsEMG dataset. Removing the
192 mean from the data before PCA did not change the results of the study, so the data are
193 presented for non-centered data. In line with previous studies that used separate
194 factorization analyses for different conditions or groups (28, 29), PCA was applied
195 separately for PFP and controls. As opposed to running PCA pooling all participants
196 together, this approach enables identification of between-group differences in spatial
197 weights; but limits between-group comparison of temporal coefficients to PCs that have
198 similar spatial weights ($R > 0.95$ in this study). PCA identifies clusters of channels with
199 large covariance in time, factorizing the signal in principal components (PC); PCs
200 represent the general activation pattern (PC1) or the major ways in which this pattern
201 could be modulated (PC2-4) at any instant in time. Based on a recent study (22), it is
202 expected that PC1 will have only positive values and will describe a general VM/VL
203 activation; instead PC2 and above will have both positive and negative values, and will
204 describe how the activation of regions within VM and VL is modulated (i.e.:
205 increases/decreases compared to PC1). Preliminary analyses showed that the first four
206 components described patterns of activation of the four regions of interest in this study
207 (proximal/distal VM; proximal/distal VL), hence four PCs were considered. Each PC can
208 be described by three indices (Fig. 2; Fig. 3): 1) spatial weights: the location of the
209 channels where the PC is most represented; 2) temporal coefficient: the time profile of
210 the activation of the PC; 3) the variance explained: how much of the variance of the
211 signal is accounted for by the PCA. Each EMG envelope matrix **M** was factorized into
212 128 PCs, each consisting of 128 weights and N coefficients. Spatial weights were

213 calculated as the eigenvectors ζ of the covariance matrix of \mathbf{M} . Temporal coefficients
214 were calculated as $\zeta^T * \mathbf{M}$, which is the matrix product between the transposed
215 eigenvectors and the EMG envelope matrix. PCs were sorted according to their
216 eigenvalues. Spatial weights, temporal coefficients and variance explained of the PCs
217 extracted from the PFP and from the control participants were compared between
218 groups. For each participant, the temporal coefficients of the first 4 PCs corresponding
219 to the concentric and the eccentric phase of each repetition were identified and
220 averaged across knee angles and repetitions. The coefficient of determination ($CD=1-$
221 SSE/SST , where SSE is the sum of squared residuals, and SST is the total variance of
222 the original signal) was used to calculate the variance explained for the first 4 PCs,
223 separately for the concentric and eccentric phase of each participant. The mean total
224 variance explained was calculated separately for the concentric and eccentric phase of
225 the movement for each participant by varying the number of PCs between one and ten.
226 The minimum number of PCs that accounted for at least 90% of the variance was
227 identified for each participant, separately for the concentric and eccentric phase of the
228 movement.

229 **Statistical analysis:**

230 All statistical analyses were performed using SPSS v.22 (IBM Inc., Armonk, NY,
231 USA). Parametric tests were used if data were normally distributed and had equal
232 variance, non-parametric tests were used if these assumptions were not met.
233 Anthropometric parameters and clinical measures were compared between groups
234 using independent T-tests.

235 To investigate whether the thickness of subcutaneous tissues differed between
236 females with and without PFP, the thickness was compared between *groups* (PFP or
237 control, between-subject factor), *muscles* (VM or VL, within-subject factor) and *locations*
238 (proximal or distal, within-subject factor) using a 3-way mixed model analysis of
239 variance (ANOVA).

240 Pooling data across participants for the PCA, and using the PCA to distinguish
241 differences in patterns of activation between groups with and without PFP pain requires
242 the general patterns of activity to be similar within the participants for each group. We
243 tested this by applying PCA to individual participants (separately for females with and
244 without PFP) and then evaluating the Pearson correlation coefficients between the
245 spatial weights for each participant with the mean spatial weights for their group.

246 The three descriptors of muscle activation identified with PCA were compared.
247 The complexity of muscle activation patterns is reflected by the number of PCs that
248 accounted for at least 90% of the variance, and this was compared between groups
249 using Wilcoxon tests, separately for the concentric and the eccentric phase of the
250 movement. To describe whether the spatial localization of the PCs was similar between
251 groups, Pearson correlation was run on the spatial weights of the first four PCs between
252 groups. PCs with spatial weights that correlated with $R > 0.95$ were considered similar
253 between groups. When PCs for the two groups were not significantly correlated, the
254 maps of spatial weights across electrode sites was view qualitatively to identify
255 differences in distribution that would explain the between-group difference. For PCs with
256 a similar spatial structure ($R > 0.95$), it was considered valid to compare the temporal
257 coefficient of activation of the vasti muscles between *groups* and *phases* (concentric or

258 eccentric, within-subject factor) using 2-way mixed model ANOVA, separately for each
259 component. Student's t-tests with Bonferroni correction for multiple comparisons were
260 used for post-hoc comparisons. For PCs with spatial structure that differed between
261 groups, temporal coefficients were compared between the phases (concentric and
262 eccentric) only using paired Student's t-tests.

263 To identify any relation between clinical measures and EMG dysfunction,
264 Spearman correlation was used to test associations between the EMG indices that were
265 significant in the between-group comparisons and KES, nKES, dynamic midfoot width
266 and dynamic foot height. Statistical significance was set at $p<0.05$.

267

268 **RESULTS:**

269 **Participant characteristics and clinical tests:**

270 The two groups did not differ for age (participants PFP: 27 ± 4 ; controls: 26 ± 4
271 years old, $p=0.38$), weight (62 ± 9 vs. 58 ± 9 kg, $p=0.10$), height (166 ± 8 vs. 168 ± 9 cm,
272 $p=0.59$), or physical activity level (4018 ± 2961 vs. 3153 ± 2034 METmin/week, $p=0.20$). A
273 significant difference was identified for body mass index, although the average value for
274 both groups fell within the normal range (22.5 ± 5.2 vs. 20.6 ± 1.7 , $p<0.01$). Participants
275 with PFP reported a history of knee pain for 12-60 (interquartile range) months, average
276 pain of 4.1 ± 1.3 in the previous week and their Anterior Knee Pain Score was 74.8. Both
277 KES (116.5 ± 30.6 vs. 135.3 ± 32.9 Nm, $p<0.05$) and nKES (1.88 ± 0.54 vs. 2.31 ± 0.41
278 Nm/kg, $p<0.01$) were lower in females with PFP compared to controls. Foot height
279 mobility (14.3 ± 1.7 vs. 11.8 ± 2.9 mm, $p<0.01$) but not midfoot width (8.8 ± 4.0 vs. 8.2 ± 1.7
280 mm, $p=0.44$) was higher in females with PFP compared with controls.

