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Neuromotor Control During Stair Ambulation in Individuals with Patellofemoral Osteoarthritis Compared to Asymptomatic Controls

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Highlights:

- Vastii activation duration is longer during stair descent in people with PFOA.
- Vastus lateralis onset is earlier for ascent and descent.
- Soleus onset is earlier during descent.
- Gluteus maximus activation duration is shorter during stair ascent.
- Clinical assessment of gluteal and quadriceps function may be important in PFOA.

1. Introduction:

Knee osteoarthritis (OA) affects up to 25% of people aged 40 to 50 years with knee pain [1], and approximately 50% of people over 60 years of age [2]. People with knee OA have significantly poorer quality of life compared with age matched, healthy controls [3]. There is currently no cure for knee OA and those with end-stage disease typically undergo total knee replacement with the number of total knee replacement procedures performed yearly expected to increase by 670%, by the year 2030 [4].

In people with knee pain or radiographic OA, 50% have patellofemoral (PF) involvement [5]. Compared to tibiofemoral (TF) OA, isolated PFOA is associated with greater pain and functional limitations during activities of daily living [6]. Further the presence of isolated symptomatic PFOA is a marker for subsequent development

and progression of TFOA [7]. Despite the prevalence and impact of PFOA, few studies have considered biomechanical contributors to its etiology, and there remains limited evidence for treatments to target specific biomechanical impairments.

Individuals with PFOA regularly report discomfort during activities that involve knee flexion, such as prolonged sitting, squatting, and stair ambulation [8]. Not surprisingly, individuals with PFOA ascend and descend stairs with altered kinematics (e.g. increased anterior pelvic tilt, smaller knee flexion angles) and lower PF joint forces compared to age- and sex-matched controls [9]. However, muscle activation strategies and dynamic stability during stair ambulation have not been described in this population.

If individuals with PFOA have altered activation of lower limb muscles during tasks such as stair ambulation, optimizing muscle recruitment strategies may be a potential target for clinical interventions aimed at improving pain and function, potentially delaying or reducing disease progression.

The purpose of this study was to determine if lower limb neuromotor control is different in individuals with PFOA compared to asymptomatic controls when performing stair ascent and descent tasks.

2. Methods

Study design: Cross-sectional

Participant recruitment: Participants with PFOA were recruited as part of a pilot randomized controlled trial (RCT) investigating the effects of footwear and foot orthoses on pain and function. Full details of the recruitment procedures have been published elsewhere [10]. Inclusion and exclusion criteria are described in Table 1. Briefly, the first 23 people with PFOA recruited into the RCT were invited to have electromyographic recordings taken of 14 lower limb muscles while stepping up and down on a single step. All participants provided written informed consent. This study was approved by The University of Queensland's Medical Research Ethics Committee (approval number: 2014000068).

Insert Table 1

Quality of life and physical activity questionnaires: As general measures of health status and physical activity levels, participants in both groups were asked to complete the EQ-5D questionnaire [11] and the International Physical Activity

Questionnaire (IPAQ) [12]. The IPAQ was scored as a continuous measure, expressed as metabolic equivalent of task (MET) minutes per week.

Force plate data: A 17cm high platform, with two force plates at the bottom (Kistler, Switzerland) and a single force plate on the top (Bertec, Ohio, USA) was used to record ground reaction forces during the stepping tasks. Taped markings on the force plates were used to standardise starting position (Figure 1A and 1B). Force plate data were sampled at 2048Hz using Spike2 software (Cambridge Electronic Design Ltd, UK).

Electromyography (EMG): Neuromotor control of the lower limbs was assessed during stair ascent and descent tasks. In the PFOA group, approximately 68% of participants reported bilateral symptoms, thus stepping with the *most painful side* first was chosen as the task of interest. In the control group, stepping with the *dominant side* was chosen [13], equating to ~60% of right limbs being tested in both populations (see Table 1). Surface EMG signals were recorded from the following muscles: gluteus maximus (GMax), gluteus medius (GMed), medial hamstrings (MH), lateral hamstrings (LH), vastus medialis (VM), vastus lateralis (VL), medial gastrocnemius (MG), lateral gastrocnemius (LG), and soleus (Sol). All sites were shaved, abraded and cleaned with alcohol. Electrode placement was according to standardised protocols (Ambu[®] BlueSensor bipolar electrodes, inter-electrode distance 20mm, www.seniam.org). Electromyographic recordings were acquired using a 16 channel TMSi Porti Device (TMSi, Netherlands), sampled at 2048 Hz

(PortiLab, TMSi, Netherlands). The small, portable EMG base-unit, was held by participants in front of their abdomen, controlling arm position during the tasks.

