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Hip abductor muscle activity during walking in individuals with gluteal tendinopathy

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ABSTRACT

The external hip adduction moment during walking is greater in individuals with gluteal tendinopathy (GT) than pain-free controls. Although this likely represents a greater demand on the hip abductor muscles implicated in GT, no study has investigated activation of these muscles in GT. For this purpose, fine wire electrodes were inserted into the segments of the gluteus minimus and medius muscles, and surface electrodes placed on the tensor fascia lata, upper gluteus maximus and vastus lateralis muscles of thirteen individuals with, and thirteen without, GT. Participants underwent six walking trials. Individual muscle patterns were compared between groups using a wavelet- based linear effects model and muscle synergy analysis performed using non-negative matrix factorisation to evaluate muscle activation patterns, within- and between-participant variability. Compared to controls, individuals with GT exhibited a more sustained initial burst of the posterior gluteus minimus and middle gluteus medius muscle segments. Two muscle synergies were identified; Synergy-1 activated in early-mid stance and Synergy-2 in early stance. In GT participants, posterior gluteus minimus and posterior gluteus medius and tensor fascia lata contributed more to Synergy-1 active during the period of single leg support. Participants with GT exhibited reduced within-participant variability of posterior gluteus medius and reduced between-participant variability of anterior gluteus minimus and medius and upper gluteus maximus. In conclusion, individuals with GT exhibit modified muscle activation patterns of the hip abductor muscles during walking, with potential relevance for gluteal tendon loading.

Key words

Hip abductor muscle; fine-wire electromyography (EMG); gait; greater trochanteric pain syndrome

INTRODUCTION

Gluteal tendinopathy (GT) is a debilitating, often insidious, cause of lateral hip pain most commonly found in middle aged women (Woodley et al. 2008, Fearon et al. 2014). Symptoms are typically aggravated during walking (Fearon et al. 2014) affecting physical activity levels and with long term consequences for health and well-being (Fearon et al. 2014). Treatments for GT are not yet optimal, likely due to the paucity of studies evaluating impairments in this group. Recent data show the external hip adduction moment during the stance phase of walking gait is greater in people with GT than pain-free controls (Allison et al. 2016). Although this likely represents a greater demand on hip abductor muscles (Winter 1995, Henriksen et al. 2009), no study has investigated activation patterns of these muscles during gait in GT. Alterations in muscle activation might be potentially modifiable targets for intervention for GT.

GT involves tendinopathic change of two primary hip abductor muscles (gluteus medius (GMED) and minimus (GMIN) (Bird et al. 2001, Woodley et al. 2008)) responsible for control of the pelvis with respect to the femur during gait (Al-Hayani 2009, Retchford et al. 2013). Activation of the multiple overlapping hip abductor muscles, which each include multiple segments with distinct mechanical actions, is complex. Fine-wire electromyographic (EMG) recordings of the GMED (Semciw et al. 2013) and GMIN (Semciw et al. 2014) muscles in healthy individuals have identified muscle segments that function independently during gait, evident as differences in timing and amplitude of muscle activity. However some muscle segments and other hip abductor muscles may operate synchronously for synergistic and/or complementary roles. Such sophisticated motor control may be altered in the presence of tendinopathic changes and hip abductor weakness (Woodley et al. 2008, Allison et al. 2016). Activation of other hip abductor muscles which generate force via insertions into the iliotibial band (ITB) (i.e. tensor fascia lata (TFL), upper gluteus maximus (UGM, and vastus lateralis (VL) (Al-Hayani 2009, Stecco et al. 2013)), may also be modified and would be relevant as ITB tension increases compressive forces against the greater trochanter (Birnbaum and Pandorf 2011) into which the gluteal tendons insert (Al-Hayani 2009). Changes in activation of the complex of hip abductor muscles may modify tension transmitted by the gluteal tendons and/or

applied compression with relevance for GT (Cook and Purdam 2011, Grimaldi et al. 2015).

Thorough investigation of muscle activation patterns during cyclic tasks such as walking requires assessment of both *timing* and *amplitude* (which can be performed using wavelet EMG analysis (McKay et al. 2013)) and synergy analysis which can provide insight into *muscle coordination* (Ivanenko et al. 2006, Hug 2011). These complimentary analysis methods can also provide relevant information about within- and between-person *variability*; thought to be a hallmark feature of flexibility of joint coordination during gait in healthy individuals (Hamill et al. 1999, Heiderscheit 2000). Low variability of kinematics (Heiderscheit 2000, Diamond et al. 2015) and muscle activation (Diamond et al. 2016) during walking have been associated with other lower limb conditions, and may be relevant to GT.

