### Accepted Manuscript

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 PII:
 S0268-0033(16)30152-8

 DOI:
 doi:10.1016/j.clinbiomech.2016.10.003

 Reference:
 JCLB 4225

To appear in: Clinical Biomechanics

Received date:31 March 2016Accepted date:4 October 2016



Please cite this article as: Allison, Kim, Vicenzino, Bill, Bennell, Kim L, Wrigley, Tim V, Grimaldi, Alison, Hodges, Paul W., Kinematics and kinetics during stair ascent in individuals with Gluteal Tendinopathy, *Clinical Biomechanics* (2016), doi:10.1016/j.clinbiomech.2016.10.003

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#### Title

Kinematics and kinetics during stair ascent in individuals with Gluteal Tendinopathy

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Word count abstract: 250

Word count main text: 3998

#### Abstract

*Background*: Individuals with gluteal tendinopathy commonly report lateral hip pain and disability during stair ascent. This study aimed to compare kinematics and kinetics between individuals with and without gluteal tendinopathy during a step up task.

*Methods*: 35 individuals with unilateral gluteal tendinopathy and 35 pain-free controls underwent three-dimensional motion analysis of stance phase during stair ascent. An analysis of covariance was performed to compare hip, pelvis and trunk kinematic and kinetic variables between groups. A K-means cluster analysis was performed to identify subgroups from the entire group (n=70) based on the characteristics of the external hip adduction moment. Finally, a Newcombe-Wilson test was performed to evaluate the relationship between group and cluster codes and a 3x2 ANOVA to investigate the differences in kinematics between groups and cluster codes.

*Findings*: Individuals with gluteal tendinopathy exhibited a greater hip adduction moment impulse during stair ascent (ES=0.83), greater internal rotation impulse during the first 50% stance phase (ES=0.63) and greater contralateral trunk lean throughout stance than controls (ranging from ES=0.67-0.93). Three subgroups based on hip adduction moment characteristics were identified. Individuals with GT were 4.5 times more likely to have a hip adduction moment characteristic of a large impulse and greater lateral pelvic translation at heel strike than the subgroup most likely to contain controls.

*Interpretation:* Individuals with GT exhibit greater hip adduction moment impulse and alterations in trunk and pelvic kinematics during stair ascent. Findings provide a basis to consider frontal plane trunk and pelvic control in the management of gluteal tendinopathy.

#### Keywords

Gluteal tendinopathy; kinematics; external hip adduction moment; stair ascent

#### Highlights

- Hip adduction moment is larger during step up in those with gluteal tendinopathy
- Contralateral trunk lean in step up is greater in those with gluteal tendinopathy
- Lateral shift of the pelvis is associated with gluteal tendinopathy in step up
- Addressing step up biomechanics may be relevant for gluteal tendinopathy management

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#### 1. Introduction

Gluteal tendinopathy (GT) is a debilitating, recalcitrant cause of lateral hip pain (Fearon et al., 2014; Woodley et al., 2008) most prevalent in women aged over 40 years (Segal et al., 2007). The condition is associated with moderate to severe pain, disability and reduced quality of life (Fearon et al., 2014); with pain aggravated during everyday activity including walking and stair climbing (Fearon et al., 2012; Segal et al., 2007). Despite provocation of symptoms with stair ascent, no studies have evaluated the kinematics and kinetics during this task in individuals with GT. Analysis of movement patterns is necessary to understand the condition and may guide future studies evaluating conservative strategies for management of GT.

GT involves tendinopathic change of two primary hip abductor muscles, the gluteus minimus and medius (Al-Hayani, 2009; Retchford et al., 2013), at or above their insertion into the greater trochanter (Bird et al., 2001; Kingzett-Taylor et al., 1999; Lequesne et al., 2008a). Similar to other insertional tendinopathies, excessive compressive loads are thought to contribute to the tendon pathology (Almekinders et al., 2003; Benjamin and Ralphs, 1998; Docking et al., 2013). The gluteal tendons are vulnerable to compression against the greater trochanter and iliotibial band (ITB) (Dwek et al., 2005) as the hip moves into adduction and ITB tension increases (Birnbaum and Pandorf, 2011; Birnbaum et al., 2004). Contraction of the muscles that insert into the ITB (i.e. tensor fascia lata (TFL) (Stecco et al., 2013), a hip flexor and abductor (Al-Hayani, 2009; Retchford et al., 2013); gluteus maximus (Stecco et al., 2013) a hip extensor, external rotator and abductor (Retchford et al., 2013); and vastus lateralis (VL), a knee extensor (Becker et al., 2010)) can also augment ITB tension (Stecco et al., 2013), with relevance for the demands for stair climbing. Stair ascent involves greater ranges of hip flexion and adduction than level walking (McFadyen and Winter, 1988; Nadeau et al., 2003; Protopapadaki et al., 2007) and requirement for internal knee extensor moment