Experimental Task: Participants completed 12 repetitions of both the stair ascent and descent tasks. A light positioned ~ 1.5 m in front of participants, at ground level, indicated when to commence the stepping task. Participants were instructed to step immediately in response to the light stimuli. Speed of response was not assessed, but if a significant delay in response occurred, or the incorrect limb was used to perform the stepping task, the trial was repeated.

For the stair ascent task, participants stood with one foot on each of the lower force plates, 6 cm from front of the force plate and 16 cm apart. Participant were instructed to step up onto the platform (leading with their test limb), pause to regain balance, then step back down on to the bottom force plates. For the stair descent task, participants stood with both feet positioned 4cm from the front of the top force plate and 16 cm apart. When indicated by the light stimulus, participants stepped down on to the bottom force plates (leading with their test limb), paused to regain balance, then returned to the top plate. The return stepping data were not assessed.

Data Processing: EMG and force plate data were synchronized via a 5V TTL pulse, generated by Spike2 software, and delivered with each light stimulus to both recording systems. Data were extracted from both recording systems and analysed using custom written Matlab software (R2017a). Force plate data were low pass

filtered at 20Hz using a second-order Butterworth filter. Onset of the anticipatory postural adjustment (APA) was detected manually from the center of pressure signal, for each trial for each participant, considering medial-lateral displacement only (Figure 1). The peak APA (i.e. peak medial-lateral displacement within approximately 500ms after onset of APA) was detected automatically, then manually checked for each trial. Center of pressure data were also used to determine the timing of the start and end of single-leg stance phase for the most painful (or dominant) leg i.e. timing of the first foot down relative to APA onset, and timing of the second foot down relative to APA onset.

Insert Figure 1

EMG data were bandpass filtered at 20-500Hz using a second-order Butterworth filter. EMG epochs of 10ms root-mean-squared were calculated over the length of the recording for each muscle. For each step, a threshold for a muscle

to be considered “active” was calculated as the mean+2 standard deviations of that recorded during quiet standing (Figure 1), from a 500ms window prior to APA onset. Epochs that exceeded this threshold during the stance phase of the painful limb were considered “active”. The “% muscle activation” ratio was calculated as the number of active epochs divided by the total number of epochs for the stance phase of the stepping tasks.

Synchronisation between equipment was insufficient to enable analysis of EMG data from 5 individuals with PFOA and 5 control participants. As such, force plate data are reported from all participants in each group, and EMG data are reported from 17 people with PFOA, and 15 controls (Table 1). Force plate data were also analysed when considering only participants with synchronised EMG data, with results consistent with the total group (data not reported).

Statistical Analysis: The distribution of data for each outcome measure was assessed for normality by group using Shapiro-Wilks test. To account for data that was not normally distributed, between-group comparisons were made using Mann-Whitney U Tests. Significance was set at 0.05. Corrections for multiple comparisons were deliberately not made [14]. Instead, effect sizes for non-parametric data were calculated using the Mann-Whitney U statistic and the median difference between groups and interquartile ranges within groups presented. An online calculator (http://psychometrica.de/effect_size.html) was used to calculate Cohens d and the r statistic for between group effects from the Mann-Whitney U statistic. All other

analyses were run in SPSS v 25 (New York, USA). As data were not normally distributed, data are presented as median differences unless otherwise stated.

Results:

Twenty-two people with PFOA and 20 controls were recruited (see Supplementary Table 1 for demographics of the full cohort). For the EMG analyses (primary aim), 17 PFOA and 15 controls had sufficient data; participant characteristics are detailed in Table 2. The PFOA group consisted of 17 females, compared to 12 females in the control group. There were no differences between groups for age, height and weight. Based on the EQ-5D, 50% of the PFOA group reported difficulties performing usual activities compared to 0% in the control group, and 92% of the PFOA group reported *general* pain and discomfort compared to 8% in the control group. Note that the pain experienced by the control group was in a location other than their lower limb. The control group rated their overall health higher than those with PFOA (86% vs. 81%), and the PFOA group reported greater walking MET hours per week than the controls (Table 2).