281 **Subcutaneous tissue thickness:**

282 Ultrasound measurement of thickness of subcutaneous tissues did not differ
283 between groups (PFP: 9.2 ± 3.5 mm; controls: 8.6 ± 3.5 mm, $p=0.42$). Subcutaneous
284 tissues were thicker over VL than VM (9.1 ± 3.4 vs 8.0 ± 3.4 mm; main effect of *muscle*,
285 $p<0.01$), and proximally than distally (9.4 ± 3.8 vs 7.7 ± 2.8 mm; main effect of *location*,
286 $p<0.001$). No interactions were observed ($p>0.25$).

287 **Number of principal components:**

288 A lower number of PCs was needed to explain 90% of the variance for
289 participants with PFP (median: 2; 25th-75th percentiles: 2-3; Fig. 4) than for controls (3;
290 2-4.5) in the concentric phase of the movement ($p<0.05$). No differences were observed
291 in the eccentric phase of the movement ($p=0.20$). These results were confirmed when
292 the variance explained (calculated by applying PCA on each participant separately) was
293 compared between groups ($p<0.05$). Given that four PCs explained $92.2\pm 4.0\%$ and
294 $94.7\pm 2.4\%$ for controls and participants with PFP respectively (N=20 and N=36; figure
295 S1, variance explained by different number of PCs), all remaining analyses were
296 performed using the first four PCs.

297 **Spatial features of principal components:**

298 The median correlation coefficient between spatial weights extracting using PCA
299 separately for each participant and their group average spatial weight was high (median
300 (interquartile range); PC1: 0.75 (0.67-0.85); PC2: 0.97 (0.94-0.99); PC3: 0.80 (0.72-
301 0.88); PC4: 0.81 (0.47-0.88); all N=56), supporting the use of PCA on group data. Visual
302 assessment of the spatial location of the PCs enables the determination of regional
303 activation patterns described by each PC. PC1 which we refer to as PC1_{General activation},

304 had positive spatial weights for all the channels, describing simultaneous activation of
305 both vasti, and was similar between groups ($R = 0.96$). The PCs other than $PC1_{\text{General activation}}$
306 had both positive and negative values in their spatial weights and temporal
307 coefficients, and described modulation (increase and decrease of activation) of
308 $PC1_{\text{General activation}}$ (22). In control participants (Fig. 2), $PC3$ has positive spatial weights
309 (light shading) in the distal region of both VM and VL, and negative values (dark
310 shading) proximally. When the temporal coefficients are positive, muscle activation
311 increases in the channels with positive spatial weights (distally) and decreases where
312 they are negative (proximally); by contrast, when the temporal coefficients are negative,
313 muscle activation increases proximally (channels with negative spatial weights) and
314 decreases distally (channels with negative spatial weights). Taken together, $PC3$ in
315 controls describes co-activation of the distal region of VM and VL (when the temporal
316 coefficients are positive; start of concentric and end of eccentric) and of the proximal
317 regions (when the temporal coefficients are negative; start of concentric and end of
318 eccentric); for this reason, it was referred to as $PC3_{\text{Vasti co-activation}}$. $PC3_{\text{Vasti co-activation}}$
319 differed between groups ($R = 0.75$); for PPF $PC3$ described regional activation within
320 the VL that was similar to controls, but no concomitant regional activation in VM. In
321 controls $PC4$ described the co-activation of proximal VL and distal VM or vice versa
322 (Fig. 2), and was referred to as $PC4_{\text{Proximal-distal vasti co-activation}}$. This was different in PPF (R
323 = 0.73) where $PC4_{\text{Proximal-distal vasti co-activation}}$ identified regional activation within the VM
324 similar to controls, but did not represent VL activation (Fig. 3). The spatial weight values
325 for $PC2$, were positive for VM and negative for VL, hence describing a bias to

326 contraction for VM from this PC, thus referred to as, PC2_{Vastus-specific activation}. The spatial
327 distribution of PC2_{Vastus-specific activation} was similar between groups ($R = 0.99$).

328 **Temporal features of principal components:**

329 As the spatial weights of PC3_{Vasti co-activation} and PC4_{Proximal-distal vasti co-activation}
330 differed between groups in their location, temporal coefficients could not be directly
331 compared for these PCs. Thus, only temporal coefficients of PC1_{General activation} and
332 PC2_{Vastus-specific activation} were compared between groups. PC1_{General activation} was more
333 active in the concentric than the eccentric phase of the movement (main effect of *phase*,
334 $p < 0.001$; Fig. 5) and this did not differ between groups (main effect; $p = 0.14$, interactions
335 $p = 0.99$). A significant interaction was identified between *groups* and *phases* for the
336 temporal coefficient of PC2_{Vastus-specific activation} ($p < 0.05$, Fig. 5), meaning that redistribution
337 of VM/VL activation between the concentric and the eccentric phase of the movement
338 was lower in participants with PFP compared to controls (i.e.: participants with PFP had
339 more co-activation of VM and VL). Both groups showed negative PC2_{Vastus-specific activation}
340 temporal coefficients (i.e. activation to expression of PC2, and thus bias to VL
341 activation) in the concentric phase of the movement and positive PC2_{Vastus-specific activation}
342 temporal coefficients (bias to VM activation) in the eccentric phase of the movement;
343 this resulted in significantly lower temporal coefficients during the concentric phase than
344 the eccentric phase of the movement ($p < 0.001$). In controls, the temporal coefficients of
345 PC3_{Vasti co-activation} (0.02 ± 0.26 and 0.03 ± 0.23 , $p = 0.67$) or PC4_{Proximal-distal vasti co-activation}
346 (0.01 ± 0.20 and 0.02 ± 0.17 , $p = 0.36$) did not differ between concentric and eccentric
347 phase of the movement. In PFP, PC3_{Within-VL activation} was lower in the concentric than the
348 eccentric phase of the movement (-0.02 ± 0.26 and 0.03 ± 0.17 , $p < 0.05$); a similar

349 tendency, although not-significant, was observed for PC4_{Within-VM activation} (0.00 ± 0.15 and
350 0.03 ± 0.17 , $p=0.06$).