This study aimed to investigate the EMG patterns of the hip abductor muscle complex during walking in individuals with and without GT, using wavelet analysis of individual muscle patterns, and muscle synergy analyses to evaluate muscle activation patterns, variability and contribution of muscles to each synergy. We hypothesized that GT would be characterised by altered patterns of activation, and reduced variability during gait.

METHODS

Sample size calculation

This was an exploratory study inclusive of invasive methodology, with no comparative studies to derive a sample size for a priori. Previous studies using fine wire EMG and similar methodology have reported significant results with sample sizes of 8-12 participants (Park et al. 2012, Semciw et al. 2014).

Participants

Eight individuals (5 females) with a clinical, and magnetic resonance imaging (MRI) diagnosis (Blankenbaker et al. 2008), of GT [mean (SD) age 54(10) years; height 166(1) cm; mass 67(15) kg] and eight age-comparable (5 females) healthy controls

[51(10) years; 168(1) cm; 72(15) kg] were recruited. Data from 10 additional participants could not be included as EMG from ≥1 muscle was corrupted by artifact; signals from all muscles are required for synergy analysis. Inclusion criteria for the GT group were a primary clinical diagnosis of GT defined as: a primary complaint of lateral hip pain at the greater trochanter, for ≥3 months, at an intensity of ≥4 on an 11-point numerical rating scale (NRS) ['0'- no pain; '10'- worst pain imaginable]; reproduction of pain (≥4/10) during examination with palpation of the greater trochanter (Woodley et al. 2008, Fearon et al. 2013) and ≥1 of six pain provocative clinical tests designed to impart a compressive or tensile load through the gluteal tendons (Grimaldi et al. 2015) and a primary MRI diagnosis of GT as per the criterion of Blankenbaker et al., (2008). Exclusion criteria were: radiological evidence of hip osteoarthritis (Kellgren and Lawrence Grade ≥2 (Kellgren and Lawrence 1957)), Body Mass Index >36kg/m^2, low back pain or reproduction of symptoms with lumbar active range of motion, and other musculoskeletal or neurological conditions that could affect gait. Control participants were free of neurological and leg/lumbar musculoskeletal conditions. The institutional Human Research Ethics Committee approved the study, participants provided written informed consent.

Instrumentation

The 'affected' hip for GT participants and a 'test' hip selected by coin-toss for controls were tested. Bipolar fine-wire electrodes were fabricated from two strands of Teflon-coated stainless steel wire (75µm, A-M Systems, USA) threaded into a hypodermic needle (22Gx3$^1/_2$"; Terumo, Japan) with 1 mm of Teflon removed, and bent back 1 and 3 mm to form hooks. Electrodes were inserted into anterior (AGMED), middle (MGMED) and posterior (PGMED) GMED, and anterior (AGMIN) and posterior (PGMIN) GMIN, according to Semciw et al. (2012) (with the exception of AGMED where the electrode was inserted into the deepest part of the muscle segment); with guidance by ultrasound imaging (DP-660; Mindray Medical, China). Surface electrodes (10mm disposable electrodes; Covidien, Ireland) were placed over TFL, UGM and VL, approximately in parallel with the muscle fibres (inter-electrode distance-20mm)(Cram et al. 1998). EMG signals were amplified 500 times (common mode rejection ratio > 100dB, input impedance > 100 Mohm), band-

pass filtered (10-1000 Hz), wireless-transmitted via Noraxon Telemyo 2400 DTS system (Noraxon, USA), output via DTS Analog Module, and digitized by Vicon (Vicon, UK) 16-bit analog-to-digital converter at 3000 Hz. A fixed 312ms wireless transmission delay was post-corrected in software.

Experimental protocol

Participants performed six walking trials along a walkway (three each direction) at self-selected speed at the university motion analysis laboratory (Melbourne, Australia). Ground reaction force data were digitized at 3000 Hz using two floor-mounted OR6-6 force plates (Advanced Mechanical Technology, USA) to determine heel-strike and toe-off. Participants rated pain experienced during the task, from GT or intramuscular electrodes, on the NRS.

EMG data analysis

Pre-processing

All processing was performed in Matlab (Mathworks, USA). Surface and intramuscular EMG were digitally filtered using a zero-lag fourth-order Butterworth filter between 50-500 Hz and 50-1400 Hz respectively, then full-wave rectified, and low-pass filtered at 8 Hz, in accordance with recent recommendations (Hug 2011). From the six walking trials, five complete stride cycles without artifact were selected from each participant. As performed in previous studies which extracted muscle synergies, EMG envelopes were normalized to the average of the peak amplitudes of each muscle across the five strides (Hug 2011, van den Hoorn et al. 2015) to (1) ensure each muscle has an equal variance within the synergy analysis and (2) as normalization to maximal or sub-maximal tasks has methodological limitations in populations with pain and weakness (Branch et al. 1989). Each stride was time-normalized to 100 samples (stance: 60 samples; swing: 40).