Force plate data: There were no differences in force plate data for center of pressure (CoP) or timing except for a 70ms (median difference) later peak Fz timing (the time between the light stimulus and the maximum force recorded in the Z plane i.e. the force perpendicular to the force plate) observed in the PFOA group during stair descent ($d = 0.91$, $p = 0.01$, see Table 3).

EMG data: *Stair ascent:* For the stepping leg, VL median onset was ~ 3 ms earlier (median difference) at the initiation of stair ascent in the PFOA group ($p < 0.01$, $d = 1.10$, $r = 0.48$). During single leg stance on the top step, the PFOA group used GMax for a significantly shorter duration (median difference = 11%, $p = 0.05$, $d = 0.76$, $r = 0.35$). *Stair descent:* At initiation of stair descent, VL onset was 1ms earlier ($d = 0.78$, $r = 0.36$, $p = 0.04$), and Sol onset 5ms earlier ($d = 0.77$, $r = 0.36$, $p = 0.04$) in the PFOA group compared to controls (Table 4). During stance phase on the bottom step, there was a longer duration of VM and VL activation in the PFOA group (VM: median difference = 30%, $d = 0.99$, $r = 0.45$, $p = 0.01$; VL: median difference = 42%, $d = 0.88$, $r = 0.40$, $p = 0.02$). While not statistically significant ($p = 0.12$), LH activation duration was 19% shorter (median difference) in the PFOA group ($d = 0.58$, $r = 0.28$). Differences in muscle activation duration are represented in Figure 2.

Figure 2

Table 2

Table 3

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Table 4**Discussion:**

This study compared neuromotor control of lower limb muscles during stair ambulation in people with PFOA and age matched healthy individuals. The PFOA group demonstrated different neuromotor strategies compared to controls. There are a number of potential explanations for these findings, including these strategies reflecting attempts to minimize knee pain on loading of the affected limb. However, as this is one of the first studies to explore neuromotor control in PFOA, there are few studies with which to compare our results.

Both groups demonstrated similar timing and ground reactions forces during stair ambulation (Table 3). The only between-group difference in movement observed was a 70 ms later peak (Fz) force timing relative to APA onset in the PFOA group, compared to controls, when descending onto the most painful leg. This

meant that the peak Fz occurred ~60ms *before* the second foot touched down in the control group, and ~11ms *after* the second foot touched down in the PFOA group. The longer period between APA onset and peak Fz force, and the occurrence of peak Fz after the second foot touchdown in the PFOA group may reflect an attempt to reduce load on the painful limb i.e. they lightly loaded their painful side, then dropped heavily onto the non-painful side, during stair descent. This was the only between-group difference in the relative timing of movement.

There were multiple between-group differences in EMG timing. When comparing muscle onsets at the initiation of stair ascent, the PFOA group demonstrated an earlier onset of VL (3ms), and to a lesser non-significant extent, VM (4ms), as the most painful limb moved to step up onto the top step. Similarly, VL onset was also 1ms earlier in the PFOA group during stair descent. To the authors knowledge, there are no existing studies in isolated PFOA with which to compare our findings. However, we propose that there is likely to be very little clinical relevance of a 1-7ms difference in muscle onset timings between groups during stair descent [15], as this onset difference is well within the margin of error of this measure [15]. Further, a delay in EMG does not provide direct information about a delay in force production [15].

The PFOA group did however demonstrate a significantly longer VL (42%) and VM (30%) activation duration during stair descent. This finding is similar to that observed by Childs et al [16] who found that individuals with mixed compartment knee OA have 1.5 times longer VL activation duration than controls during stair

descent. In PFOA, the quadriceps have been shown to be weaker [17] with smaller cross-sectional area [18], and generate less force during stair ambulation [9].

Therefore, the prolonged duration of activation of the quadriceps, in particular VL, during stair ambulation may relate to a need for greater activation of a muscle with reduced force generating capacity to match the same total muscle force produced.