generation (Nadeau et al., 2003), but with a similar requirement for internal hip abductor and extensor moment generation (Kirkwood et al., 1999; Nadeau et al., 2003). Recently, a greater external hip adduction moment (HADM) has been reported in individuals with GT during walking (Allison et al., 2016b). This might be exaggerated during the more challenging stair ascent where hip abductor pathology and weakness (Allison et al., 2016a), greater HADM and/or suboptimal control of pelvis on the femur (hip adduction) could all modify loading of the gluteal tendons, with relevance for GT. The aim of this study was to compare kinematics of the hip, pelvis and trunk and the features of the external hip adduction and flexion moment during step up between individuals with and without GT.

#### 2. Methods

#### 2.1 Participants

Thirty-five people with unilateral GT and 35 asymptomatic controls aged 35 to 70 years were recruited from the community over 14-months. Although the groups were comparable in age and gender, the GT group had significantly greater BMI and inter-ASIS width (both P<0.05) (**Table 1**). The median (IQR) values of average and maximum lateral hip pain reported during the last week by GT participants on an 11-point numeric rating scale (NRS) ('0' - no pain; '10' - worst pain imaginable) were 4(1) and 7(1) respectively, but were low during testing (0(2)). Ethical approval was obtained from the institutional Human Research Ethics Committee. All participants provided written informed consent.

For this study, GT was defined clinically (Fearon et al., 2013; Segal et al., 2007; Woodley et al., 2008) with subsequent magnetic resonance imaging (MRI) confirmation of tendon pathology (Blankenbaker et al., 2008). Initial inclusion criteria were the presence of unilateral lateral hip pain (Fearon et al., 2013; Segal et al., 2007; Woodley et al., 2008)  $\geq$  4/10 on the

NRS for  $\geq$  3 months; in the absence of groin, low back or knee pain, known hip or knee osteoarthritis, or any systemic diseases affecting the muscular or nervous systems. Physical screening was performed by a physiotherapist to confirm a primary clinical diagnosis of GT, defined as reproduction of trochanteric pain  $\geq$  4/10 with palpation of the greater trochanter (Fearon et al., 2013; Martin and Sekiya, 2008) and during  $\geq$  1/6 diagnostic clinical tests for GT (Fearon et al., 2013; Grimaldi et al., 2014; Lequesne et al., 2008b) (*Supplementary material*). MRI diagnosis of GT was defined by published classification criteria (Blankenbaker et al., 2008). Exclusion criteria were: (1) clinical or radiological diagnosis of intra-articular hip pathology, the former defined as reproduction of groin pain during passive hip quadrant (Martin et al., 2008; Troelsen et al., 2009) and the latter by evidence of avascular necrosis, bony lesions or evidence of osteoarthritis (Kellegren and Lawrence Grade 2 or above) on plain X-ray and (2) BMI>36kg/m<sup>2</sup> (due to difficulties with skin marker placement for 3D gait analysis).

Control participants were free of any lateral hip or lower limb pain and were recruited to be comparable in age and sex to GT participants. Exclusion criteria were: (1) any hip, lower limb or lumbar pain that interfered with function, walking or that caused the participant to seek treatment in the preceding 12 months; (2) lumbar spine or lower limb surgery in the previous six months; (3) systemic disease affecting the muscular or nervous systems; or (4) BMI>36kg/m<sup>2</sup>.