Group comparison of individual muscle EMG patterns

To identify differences in EMG patterns of individual muscles, the wavelet-based linear based mixed effect (LME) model was used to statistically compare the shape of

EMG patterns between groups, based on the technique of wavelet functional ANOVA (wfANOVA) (McKay et al. 2013). The advantage of transforming EMG envelopes into the wavelet domain is that temporally localized features are represented by a small number of orthogonal (independent) wavelet coefficients rather than many correlated time samples, while preserving the structure of the original signals (Angelini and Vidakovic 2003). EMG envelopes from each participant (5 strides/participant) were expressed in the wavelet domain using third-order coiflets and analysed in Matlab (Matlab Wavelet Toolbox, Statistics and Machine Learning Toolbox) using Matlab code from McKay et al. (2013), yielding a signal with the same sample number as the original EMG waveform. The resultant wavelet coefficients (100 samples) for each muscle were compared between groups using a LME model to identify significant group contrasts. For visualization and interpretation, these were transformed back to the time domain (**Figure 1**).

Intra-participant variability of individual muscle EMG patterns

For each participant, the cross-correlation was calculated between EMG envelopes of the 10 possible pair-wise combinations of the 5 strides. Cross-correlation coefficients (zero time lag) were Z-transformed (Hug 2011), averaged across all pair-wise combinations, and used to assess similarity of EMG patterns across the 5 strides. Average amplitude of each EMG envelope was calculated over each of the ten 10-percentile-wide time windows for the gait cycle, and the coefficient of variation (CV=SD/mean) across the five strides calculated for each time window. Similar EMG envelopes across strides would result in a small CV of averaged EMG windows.

Muscle synergies

Muscle synergies are groups of synchronously-activated muscles, proposed to reflect a simplified neural control strategy (Ivanenko et al. 2004, Cappellini et al. 2006). That is, if amplitude of activation of two or more muscles is modulated in a similar pattern over time they are deemed to act in synergy.

For muscle synergy analysis, non-negative matrix factorization was performed on the pre-processed EMG dataset using the algorithm described by Lee and Seung (2001).

Briefly, the matrix of EMG signals was factorized into two components: "muscle synergy vectors" (relative weightings of muscles within each synergy), and "synergy activation coefficients" (activation pattern of each synergy across the gait cycle) (**Figure 2**). Muscle synergies were extracted for each group from an 8-participant-row x 4000-column EMG matrix containing the five stride cycles of all 8 participants (i.e. 100 samples/stride x 5 strides x 8 muscles=4000 columns). Analysis was iterated by varying the number of synergies between 1 and 8 (i.e. number of recorded muscles), and the total Variance Accounted For (VAF) calculated (Hug 2011). Two synergies were selected for analysis, as addition of a third muscle synergy increased the VAF by <4%.

Cross validation of muscle synergies for between-participant variability

The same method of non-negative matrix factorization was used to extract muscle synergies and corresponding VAF from each participant (i.e. initial 4000-column EMG matrix). Similarity of muscle synergies was assessed as previously described (Torres-Oviedo and Ting 2007, Hug et al. 2011, Frère and Hug 2012). Briefly, synergy vectors matrices were extracted from each control participant and used to reconstruct the EMG patterns of the other participants in the control and GT groups (8x7=56 and 8x8=64 pair-wise comparisons, respectively). The procedure was repeated using each GT participant as reference. Total VAF was calculated to quantify the success to reconstruct the original EMG patterns. With this procedure, strong pair-wise similarity in synergy vectors result in large cross-validation VAF values.

Statistical analysis

The wavelet-based LME technique was used to compare synergy coefficients between groups. All other statistical analyses were performed in Stata (StataCorp, USA). Data were normally distributed and parametric tests were used. Intra-participant variability (CV of EMG envelopes across strides) was compared between groups (Control, GT; between-participant factor) and time window (1 to 10; within-participant factor) using a 2-way-ANOVA. Duncan's Multiple Range test was used for post-hoc testing. Unpaired t-tests were used to assess group differences in pain scores and one-tailed t-tests to evaluate synergy cross-correlation coefficients, individual VAF, and cross-

validation VAF, based on our hypothesis that individuals with GT would show lower variability than controls. Significance was set at P=0.05.

RESULTS

Pain during walking

Participants with GT reported higher pain during walking than controls, although some discomfort related to the intramuscular electrodes was reported by controls (mean(SD) GT: 5.1(2.4), controls: 2.1(2.2) t=2.61, P=0.02).