351 **Associations between clinical tests and neuromuscular activation patterns:**

352 Correlations were assessed separately for participants with and without PFP to
353 investigate associations between the EMG indices that were found to differ significantly
354 between groups (temporal coefficients of PC2_{Vastus-specific activation}; number of PCs
355 necessary to reconstruct 90% of the variance in the concentric phase of the movement;
356 see above) and clinical measures (KES; nKES; midfoot width; foot height). One
357 individual was identified as a potential outlier and this was statistically confirmed by
358 inputting the data to a linear regression model. For that participant, Cook's distance
359 measures were 0.93 (temporal coefficients of PC2_{Vastus-specific activation} and nKES) and 0.95
360 (temporal coefficients of PC2_{Vastus-specific activation} and KES), much higher than the cut-off
361 value for outliers ($4/N=0.11$). After exclusion of data for that individual, an inverse
362 correlation was identified between temporal coefficients of PC2_{Vastus-specific activation} during
363 the eccentric phase of the movement and KES ($p=0.01$, $R=-0.43$; nKES: $p=0.001$, $R=-$
364 0.52 ; Fig. 6), that is, participants with a lower redistribution of activation between VL and
365 VM had higher KES. Association in the same direction was observed for temporal
366 coefficients of PC2_{Vastus-specific activation} during the concentric phase of the movement,
367 although the strength of the association was lower (KES: $p<0.05$, $R=-0.38$; nKES:
368 $p=0.09$, $R=-0.3$). These associations were not observed in females without PFP
369 ($p>0.12$, $R<0.36$). No other significant correlations were identified.

370

371 **DISCUSSION:**

372 These data show that the regional activation within VM and VL during a low-force
373 dynamic knee extension task differs between females with and without PFP. The lower
374 number of PCs needed to reconstruct 90% of the variance (i.e. fewer components
375 required to explain the pattern of EMG activity) for those with PFP than controls, and the
376 lesser difference in bias to VM or VL between the concentric and eccentric task phases,
377 both suggest a simpler control strategy of vasti muscle coordination in PFP. The data
378 also show lower co-activation between VM and VL in PFP than in controls; PC3 and
379 PC4 represented activation of only VM or VL in the PFP group, unlike the controls
380 where these PCs represented coordination between the vasti muscles. The inverse
381 association between task specificity of VM/VL coordination and maximal knee extension
382 strength in PFP demonstrates a spectrum of presentations with lower knee extension
383 strength but VM/VL coordination that was similar to controls at one end, and high knee
384 strength but compromised VM/VL coordination at the other end.

385 Altered VM and VL activation patterns have been observed in PFP in this study.
386 During the concentric phase of the knee extension, vasti muscle activation of females
387 with PFP can be explained by two main activation patterns, i.e.: global activation (PC1)
388 and redistribution between VM and VL (PC2). To reconstruct the signal to a similar
389 extent (i.e. explain the same amount of variation), the control participants required
390 inclusion of an additional activation pattern that represented co-activation of distal or
391 proximal regions of VM and VL. This observation suggests that activation of the VM and
392 VL in PFP participants included a smaller component of EMG that controlled
393 coordination between medial and lateral forces during muscle shortening. Similar
394 findings of simpler control in association with a musculoskeletal condition has been

395 reported for the deep hip external rotator muscles in participants with femoro-acetabular
396 impingement syndrome (28). During the eccentric phase, the number of PCs did not
397 differ between groups; however, the additional activation patterns in PFP represented
398 regional activation of a single vastus muscle, rather than coordination between of
399 regions between VM and VL. This concurs with observation of less synchronous
400 activation of motor units in VM and VL in PFP (3) and other studies that identified
401 differences in timing and amplitude of vasti activation using conventional bipolar surface
402 EMG (4, 30).

403 Taken together, the present results suggest that a PC that accounts for co-
404 activation between regions of VM and VL explains an important component of pattern
405 variance in controls but not in PFP. Consistent with proposed theories of patellofemoral
406 joint control (1, 31), this co-activation between VM and VL could be interpreted to
407 represent a strategy coordinate forces for optimal patellar tracking in controls. Females
408 with PFP used patterns of EMG that involved lesser modulation of regional activation
409 within each vastus, but instead used overall co-activation plus components that account
410 for bias of activity to only VM or VL. It has been shown *in vivo* that load applied by each
411 vastus muscle in isolation influences the distribution of forces applied to the patella (21,
412 32). It is plausible that this would be impacted by the distribution of activity between the
413 vasti muscles and differences in this pattern between controls and participants with PFP
414 could be expected to alter patellar kinematics and pressure distribution within the
415 patellofemoral joint observed in PFP (30, 33).

416 An interesting observation was the between-group differences in the task
417 specificity of the relative activation of VM and VL during phases of dynamic knee

418 extension. As control participants showed a bias towards VL activation during the
419 concentric phase and towards VM activation during the eccentric phase of the knee
420 extension movement, this may have significance for differences in patellar tracking and
421 joint loading between the different tasks. This between-muscle redistribution of EMG
422 was limited in PFP, especially during the eccentric phase of the movement. Reduced
423 task specificity has also been observed in some other musculoskeletal conditions, such
424 as low back pain (34) and may imply a loss of the fine-tuning of the control of forces in
425 the patellofemoral joint. Te and colleagues (35) have recently shown that the
426 representations of the individual heads of the quadriceps on the motor cortex are closer
427 together for individuals with PFP than healthy controls; similar to what was suggested in
428 other studies (36, 37). Although speculative, such merging of the muscle
429 representations at the cortical level may underlie a lesser capacity to modulate
430 coordination of vasti muscles in a task specific manner. However, we cannot interpret
431 from our data where in the nervous system changes might be occurring (e.g. cortical,
432 spinal, etc) and further work is required.