#### 2.2 Kinematic and kinetic data collection during stair ascent

Participants underwent three-dimensional gait analysis of a step-up task. Twenty seven spherical retro-reflective markers were placed on the lower limbs, pelvis and trunk (Besier et al., 2003). Marker position data were recorded using a twelve camera (MX F20/F40) Vicon

motion capture system (Vicon, Oxford, UK) using Nexus version 1.8.5 at 120Hz. Ground reaction force data were collected at 1200 Hz from a 400 x 600 mm Kistler 9286AA force platform (Kistler, Switzerland) mounted on the first step, and two floor-embedded AMTI OR6-6-2000 force platforms (Advanced Medical Technology, MA, USA). The first step had a height of 240 mm and the second step was a further 200mm above. Location of functional knee joint centers were determined from mean helical axes calculated from 5 squats (Besier et al., 2003). Hip joint centers were determined from the regression equations of Harrington et al. (2007). To balance the statistical model, the hips of control participants were arbitrarily designated as 'symptomatic' and 'asymptomatic' by coin toss and the 'symptomatic' limb studied.

Participants were provided with a demonstration and standardized instructions regarding performance of the step up task. Participants were asked to march on the spot and find their comfortable (natural) standing position with one foot on each force plate (3 cm apart embedded in the laboratory floor). Instruction was then given to walk up the stairs leading with the symptomatic ('test') leg, ending on the top step with feet parallel (**Figure 1**). After demonstrating proficiency with the task (up to two practice trials), three test trials were completed. Participants reported any lateral hip pain experienced during the task on the NRS.

Stance phase of the test leg on the first step was defined using a 20N threshold on the force plate in the first step. Marker trajectory data and ground reaction force data were both low-pass filtered at 6 Hz with a dual-pass 2<sup>nd</sup> order Butterworth filter (Kristianslund et al., 2012). Hip joint adduction-abduction and frontal plane pelvis angles were calculated from the step-up trials using Vicon BodyBuilder software (Besier et al., 2003). Pelvic angles were extracted using a rotation-obliquity-tilt Cardan angle sequence (Baker, 2001). Lateral pelvic

translation in the frontal plane was defined by foot placement relative to the mid pelvis; calculated as the distance between the calcaneal marker and the floor-projected midline, defined by a vertical line from the midpoint between the ASIS markers. This distance was normalized to half the distance between the left and right ASIS, to account for wider bases of support with greater pelvic width (Winter, 1995), and expressed as a percentage. Lateral trunk lean was represented by the frontal plane angle of the trunk segment in relation to the laboratory coordinate system (McFadyen and Winter, 1988). The maximum angles of hip adduction, hip flexion, hip internal rotation, contralateral pelvic drop, lateral pelvic translation and lateral trunk lean were quantified: (1) at foot contact; (2) between foot contact and reciprocal toe off (weight acceptance); and (3) between reciprocal toe off and end of stance (vertical thrust and forward continuance).

Joint moments and positive impulse were calculated from the stance phase on the first step using inverse dynamics using the Vicon BodyBuilder model (UWA model (Besier et al., 2003)) and normalized to body weight times height (Nm/BW.Ht%) to account for body size (Moisio et al., 2003). Positive impulses [Nm.s/(BW.Ht%)] were calculated as the positive-only area under the moment curve, taking into account average magnitude and duration of the positive external moment. In order to evaluate the external moments at the hip, the first-step stance phase of the test limb (foot contact to toe off) was evaluated in two functional phases based on previous studies of the temporal features of stair ascent (McFadyen and Winter, 1988; Zachazewski et al., 1993). These were: (1) vertical thrust constituting the first ~50% of stance - including double support when the trail leg can also contribute to thrust, weight acceptance and the period of single leg support following reciprocal toe off, and (2) forward continuance - including single leg support and double leg support following heel strike of the trailing leg on the step above, equating to second ~50% of stance. According to this

definition, the peak external hip adduction, flexion and internal rotation moments, and their positive impulse, were determined for each trial during 0-50% and 50-100% of stance phase and overall maximums during 0-100% stance, and values for each participant averaged. Secondary analysis was guided by visual inspection of the waveform of each participant. This was undertaken because of an *a priori* prediction, based on clinical observation and previous data of sagittal motion (McFadyen and Winter, 1988) that different strategies may be used by separate subgroups of participants to perform the task.