Considering the PFOA groups peak Fz during step descent occurred after touchdown of the second foot, prolonged quadriceps activation may reflect attempts to minimize anticipated painful knee flexion (kinesiophobia) upon weight acceptance.

During stair ascent, GMax activation during stance phase on the top step was 11% shorter in the PFOA group than the control group ($d = 0.76$). Previous studies have shown that individuals with PFOA have lower hip strength compared to healthy controls [19, 20] and utilize different pelvic and hip kinematics to ascend stairs [9]. However, it is uncertain whether the 11% reduction in GMax activation duration identified in the PFOA group is of a sufficient magnitude to impart any clinical impact.

In the distal limb, a 5ms earlier onset of Sol in the PFOA group was observed in stair descent. Earlier activation of Sol may serve to stabilize anterior translation of the tibia upon initiation of knee flexion in preparation to step, possibly compensating for quadriceps weakness. However, as discussed above, the clinical significance of this difference is uncertain.

Limitations

There are several limitations to this study. First, as this study is cross-sectional in nature, we are unable to infer causality. Second, control participants were not imaged to confirm the absence of radiographic PFOA. However, clinical features alone are now considered sufficient for accurate diagnosis of knee OA [21] and control participants did not report any clinical signs or symptoms of PFOA. Kinematic measures were not available for this study, which limited our ability to determine joint forces. Future studies could benefit from combining kinematic, kinetic and EMG data utilizing a larger sample size to more fully describe altered strategies during stair ambulation and other similar pain inducing tasks such as sit to stand, in people with PFOA.

It was not possible to explore muscle activation based on the severity of structural PFOA as either radiographic or MRI evidence of PFOA was accepted as inclusion criteria. Therefore, not all participants with PFOA had the same baseline imaging for categorization. While previous studies have not found an association with imaging severity and onset timing of the quadriceps in combined TF and PFOA [22], future larger scale studies may benefit from using the same imaging modality to determine if neuromotor control measures are related to radiographic or MRI grade of isolated PFOA.

The findings of this study indicate that those with PFOA ascend and descend stairs with strategies that may be related to attempts to reduce knee flexion, or

result from reduced knee flexion. However, as kinematics were not recorded in this study, further research is required.

Significance

This investigation demonstrated that people with PFOA descend stairs with increased quadriceps activation duration and a delay in peak force timing. These findings may represent attempts to minimize pain in the affected knee. Clinical strengthening interventions for the quadriceps and gluteals may be beneficial to assist with the observed neuromotor strategies. This requires investigation in future studies.

Conclusions

This is the first study to demonstrate that individuals with PFOA ascend and descend stairs with some different neuromotor control strategies compared to aged matched controls. During stair ascent, people with PFOA have an earlier onset of VL, and a shorter activation duration of GMax. During stair descent, earlier VL and Sol onset, and longer VL and VM activation duration in individuals with PFOA was observed. Strengthening interventions for the quadriceps and gluteals may support the observed neuromotor strategies and improve pain and function during stair ambulation in individuals with PF OA.

Author contributions:

NW, KT, KC, NC and BV were involved in the development of the research question.

NW and KT were involved in data collection. NW, KT and RS were involved in data

processing. NW and KT were responsible for data analysis. NW, KT, KC and BV were involved in writing of the original draft. All authors have reviewed, edited and approved the final draft of the paper.

Conflict of Interest Statement:

The authors declare there are no conflicts of interest associated with the project.

Acknowledgments

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Figure 1.