433 The temporal coefficients of PC1_{General activation} suggest a greater contribution of
434 this PC during the concentric than eccentric phase of the movement, without a
435 difference between groups. This suggests that the lower muscle activation in the
436 eccentric versus the concentric phase of movement (38) is preserved in PFP and would
437 be expected base on physiological property of muscle to require less EMG activation to
438 generate equivalent force in eccentric contractions. The co-activation patterns (PC3_{Vasti}
439 co-activation and PC4_{Proximal-distal vasti co-activation}) in controls were equally observed in the
440 concentric and eccentric phase of the movement, suggesting that the within-muscle

441 redistribution of activation represented by these PCs occurred similarly for both tasks.
442 Unlike the control participants, the within-muscle regional activation patterns (PC3_{Within-}
443 VL activation and PC4_{Within-VM activation}) in PFP indicated preferential activation of one muscle
444 rather than co-activation, specifically the distal VL (PC3_{Within-VL activation}) and VM (PC4_{Within-VM activation}, trend) in the eccentric phase of the movement, similar to previous
445 preliminary observations in the VM (16). This suggests that preferential activation of
446 vasti regions that have larger potential to contribute to medio-lateral patellar forces
447 mainly occurs in the eccentric phase of the movement. Although this aspect of the
448 motor pattern was more task specific for PFP and controls, and is not consistent with
449 our suggestion of a simplified control strategy in this group, it must be taken together
450 with the fact that these PCs only explain a small percentage of the variance.
451 Regardless, this observation remains interesting because, in contrast to control
452 participants, this within-muscle redistribution was not co-activation and occurred at
453 different times for two muscles. These findings highlight differences in how females with
454 and without PFP activate the distal regions of VM and VL in dynamic contractions.

456 Contrary to our hypothesis, participants with lower knee extension strength did
457 not show the largest differences in neuromuscular control (lower redistribution between
458 VM and VL). Instead, an inverse association was observed – the weaker participants
459 had a neuromuscular activation pattern more like controls. A recent classification
460 identified two categories of adaptation to pain: major “movement avoidance” patterns
461 and subtle “redistribution within and between muscle” (39). The current data provide an
462 interesting new observation – we propose an interpretation that the adaptations in
463 females with PFP are distributed along a continuum, with some presenting with a

464 “reduced force output” strategy, whereas others present with subtle differences in
465 muscle coordination. There is of course a proportion of the PFP group with strength and
466 neuromuscular activation between these two extremes. The lower force output may be
467 associated not only with neuromuscular factors, but also to changes in muscle structural
468 parameters (40). Regardless, these two different strategies may present with different
469 consequences for long-term health of the patellofemoral joint.

470 Because of the cross-sectional design of the study, it is not possible to define
471 whether changes in force output and neuromuscular activation are a cause or
472 consequence of PFP, or whether in the long term the effects on patellofemoral joint
473 health are different. Regardless, it is tempting to speculate about the potential clinical
474 implications as it may be helpful to identify subgroups of participants that respond
475 differently to interventions. Specifically, females with PFP and lower knee extension
476 strength may benefit from interventions that focus on quadriceps strengthening,
477 whereas exercises that target motor control might be beneficial for females with PFP
478 and knee extension strength similar to controls, but concomitant differences in
479 coordination of vasti muscles. Future studies should investigate whether interventions
480 matched to these deficits in females with PFP have better clinical outcomes than
481 treatments that are not matched.

482 Due to conduction volume of soft tissues, surface electromyographic signals are
483 known to be influenced by crosstalk. One of the main contributors to crosstalk in the
484 surface EMG is the thickness of subcutaneous tissues. Despite larger BMI in females
485 with PFP, ultrasound measures of subcutaneous tissue thickness over VM and VL did
486 not differ between groups (average difference: 0.6 mm). For this reason, any effects of

487 subcutaneous tissue thickness on the crosstalk in the surface EMG activation patterns
488 would be similar between groups. Additionally, crosstalk from far sources is likely to be
489 observed as similar EMG amplitude fluctuations in most channels of the grid,
490 represented by PC1 in this study, while the other PCs representing regional activation
491 may be less influenced by crosstalk. While the amount of crosstalk present in this
492 dataset cannot be precisely defined, the absence of differences between group in
493 thickness of subcutaneous tissue and the use of PCA suggest that crosstalk had a
494 minimal influence on the results of this study.

495 In conclusion, females with PFP have simpler VM and VL activation strategies,
496 observed as lower co-activation of regions between VM and VL and lower redistribution
497 of activation from VL to VM when the concentric and eccentric phases of the knee
498 extension are compared. As VM/VL redistribution was inversely correlated to maximal
499 knee extension strength, we suggest two different presentations of PFP: prevalent lower
500 knee extension strength or prevalent lower redistribution between VM and VL. These
501 dysfunctions may be preferentially targeted by different interventions, potentially
502 resulting in improved clinical outcomes.

503

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508 The authors declare no conflicts of interest.

509 Results of the study are presented clearly, honestly, and without fabrication,
510 falsification, or inappropriate data manipulation.