Group comparison of individual EMG patterns

Significant group contrasts in EMG patterns were identified by the LME model (**Figure 1**). During early-mid stance, participants with GT exhibited sustained bursts of PGMIN and MGMED, with greater peak-normalized EMG amplitude at mid stance than controls, demonstrated by positive contrasts (**Figure 1(c)**). During terminal stance and swing, where EMG activity of all muscles was low, negative significant contrasts revealed lower peak-normalized TFL EMG amplitude in GT participants. Peak-normalisation of EMG precludes interpretation of lower EMG. Instead, activity during terminal stance/swing in the GT group was more different from the peak than for controls and could imply greater peak activity during stance in those with GT. Cross-correlation coefficient of PGMIN was higher in GT participants than controls (t=-2.1,P=0.03), indicating lower stride-to-stride variability of EMG patterns within-participants. The CV of PGMIN EMG amplitude was lower in GT than control participants in the second 10-percentile time window (interaction: group×time-$F_{9,63}$=2.99, P=0.005, post-hoc P=0.004), indicating lower within-participant stride-to-stride variation of EMG amplitude in GT participants during early-mid stance (weight acceptance).

Muscle synergies

Figure 2 depicts Synergy-1 and Synergy-2 extracted for each group. VAF was 87.7% and 87.6% for the Control and GT group respectively, using two synergies. When

synergies were extracted for each participant, mean VAF was 93.2(0.5)% and 92.3(0.7)% (t=1.0;*P*=0.33) for the control and GT participants, respectively. In controls, Synergy-1 primarily involved activation of the anterior gluteal muscles (AGMIN, AGMED) and TFL, during stance (**Figure 2**). Unlike controls, Synergy-1 for participants with GT included PGMED, TFL to a greater extent and a large contribution from PGMIN. Although no statistical analysis was performed, TFL appeared to contribute more to Synergy-1 than Synergy-2 in the GT group, which contrasts with controls where TFL was similarly represented in both synergies. Synergy-2 was composed mostly by PGMIN, MGMED, PGMED, TFL, UGM, and VL, mainly in early stance, but with smaller contributions from PGMIN, PGMED, and TFL in the GT group.

Group differences in synergy coefficients identified by the LME model (**Figure 3**) show differences in the first half of stance, which was also the time of the positive individual muscle contrasts in PGMIN and MGMED in GT participants (**Figure 1**). MGMED did not differ across synergies for the two groups. However PGMIN was more apparent in GT participants in Synergy-1 (in Synergy-2 it was more apparent in controls).

When individual synergy vectors were used to reconstruct the EMG patterns of the other participants (**Table 1**), the VAF of AGMIN, AGMED, and UGM was higher for GT participants than controls. This suggests the synergy vectors were more consistent among GT participants than controls (i.e. greater between-participant variation in controls).

DISCUSSION

These results show that individuals with GT use patterns of hip muscle activation during walking that differ from pain-free controls, with three main observations with potential relevance for gluteal tendon pathology. First, analysis of individual-muscle EMG patterns show more sustained burst activity of PGMIN and MGMED extending into mid-stance, a period when activity of these muscles reduces in controls. Second, muscle activation patterns were less variable within and between GT participants. Third, relative to controls, in GT participants the PGMIN muscle (PGMED and TFL

to a lesser extent) contributed more to the synergy that was activated throughout stance phase including the period of single leg support (Synergy-1). These differences may have implications for loading of the gluteal tendons.

Interpretation of individual muscle EMG patterns

Consistent with previous research (Winter and Yack 1987, Gottschalk et al. 1989, Semciw et al. 2013, Semciw et al. 2014), GMIN, GMED and TFL EMG envelopes included two activity bursts during the stance phase of walking gait: one during early stance and a second, typically smaller, during mid-stance. In healthy controls, PGMED and MGMED demonstrate earlier peak activation than the anterior muscle segment (AGMED) (Semciw et al. 2013) and relative peak activation of PGMIN is greater than AGMIN during early stance (Semciw et al. 2014), implying independent functional roles of segments within muscles. The dominant contribution to Synergy-1 of AGMIN, AGMED, but not the other segments (PGMIN, MGMED, PGMED), in our controls, supports the suggestion of functional differentiation of the anterior from middle/posterior segments during mid to late stance.

The most striking difference in individual muscle activation patterns in the GT group was an extended initial burst of PGMIN and MGMED relative to that of controls. Recently a sustained initial burst of MGMED during walking has similarly been identified in severe hip osteoarthritis, relative to those without or with less severe osteoarthritis (Rutherford et al. 2015). Extended burst duration in GT may have several explanations. First, prolonged activation might be required to compensate for abductor muscle weakness, as identified in GT (Woodley et al. 2008, Allison et al. 2016), or greater requirement for hip abductor force because of greater external hip adduction moment. Recent data from the cohort from which the present participants were drawn show greater external hip adduction moment during stance phase of walking in GT than controls (Allison et al. 2016). Of the gluteal muscle segments, MGMED, (and of the ITB-tensioners, TFL), has the greatest potential to generate a hip abductor moment during mid-stance (Al-Hayani 2009, Retchford et al. 2013) thus sustained contraction of MGMED (and greater contribution of TFL to Synergy-1), into mid-stance might be required to meet functional demands.