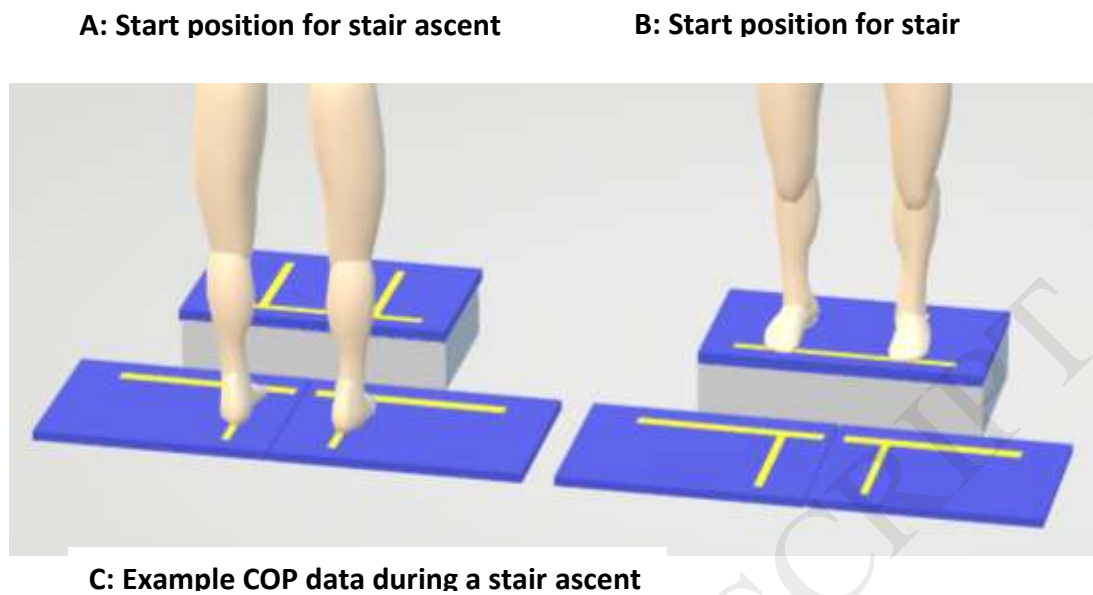


Figure 1. Force plate markings and starting positions for (A) stair ascent and (B) stair descent. (C) Example centre of pressure (COP) data collected from 1 participant during stair ascent. Timing of the light stimulus, onset of the anticipatory postural adjustment (APA), and timing of peak Medial/Lateral APA displacement is shown.

Blue = COP displacement in Medial/Lateral direction; Red = Velocity of COP in Medial/Lateral direction which was used to assist in the determination of APA onset; Green = COP in Anterior/Posterior. Baseline muscle activity (EMG) was determined

from the 50 ms prior to the light trigger which signalled to the participants to begin their movement.

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Figure 2.

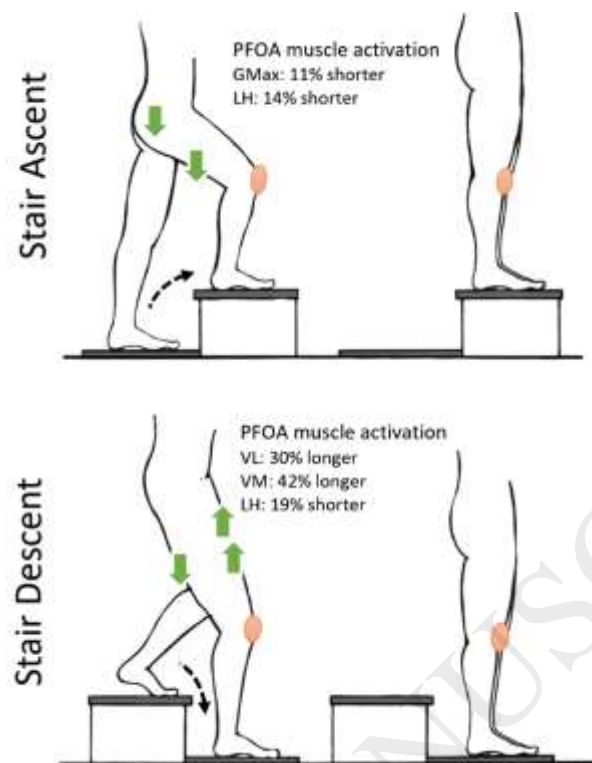


Figure 2. Group mean differences in percentage of muscle activation duration, between people with PFOA (most painful side) and aged-matched controls. Data represents the single-leg stance period on the painful side (or matched leg for controls) during stair ascent (top), and during stair descent (bottom). Differences with an effect size $d \geq 0.50$, which represents a medium effect size or greater, are represented by the green arrows. Movement of the non-painful side is represented by the black arrow. GMax: gluteus maximus; VL: vastus lateralis; VM: vastus medialis; LH: lateral hamstrings.