511 Results of the present study do not constitute endorsement by the American College of
512 Sports Medicine.

513

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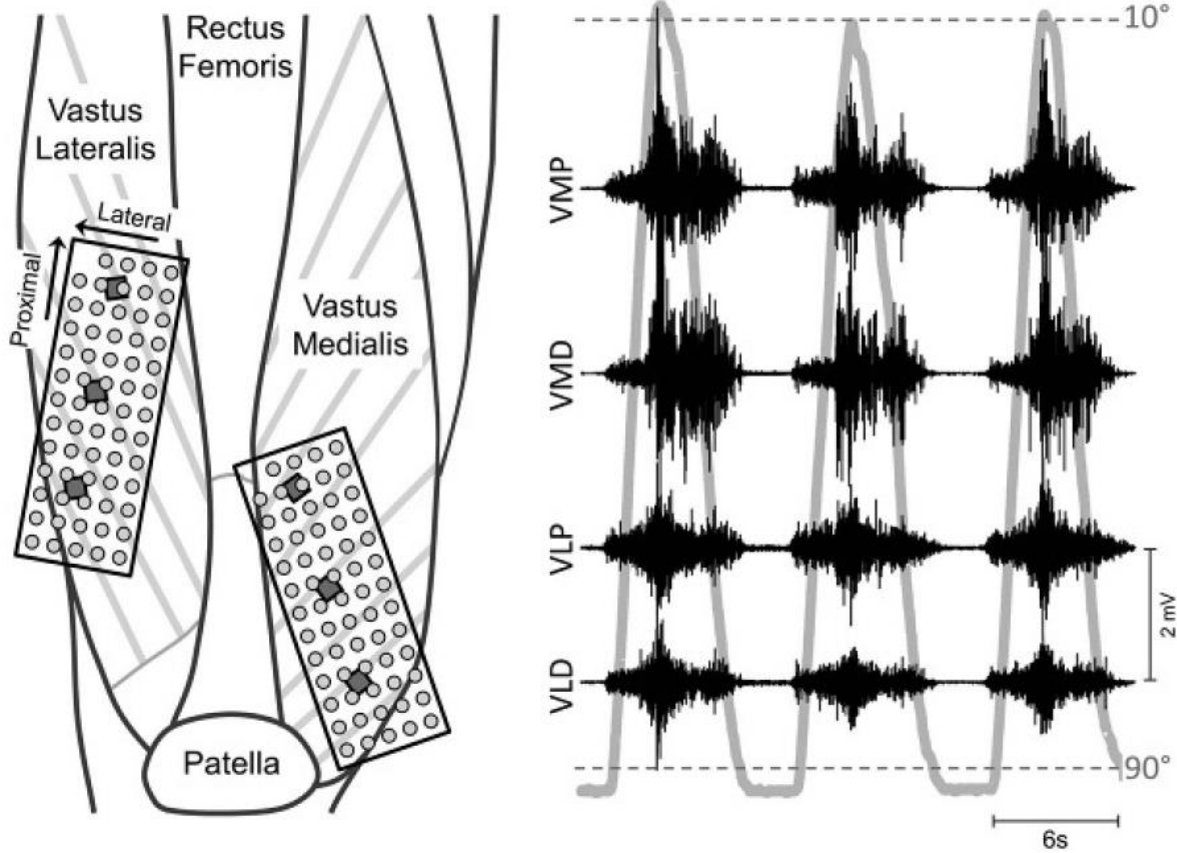
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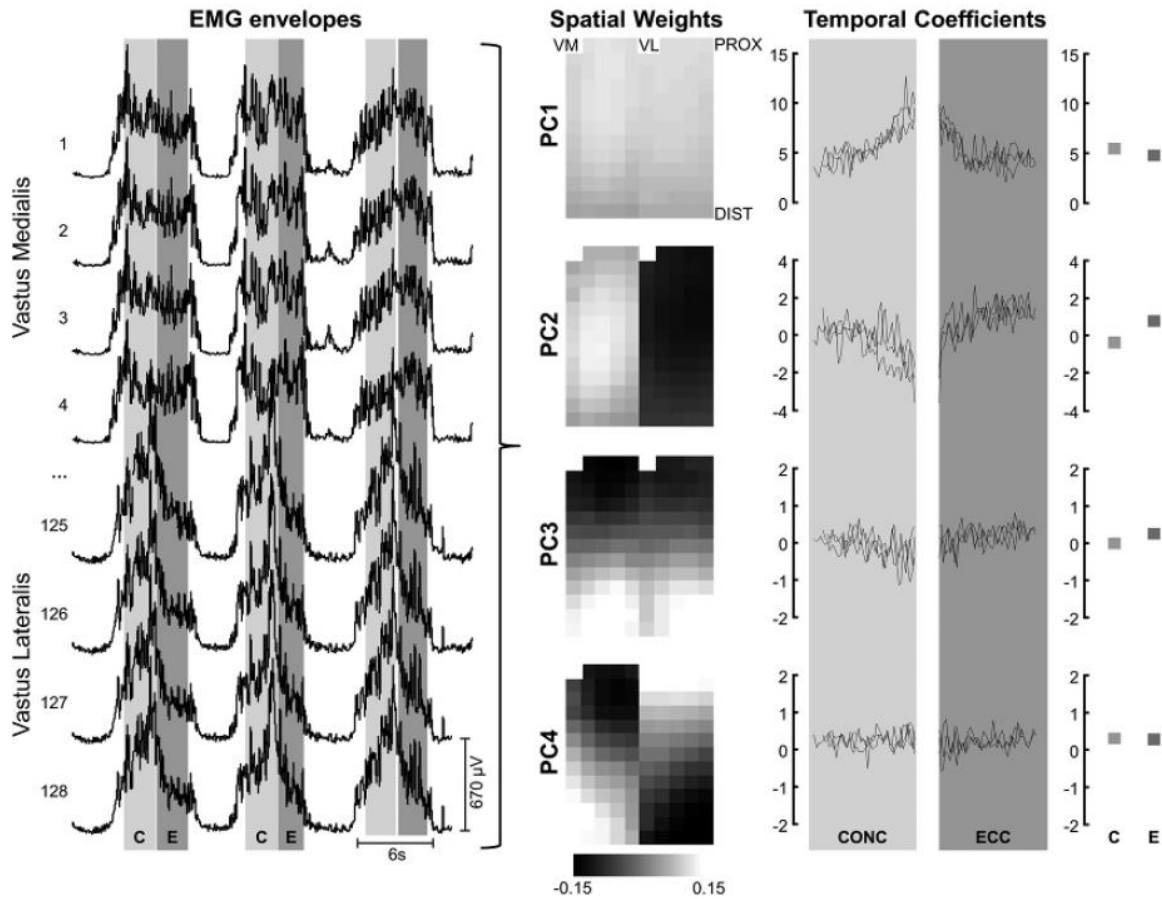
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628

629 Supplemental Figure 1.tif

630 **FIGURES:**



631
632 Figure 1: Experimental set-up. Left: placement of the electrode grids on vastus medialis
633 (VM) and vastus lateralis (VL). Gray squares identify the innervation zones. Right:
634 example of knee joint angle (thick gray line) and monopolar surface EMG collected from
635 proximal (P) and distal (D) locations within VM and VL.



636

637 Figure 2: Example of PCA analysis of high-density EMG signals for a control participant.

638 Left: 8 of the 128 EMG envelopes used for the PCA from 3 repetitions of a control

639 participant; light and dark gray boxes identify the concentric (C) and eccentric (E) phase

640 of the movement (10-90°). Middle: spatial weights of PC1-4 (from PCA using data for all

641 control participants); light and dark shades identify positive and negative weights

642 respectively. PC1_{General activation} shows positive weights for both VM and VL; PC2_{Vastus-specific}