Second, prolonged activation might represent a reaction to pain or a strategy to protect from further pain/injury. Prolonged activation of PGMIN and MGMED into mid-stance coincided with AGMIN and AGMED activation, and thus co-contraction of anterior and posterior components of muscles with antagonistic functions in the sagittal and transverse planes. Antagonist co-contraction in other conditions [e.g. low back pain (van Dieen et al. 2003, Dankaerts W. et al. 2006), anterior cruciate ligament surgery (Telianidis et al. 2014)] is postulated to enhance protection of a painful region (Hodges and Smeets 2015). With respect to GT, co-contraction may represent a reactive and not a protective response to pain, as the resultant tensile load through the gluteal tendons at a submaximal level (as occurs during gait) would be unlikely to reduce pain (Rio et al. 2015).

Third, prolonged activation could imply a lack of 'finesse' in the presence of pathology, secondary to interference by pain on motor output or sensory input (see (Hodges and Smeets 2015) for review). Irrespective of the underlying mechanism for the change in coordination in GT, prolonged GMIN and GMED activation during stance could plausibly explain the development or persistence of GT through cumulative tendon loading.

Inter- and intra-participant variability

Consistent with our hypothesis, participants with GT exhibited less between-participant variability of AGMIN, AGMED and UGM and, less within-participant variability of PGMIN than pain-free controls. Some variability may be beneficial to share load between structures and for exposure to new movement options to drive adaptation (Hamill et al. 1999, Heiderscheit 2000, Turpin et al. 2011). A plausible interpretation of reduced variability in GT participants is that greater constraint is imposed on movement with less scope to vary motor strategies in the presence of pathology and/or weakness. Reduced variability of kinematic patterns has been observed during gait in runners with patellofemoral pain (Hamill et al. 1999), as well as in patients with low back pain (Seay et al. 2011) and femoro-acetabular impingement (FAI) (Diamond et al. 2015). Less within-participant variability of muscle activation of the deep hip muscles has recently been identified in individuals

with FAI during gait (Diamond et al. 2016). The consistent selection of kinematic or neuromuscular strategies within a narrow range (low variability) might represent a compensatory strategy to enable execution of a task with minimal pain or perceived threat (Hamill et al. 1999) or with weaker muscles. As walking commonly provokes GT symptoms (Woodley et al. 2008, Fearon et al. 2014), it is plausible that constrained variability in GT may represent an adaptive strategy to minimize pain.

Interpretation of muscle synergies

Two muscle synergies were identified in each group. Muscle synergies represent a group of muscles acting together and correspond to kinematic/kinetic demands of an activity cycle (Cappellini et al. 2006, Neptune et al. 2009, Turpin et al. 2011). In controls, Synergy-1, which has an initial peak in early stance (corresponding to the period of weight acceptance and peak hip adduction moment) and a second peak into mid-stance (corresponding to the period of single leg support) was predominantly made up of the anterior muscle components (AGMIN, AGMED, TFL). This supports the notion that Synergy-1 is involved in weight acceptance, control of the pelvis relative to femur and anterior support of the hip in single leg support. Synergy-2 is defined by a large initial peak at weight acceptance, dominated by the middle/posterior elements of the gluteal muscles (PGMIN, PGMED, MGMED) plus UGMAX and VL (hip and knee extensor muscles), supporting a role of Synergy-2 in weight acceptance.

Compared to the control group, the key differences to these synergies in the GT group were a greater contribution of PGMIN, and to a lesser extent PGMED and TFL, to Synergy-1. This may represent a disproportionate contribution of the PGMIN, PGMED and TFL to frontal plane pelvic control later in stance in GT.

Clinical implications

Our data from individuals with GT may have several clinical implications. First, the extended burst duration of PGMIN and MGMED, and PGMIN and PGMED contribution to Synergy-1 suggest the gluteal tendons in GT may be under more sustained tensile load during early-mid stance [when the hip reaches peak adduction

angle (Nadeau et al. 2003)] in GT. Also corresponding to this period of stance, greater contribution of TFL to Synergy-1 similarly suggests greater duration of ITB tension and contribution of TFL to frontal plane pelvic control. It is plausible to speculate that prolonged hip abductor muscle activation is required in the presence of hip abductor weakness (Allison et al. 2016) to resist the large external moment associated with GT (Allison et al. 2016), in order to eccentrically control hip adduction and the position of the pelvis in the frontal plane in those with GT. A consequence would be greater duration of gluteal tendon and ITB tensile loading into mid-stance with additional associated potential for increases in compressive force against the greater trochanter. Both compressive and tensile overload are considered relevant for tendinopathy (Docking et al. 2013, Grimaldi and Fearon 2015) and support our first hypothesis. Consistent with our second hypothesis, the lower variation in muscle activation patterns within and between GT participants implies greater constraint of patterns, and perhaps less load sharing across the gluteal tendons.