TABLE 1. Inclusion and exclusion criteria for the PF OA and control groups.

Inclusion Criteria Both Groups
(i) aged \geq 40 years;
Exclusion Criteria Both Groups
(i) current or previous pain in the knee, hip, lumbar spine or foot that had lasted longer than 3 months and/or required intervention;
(ii) foot orthoses use in the last 12 months.
(iii) a history of hip, knee or foot surgery;
(iv) neurological or systemic arthritis conditions;
(v) planned lower limb surgery in the following 2 months;
(vi) physical inability to undertake testing procedures;
(vii) an inability to understand written and spoken English.
Specific Inclusion Criteria PF OA
(i) anterior knee pain aggravated by at least two activities that load the PF joint (e.g. squatting, stair ambulation);
(ii) pain during these activities present on most days in the past month;
(iii) pain severity \geq 30mm on a 100mm visual analogue scale during aggravating activities;
(iv) radiographic (Kellgren and Lawrence \geq grade 1 {Kellgren, 1957 #463}), or MRI evidence of PF OA {Hunter, 2011 #1065}.
Specific Exclusion Criteria PF OA
(i) concomitant pain from other knee structures (including the TF joint), hip or lumbar spine; recent treatment for PF pain (knee injections within the previous 3 months);
(ii) foot orthoses or physiotherapy within the previous 12 months)
(iii) moderate to severe concomitant TF OA (Kellgren and Lawrence grade \geq 3 on radiograph);
(iv) contraindications to x-ray.

Table 2. Participant Demographics

	Participant characteristics for EMG analysis				
Force plate analysis	PFOA: N=17	Control: N=15	Difference	P value	95% CI
Age years	59 ± 10	57 ± 10	1.9	0.59	-9 - 5
Height m	1.67 ± 8.64	1.71 ± 10.80	-3.7	0.28	-3.31 - 10.75
Weight kg	72.5 ± 13.3	72.7 ± 16.08	-0.2	0.97	-10.61 - 11.02
Sex female (%)	12/17 (71%)	8/15 (53%)	18%		
Right side tested (%)	9/17 (53%)	9/15 (60%)	-7%		
EQ-5D (% problems)					
Mobility	25%	0%	2%		
Self-care	0%	0%	0%		
Usual Activities	50%	0%	50%		
Pain & Discomfort	92%	8%	84%		
Anxiety & Depression	8%	0%	8%		
Best Imaginable Health %	81.5 ± 16.5	86.0 ± 11.7	-4.5%	0.46	-8.5 - 17.8
IPAQ					
Walk MET	3005 ± 3435	1045 ± 804	1960	0.06	-3980 - 60
Moderate MET	813 ± 906	600 ± 630	213	0.48	-827 - 402
Vigorous MET	1108 ± 1432	1560 ± 2068	-452	0.52	-967 - 1873
Total MET	4926 ± 3405	3131 ± 2278	1795	0.12	-4075 - 485

Data presented as mean ± standard deviation unless otherwise noted, CI: confidence interval, MET: metabolic equivalent of task minutes, kg: kilograms, cm: centimeter.

Table 3: Force Plate Data

	Stair Ascent				Stair Decent			
	Controls N=20 Median IQR	PFOA N=22 Median IQR	Median Diff. ES d	P valu e	Controls N=20 Median IQR	PFOA N=23 Median IQR	Median Diff. ES d	P value
APA onset ^(ms)	0.73 0.68, 0.76	0.70 0.68, 0.78	0.03 0.16	0.60	0.73 0.70, 0.77	0.74 0.69, 0.81	0.00 0.20	0.51
Peak APA ^(ms)	0.91 0.86, 0.98	0.92 0.87, 1.02	-0.01 0	1.00	1.02 0.98, 1.11	1.06 0.98, 1.18	-0.04 0.19	0.53
1 st Footdown ^(ms)	1.91 1.80, 2.00	1.81 1.73, 1.93	0.10 0.45	0.15	2.00 1.83, 2.13	1.99 1.86, 2.19	0.01 0.1	0.75
2 nd Footdown ^(ms)	2.88 2.80, 3.05	2.70 2.60, 2.98	0.18 0.49	0.12	2.81 2.66, 2.88	2.79 2.53, 2.99	0.02 0.007	0.98