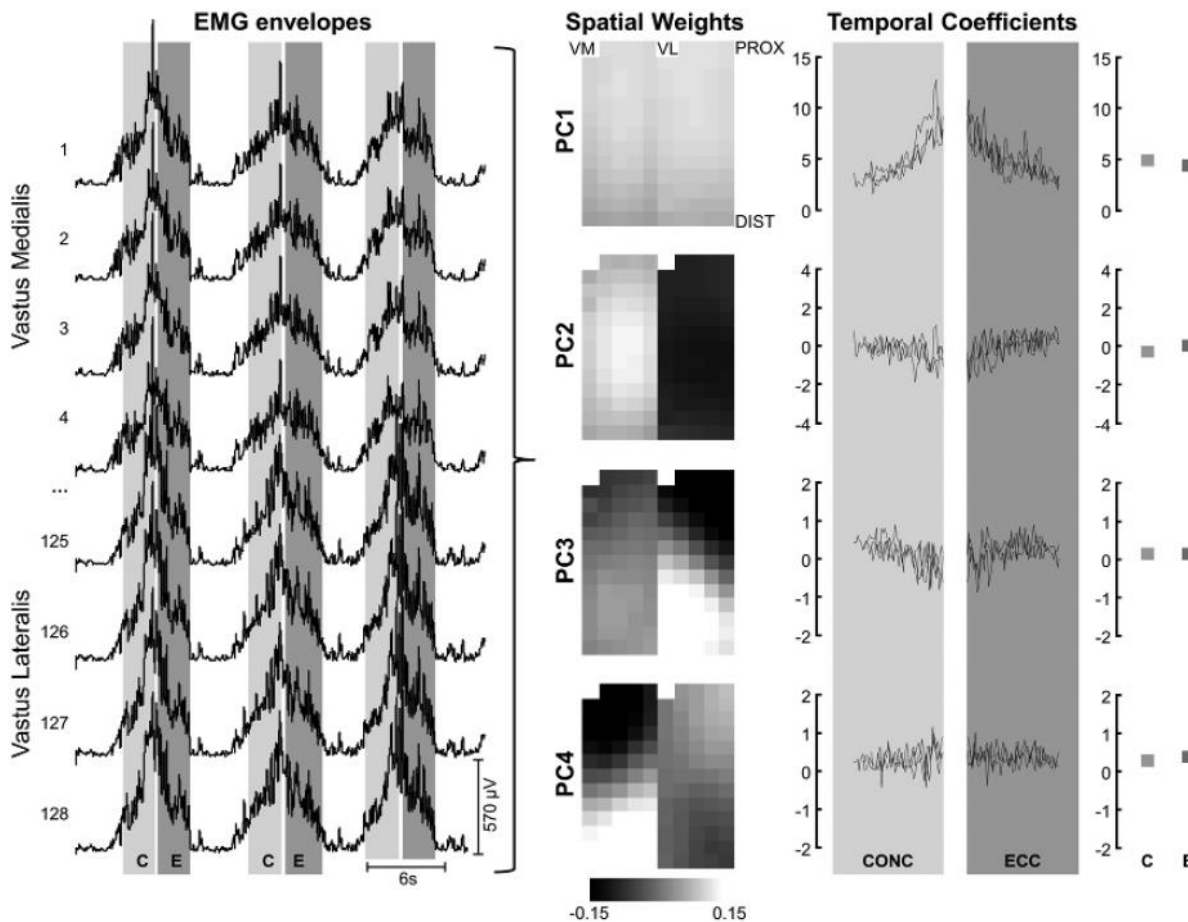
643 _{activation} shows positive weights of VM and negative weights for VL; PC3_{Vasti co-activation}

644 shows positive weights for both muscles distally and negative weights proximally;

645 PC4_{Proximal-distal vasti co-activation} shows positive weight for VM distally and VL proximally, and

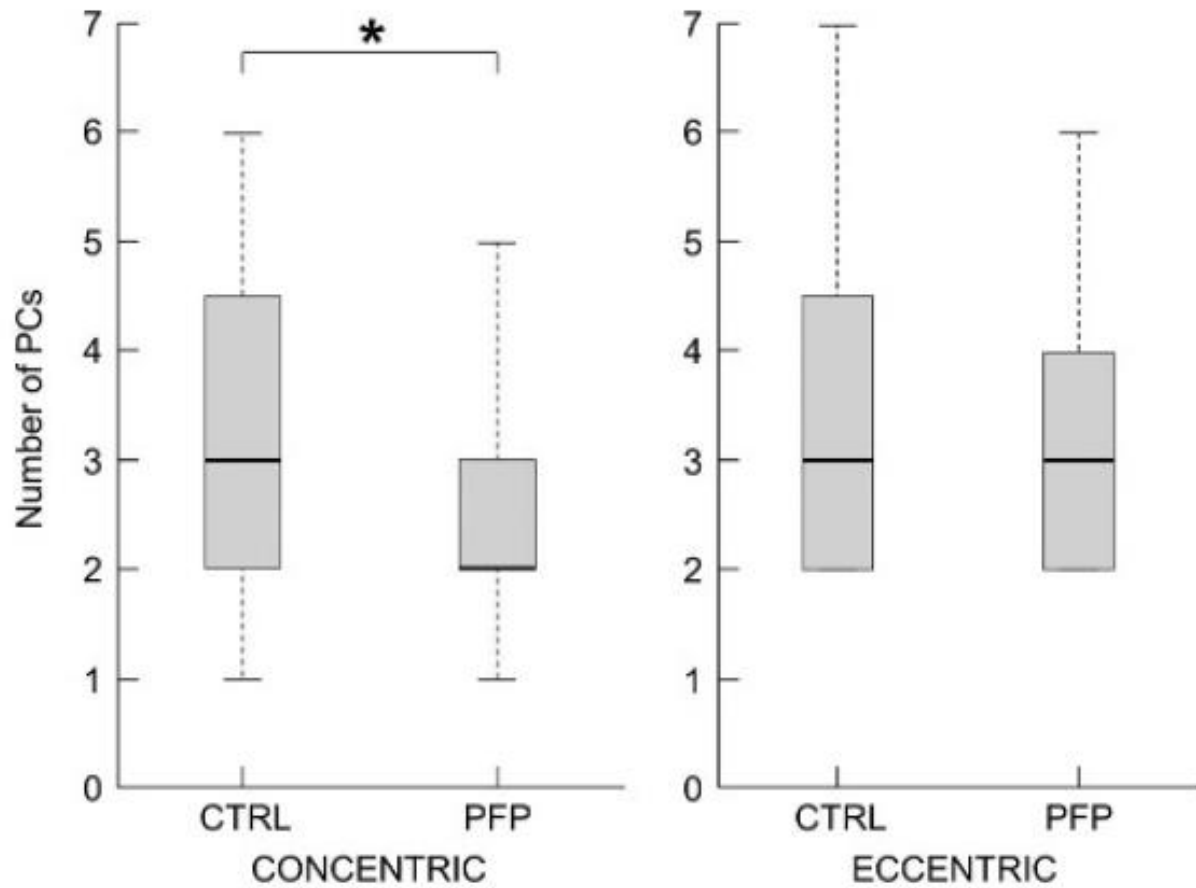
646 negative weights for VM proximally and VL distally. Right: temporal coefficients

647 calculated from the same three repetitions on the left, and average temporal coefficients
 648 calculated over 10 repetitions, separately for concentric and eccentric phase. Inspection
 649 of coefficients suggests that expression of PC1_{General activation} increases towards the end
 650 of concentric motion and beginning of eccentric motion. The converse is shown for
 651 PC2_{Vastus-specific activation} (and PC3_{Vasti co-activation} to a lesser extent); lower towards end of
 652 concentric and beginning of eccentric. Some differences were observed between
 653 phases and groups, when analyses are appropriate (i.e. PC1_{General activation} and PC2_{Vastus-}
 654 _{specific activation} which both had no difference in spatial coefficients between groups).