The cross-sectional study design prohibits definitive conclusions to be drawn to as whether the changes in muscle activation patterns identified here are a cause or consequence of symptomatic GT pathology. Although this permits only speculation, taken with evidence of hip abductor weakness (Allison et al. 2016), the results of this study may indicate that consideration of both motor control and strengthening by clinicians could enhance the management of GT (Grimaldi and Fearon 2015). Strengthening exercises have been shown to reduce co-contraction in those with symptomatic knee OA (Al-Khlaifat et al. 2016) to reduce muscle/joint load, another mechanism by which hip abductor strengthening could be effective in the treatment of GT. As pain may contribute to muscle inhibition and reduced variability with direct relevance for tendon loading, this suggests treatment could be more effective if first directed to pain reduction to ensure an optimal pattern of muscle activation prior to strengthening the injured and weak gluteal muscles. Isometric contractions have been shown to influence pain inhibition pathways in healthy controls (Kosek and Lundberg 2003) and in individuals with patella tendinopathy (Rio et al. 2015). In those with GT, it is proposed that isometric exercises would be best performed in a position of relative abduction (Grimaldi and Fearon 2015) to avoid a provocative position of adduction.

Methodological considerations

For methodological reasons, maximal contractions for EMG-normalization were not performed. A limitation of normalizing to peak EMG is the inability to compare amplitude of activity between muscles and groups. Thus, it is important to acknowledge that EMG envelopes and muscle synergy analysis characterize muscle activation patterns (Disselhorst-Klug et al. 2009, Hug 2011). Our interpretations with respect to tendon loading are based on temporal features (e.g. load duration) rather than magnitude of force, and are presented with caution. Individuals from both groups reported some pain during testing. However, controls reported low levels of pain related to electrodes, consistent with that previously reported (Semciw et al. 2012). Pain reported by the GT group was consistent with GT symptoms (Woodley et al. 2008, Fearon et al. 2014).

PERSPECTIVE

Individuals with GT experience pain and disability during walking (Allison et al. 2016) which can lead to a reduction in activity levels with detrimental effects on health and well-being (Fearon et al. 2014). Previous research has shown that individuals with GT exhibit a greater external hip adduction moment during walking than healthy controls (Allison et al. 2016), reflecting a greater demand on the hip abductor muscles implicated in GT (Bird et al. 2001). Given that the aetiology of tendinopathy is often associated with load (Docking et al. 2013), it is crucial to understand the hip abductor muscle activity during walking in this group. In summary, first, our analyses of EMG patterns in pain-free controls support for independent control of the anterior segments of GMIN and GMED as identified previously (Semciw et al. 2014). Second, activation of hip abductor muscles during gait differed between control and GT groups in a manner that has likely implications for tendon health in GT (i.e. prolonged activation, changes in contribution from muscles which insert into the ITB). Our findings suggest that hip abductor muscle activation patterns may be relevant to the development and/or perpetuation of gluteal tendon pathology in those with GT. However whether these muscle activations are a cause or consequence of GT requires longitudinal studies. Whether treatments that

target hip abductor muscle activation change loading and reduce symptoms of GT remains to be investigated.

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Table 1 Average cross-validation Variance Accounted For (VAF) (%).

Ref Vector	Target	AGMIN	PGMIN	AGMED	MGMED	PGMED	TFL	UGM	VL	TOTAL
Control	Control	76	84	76	80	80	87	82	85	82
	GT	85	85	84	77	78	87	87	83	84
t-test		**0.006***	0.555	**0.005***	0.223	0.564	0.762	**0.003***	0.150	0.132
GT	Control	75	82	81	79	83	86	86	84	82
	GT	86	79	86	76	81	86	89	82	84
t-test		**0.002***	0.285	**0.043***	0.337	0.468	0.651	**0.012***	0.350	0.312

Synergy vectors from each participant were fixed (Ref vector), and the synergy coefficients of the other participants (Target) were calculated by non-negative matrix factorization and used to reconstruct the original EMG envelopes. *P<0.05

FIGURE 1. EMG envelopes (arbitrary units) across strides from all participants of (A) the control group shown in blue (five strides for each participant) and (B) the GT group (pink). The thick black line represents the group average. Individual envelopes were normalized by the average peak of each participant across strides. (C) Group differences in EMG waveforms (contrast, bottom row) show the differences in muscle activation patterns between groups (assessed in the wavelet domain and transformed back into the time domain). The vertical dashed line represents the end of stance phase). The significant contrasts shown suggest a pattern with longer bursts of muscle activity of PGMIN and PGMED for the GT group than controls. AGMIN = anterior gluteus minimus; PGMIN= posterior gluteus minimus; AGMED = anterior gluteus medius; MGMED = middle gluteus medius; PGMED = posterior gluteus medius; TFL = tensor fascia lata; UGM = upper gluteus maximus; VL = vastus lateralis.