Peak Fz ^(ms)	3.12 1.94, 4.70	4.14 1.80, 4.96	-1.02 0.20	0.51	2.20 1.74, 2.69	2.90 2.41, 4.04	-0.70 0.91*	0.01*
Peak Fz ^(N)	664 576, 865	679 593, 789	-15.35 0.12	0.69	805 648, 939	787 658, 886	17.34 0.06	0.85
Size APA ^(mm)	12.99 8.36, 19.31	13.71 11.03- 17.58	-0.72 0.27	0.39	54.63 44.2- 63.10	57.60 45.07-64.91	-2.97 0.11	0.71
Phase 1: APA onset to 1st foot contact								
ML range	13.00 7.81, 19.36	13.71 11.44, 17.61	-0.71 0.27	0.39	55.27 45.23- 63.10	57.72 45.18, 65.89	-2.45 0.15	0.63
AP range	3.66 1.81, 5.13	3.39 2.44, 5.58	0.27 0.13	0.67	21.70 17.92- 28.19	20.88 19.03, 24.25	0.82 0.1	0.75
ML SD	4.41 2.73, 6.95	4.71 4.09, 6.08	-0.30 0.28	0.36	20.33 16.22- 23.23	21.72 16.66, 23.97	-1.40 0.15	0.63
AP SD	1.06 0.60, 1.62	1.06 0.71, 1.73	0.00 0.16	0.61	7.87 6.07, 10.23	7.36 6.68, 8.79	0.51 0.13	0.68

Path	74.05 52, 103	73.62 67, 97	0.43 0.24	0.45	201 171, 248	224 170, 266	-23.35 0.16	0.61
Phase 2: 1st foot contact to 2nd foot contact								
ML range	22.25 16.71, 30.01	21.34 15.90, 28.07	0.92 0.09	0.76	120.15 95.65- 143.6	109.23 96.19- 136.22	10.91 0.27	0.38
AP range	79.48 70.05, 96.95	87.61 9.06, 99.39	-8.13 0.46	0.14	29.96 20.42-9.21	44.01 24.66, 57.37	-14.05 0.33	0.28
ML SD	5.20 4.20, 6.67	5.28 4.03, 6.72	-0.07 0.05	0.86	33.12 27.21- 40.63	30.37 25.36, 36.87	2.75 0.39	0.21
AP SD	18.29 15.45, 23.46	20.79 8.20, 23.51	-2.50 0.45	0.16	8.02 4.89, 8.73	6.98 4.90, 9.28	1.04 0.03	0.92
Path	163 134, 257	192 160, 24	-28.43 0.37	0.24	225 195, 256	238 186, 291	-12.68 0.23	0.45

IQR: interquartile range; Diff.: difference; ES: effect size; APA: anticipatory postural adjustment; ms: milliseconds; ML: medial to lateral; AP: anterior to posterior, SD: standard deviation. All timings are presented relative to APA onset.

Table 4. EMG Onset and Activation Duration of The Stepping Leg During Stair Ambulation

	Stair Ascent				Stair Descent			
	Controls	PFOA	Median Difference	P value	Controls	PFOA	Median Difference	P value
	Median (IQR)		ES (<i>d</i> , <i>r</i>)		Median (IQR)		ES (<i>d</i> , <i>r</i>)	
Muscle Onset Timing, relative to APA onset (ms)								
GMax	0.04 (0.03, 0.18)	0.05 (0.03, 0.11)	-0.01 (0.007, 0)	0.98	0.10 (0.05, 0.17)	0.06 (0.04, 0.10)	0.04 (0.43, 0.21)	0.25
GMed	0.03 (0.02, 0.08)	0.04 (0.03, 0.08)	-0.01 (0.41, 0.2)	0.28	0.04 (0.02, 0.07)	0.04 (0.02, 0.07)	0.00 (0.22, 0.11)	0.55
MH	0.09 (0.06, 0.19)	0.11 (0.07, 0.13)	-0.02 (0.07, 0.03)	0.86	0.15 (0.10, 0.27)	0.14 (0.09, 0.25)	0.01 (0.20, 0.09)	0.60
LH	0.09 (0.08, 0.15)	0.12 (0.08, 0.19)	-0.03 (0.23, 0.11)	0.54	0.14 (0.10, 0.34)	0.16 (0.07, 0.25)	-0.02 (0.07, 0.03)	0.85
VM	0.12 (0.05, 0.17)	0.07 (0.04, 0.11)	0.05 (0.51, 0.25)	0.17	0.08 (0.04, 0.10)	0.05 (0.04, 0.11)	0.03 (0.007, 0)	1