655
 656 Figure 3: Example of PCA analysis of high-density EMG signals for a participant with
 657 PFP. Left: 8 of the 128 EMG envelopes used for the PCA from 3 repetitions of a control

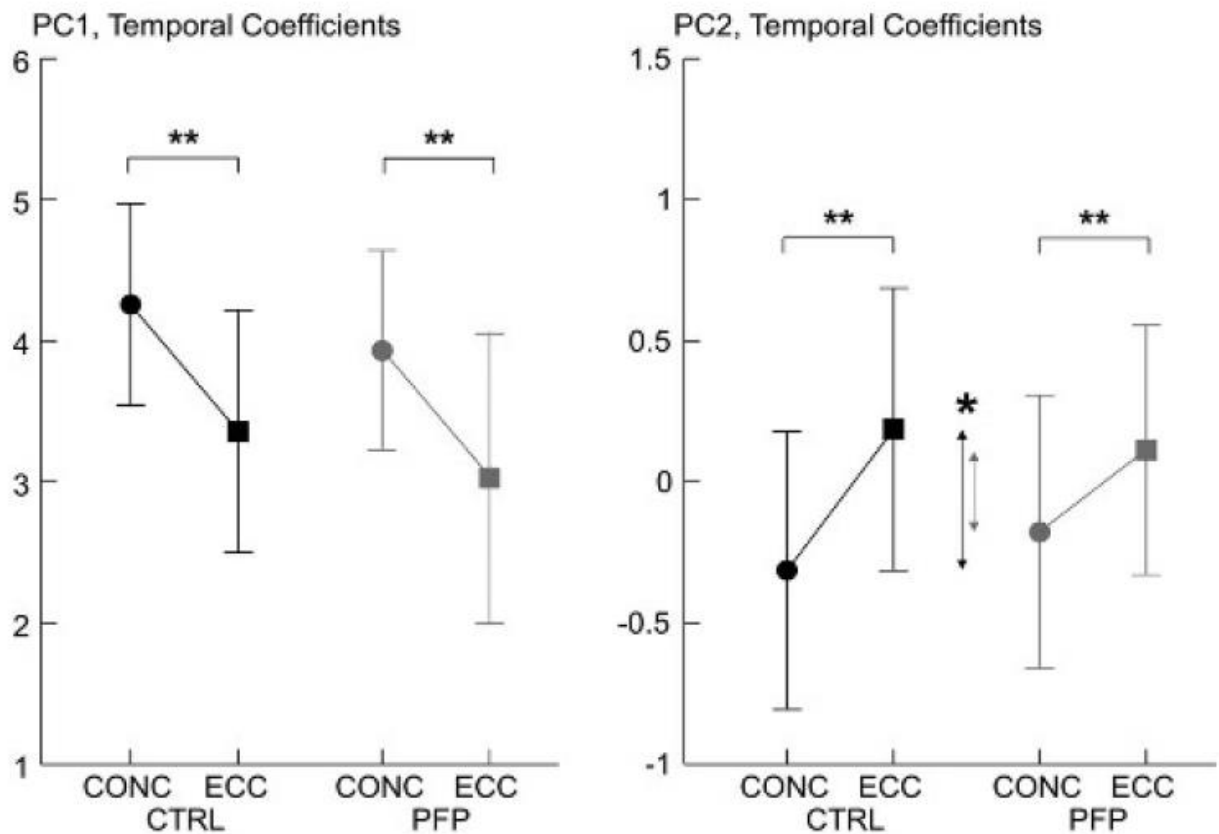
658 participant; light and dark gray boxes identify the concentric (C) and eccentric (E) phase
659 of the movement (10-90°). Middle: spatial weights of PC1-4 (from PCA using data for all
660 the participants with PFP); light and dark shades identify positive and negative weights
661 respectively. Weights for PCs are similar to that for control participants (see Fig. 2),
662 except, unlike controls, PC3_{Within-VL activation} and PC4_{Within-VM activation} reflect activity of single
663 muscles rather than a pattern of coordination between muscles (no regional variation in
664 VM in PC3_{Within-VL activation} or VL in PC4_{Within-VM activation}). Right: temporal coefficients
665 calculated from the 3 repetitions on the left, and average temporal coefficients
666 calculated over 10 repetitions, separately for concentric and eccentric phase. Only
667 temporal coefficients for PC1_{General activation} and PC2_{Vastus-specific activation} were compared
668 between groups for both control and PFP groups.



669

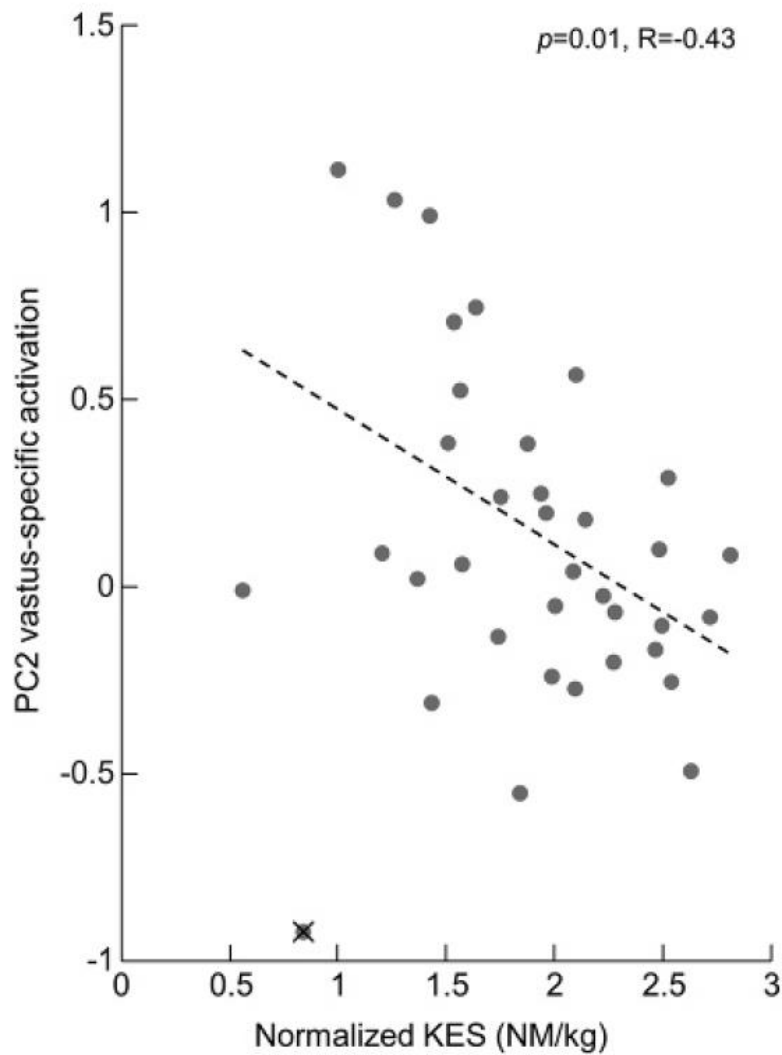
670 Figure 4: Comparison between the minimum number of PCs that explains at least 90%
 671 of the variance in the concentric (left) or eccentric (right) phase of the movement. *