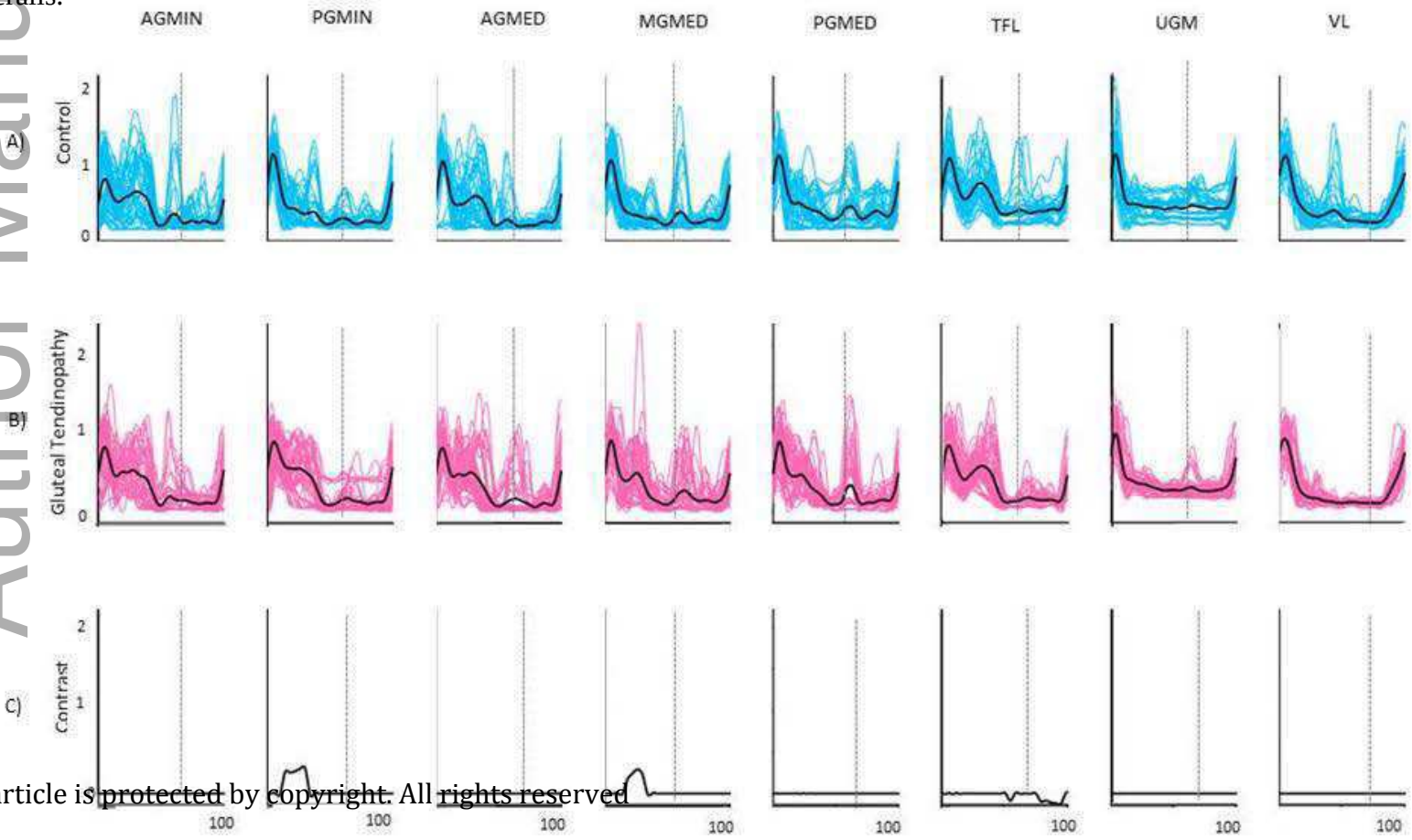

FIGURE 2. Muscle synergies (Groups of muscles acting together). (A) Two muscle synergy activation coefficients (patterns of activation of the muscle synergy) were extracted from each group during gait; coefficient 1 is shown in green, coefficient 2 in yellow. (B) Muscle synergy vectors (relative weighting of the muscles that contribute to a synergy) for each coefficient; Colours represent muscles as per panel; (C) Reconstructed EMG envelopes of each individual muscle, the thin grey line represents the group average original EMG envelopes and the filled colored curves the reconstructed EMG using synergy activation coefficients and vectors for the two chosen synergies (indicating good reconstruction). a.u., arbitrary units.