VL	0.07 (0.05, 0.16)	0.04 (0.03, 0.06)	0.03 (1.1, 0.48)	<0.01*	0.06 (0.04, 0.17)	0.05 (0.02, 0.07)	0.01 (0.78, 0.36)	0.04*
MG	0.08 (0.06, 0.13)	0.08 (0.06, 0.16)	-0.01 (0.29, 0.14)	0.45	0.18 (0.10, 0.28)	0.16 (0.11, 0.24)	0.02 (0.17, 0.08)	0.65
LG	0.07 (0.05, 0.11)	0.07 (0.05, 0.13)	0.00 (0.13, 0.06)	0.74	0.16 (0.11, 0.22)	0.12 (0.08, 0.19)	0.05 (0.29, 0.14)	0.43
Sol	0.08 (0.06, 0.11)	0.08 (0.06, 0.12)	0.00 (0.04, 0)	0.92	0.19 (0.15, 0.24)	0.15 (0.10, 0.18)	0.05 (0.77, 0.36)	0.04*
Duration of Muscle Activation, % of stance phase duration								
GMax	0.77 (0.71, 0.84)	0.66 (0.55, 0.76)	0.11 (0.76, 0.35)	0.05*	0.66 (0.41, 0.79)	0.64 (0.50, 0.79)	0.02 (0.03, 0)	0.94
GMed	0.79 (0.70, 0.83)	0.75 (0.63, 0.81)	0.04 (0.44, 0.21)	0.25	0.74 (0.48, 0.79)	0.77 (0.67, 0.81)	-0.03 (0.33, 0.16)	0.37
MH	0.71 (0.41, 0.94)	0.77 (0.50, 0.87)	-0.06 (0.09, 0.04)	0.83	0.70 (0.52, 0.84)	0.64 (0.37, 0.80)	0.06 (0.42, 0.20)	0.26
LH	0.70 (0.45, 0.91)	0.57 (0.25, 0.79)	0.14 (0.50, 0.24)	0.19	0.58 (0.40, 0.70)	0.40 (0.21, 0.57)	0.19 (0.58, 0.28)	0.12

VM	0.87 (0.69, 0.95)	0.88 (0.81, 0.92)	0.00 (0.07, 0.03)	0.86	0.53 (0.40, 0.65)	0.83 (0.70, 0.89)	-0.30 (0.99, 0.44)	0.01*
VL	0.84 (0.62, 0.99)	0.85 (0.71, 0.93)	-0.01 (0.06, 0.03)	0.89	0.41 (0.27, 0.83)	0.83 (0.59, 0.95)	-0.42 (0.88, 0.40)	0.02*
MG	0.28 (0.18, 0.41)	0.36 (0.18, 0.47)	-0.08 (0.26, 0.13)	0.49	0.63 (0.45, 0.80)	0.57 (0.43, 0.65)	0.06 (0.47, 0.23)	0.20
LG	0.38 (0.24, 0.53)	0.39 (0.30, 0.56)	-0.01 (0.03, 0)	0.95	0.68 (0.53, 0.73)	0.63 (0.49, 0.70)	0.05 (0.25, 0.12)	0.50
Sol	0.24 (0.20, 0.38)	0.29 (0.19, 0.40)	-0.05 (0.26, 0.13)	0.49	0.33 (0.17, 0.47)	0.33 (0.24, 0.46)	0.00 (0.11, 0.05)	0.77

IQR: interquartile range; ES: effect size; ms: milliseconds; GMax: gluteus maximus; GMed: gluteus medius; MH: medial hamstrings; LH: lateral hamstrings; VM: vastus medialis; VL: vastus lateralis; MG: medial gastrocnemius; LG: lateral gastrocnemius; Sol: soleus.