672 $p < 0.05$



673

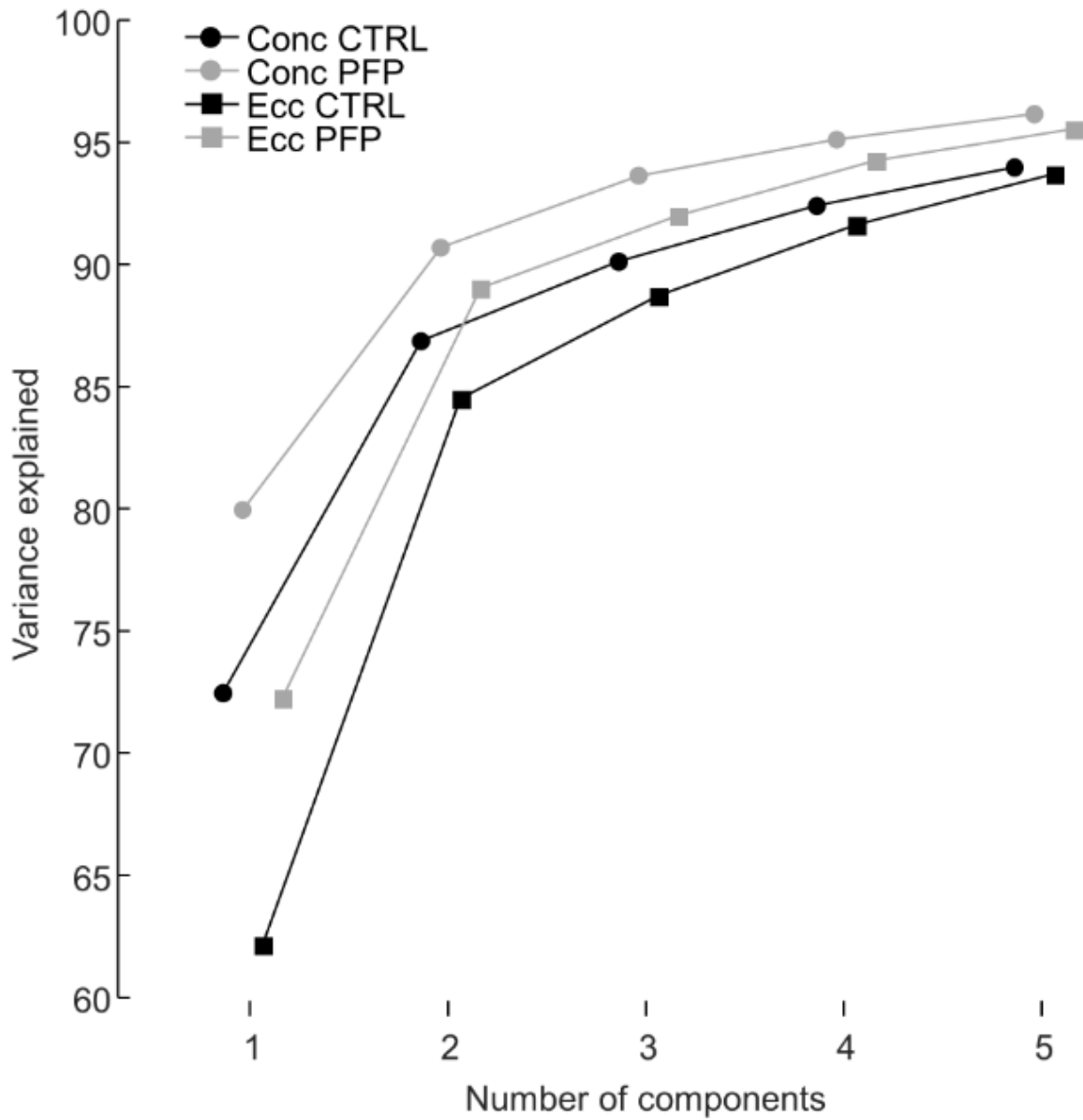
674 Figure 5: Comparison of mean temporal coefficients of PC1_{General activation} (left)
 675 and PC2_{Vastus-specific activation} (right). The contribution of PC1_{General activation} was larger during
 676 the concentric than the eccentric phase of the movement, regardless of the group. For
 677 both groups, PC2_{Vastus-specific activation} was negative (prevalent VL activation) in the
 678 concentric and positive (prevalent VM activation) in the eccentric phase of the
 679 movement; however, this redistribution was smaller in the PFP than in control
 680 participants (interaction effect identified by the arrows). * $p < 0.05$; ** $p < 0.01$



681

682 Figure 6: Scatter plot of KES and PC2_{vastus-specific activation} (eccentric phase) in females with
 683 PFP; higher values indicate preferential VM activation during the eccentric phase of the
 684 contraction. The data point of the participant excluded from this analysis was crossed.
 685 Spearman R identified a moderate inverse correlation between the two variables.

686



687
 688 Figure S1: Variance explained by different number of PCs. Gray and black lines identify
 689 participants with and without PFP. Circles and squares identify concentric and eccentric
 690 phases of the movement.