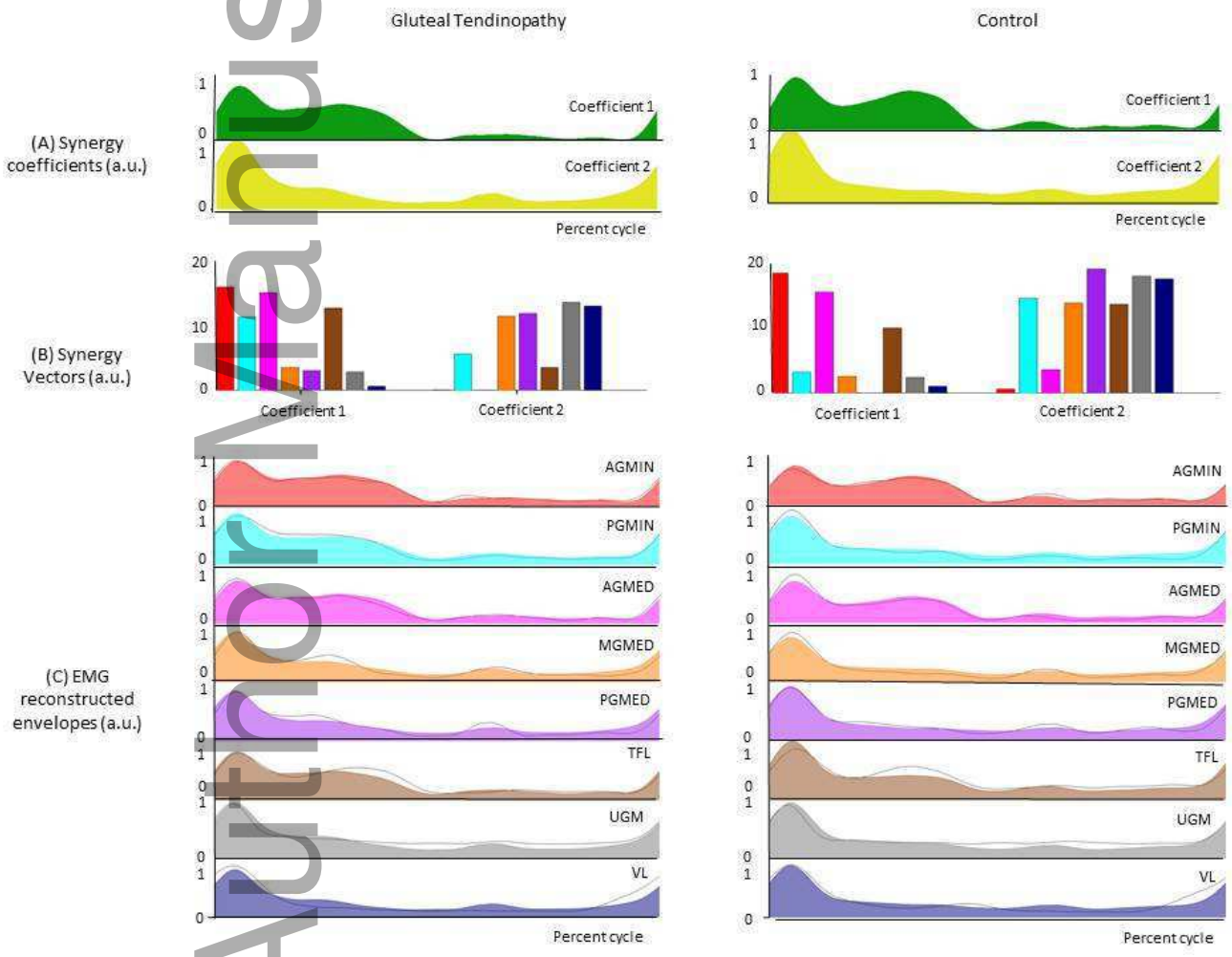

FIGURE 3. (A) Synergy coefficients (patterns of activation of a muscle synergy) across five strides from all participants of the control group (blue) and the GT group (red) (thick line: group average, shaded represents the 95% CI). (B) Group differences (contrasts) in the coefficient waveforms were identified in the wavelet domain and transformed back into the time domain (as shown here). The significant contrasts shown suggest a pattern with longer bursts of muscle activity after heel strike for the GT group than healthy controls. a.u., arbitrary units.