#### 2.3 Data management and analysis

Data analysis was undertaken using the Statistical Package for the Social Sciences (SPSS) statistical software, version 22 (IBM, New York, USA). All data were explored for normality. Continuous descriptive data for each group were expressed as mean (SD) for normally distributed data, and median and interquartile range (IQR) for non-normally distributed data. Independent t-tests were used to compare the normally distributed data between groups and Mann-Whitney U tests used for non-normal data.

#### 3. Results

Kinetic data were not analysed for one control and three GT participants because of a fault with the force plate.

GT participants completed the first-step stair ascent task with greater stance duration than controls (mean difference = 00.15s; 95% CI 0.07, 0.22; P < 0.001). Significant between group differences were evident in kinetic and kinematic variables in the stance phase of first-step stair ascent (**Table 2 and Figure 2**). Key kinetic differences were: a greater peak HADM moment (mean difference 2.6 Nm/(BW.Ht%) 95%CI 0.8,4.5, P=0.01), greater HADM impulse during stair ascent (mean difference 2.3 Nm.s/BW.Ht(%); 95%CI 0.9, 3.8; P=0.03),

most apparent during the second 50% stance (mean difference 1.9 Nm.s/(BW.Ht%); 95% CI 0.4, 3.4; P=0.01); and greater internal rotation positive impulse during the first 50% stance (mean difference 0.1 Nm.s/(BW.Ht%); 95% CI 0.0, 0.2; P=0.01) for GT participants. With respect to kinematics, individuals with GT demonstrated greater contralateral trunk lean at heel strike (mean difference -3.1 degrees; 95% CI -4.8, 1.4; P=0.001), during heel strike to reciprocal toe off (mean difference -3.1 degrees; 95% CI -4.8, 1.5; P=0.001) and during reciprocal toe off to end of stance (mean difference -2.2 degrees; 95% CI -3.8, -0.6; P=0.01) than controls. Adjusting for pain did not alter the significance of between-group comparisons.

Three distinctive HADM waveforms were identified amongst participants which contributed to the large variability in the direction and magnitude of the HADM during the second 50% stance in the group average ensemble curves (**Figure 2**). Failure to consider these different moment patterns within the group data masked identification of differences. To investigate the prevalence of these subgroups in the GT and control groups, a cluster analysis was performed using the dependent variable HADM impulse during the  $2^{nd}$  50% stance. We considered this feature to be most indicative of the moment pattern differences (**Figure 3a**). A K-means cluster analysis was performed for 3 clusters, with 10 iterations and three final clusters identified in each group (Supplementary material for full details). Ensemble curve averages generated for each cluster validated the characteristics of the HADM waveforms visually identified from the individual participant data (**Figure 3a**). Cluster 1 demonstrated a low HADM impulse during second 50% of stance (less than Clusters 2 and 3, both *P*<0.05); Cluster 2 a high positive HADM impulse during second 50% of stance (greater than Clusters 1 and 3, both *P*<0.05), and Cluster 3 a positive HADM impulse during the second 50% of stance (greater than Cluster 1 and less than Cluster 2; both *P*<0.05) (**Figure 3a & b**). There

were no significant between-group differences in cluster centres within each cluster (**Figure 3b**).

A Chi-square test for independence indicated a significant association between group and cluster code (Pearsons chi square = 7.0, P=0.03). Participants in the GT group were relatively evenly distributed amongst the three clusters: 12 (37.5%) participants in Cluster 2; 11 (34.8%) in Cluster 1; and 9 (28.1%) in Cluster 3. In contrast, most controls were allocated to Cluster 1 (21 [61.7%]), with only 9 (26.5%) and 4 (11.8%) participants in Cluster 3 and Cluster 2, respectively. The Newcombe-Wilson method to compare the incidence of cluster codes in the GT and control groups (**Table 3**), identified individuals with GT were 4.5 times more likely to be Cluster 2 (high HADM impulse second 50% of stance) and less likely (0.32 times) to be Cluster 1 (low positive HADM impulse second 50% of stance). Further, allocation to Cluster 2 increased relative risk of GT by 22% (95%CI -79, -15), whereas Cluster 1 reduced relative risk by 44% (95% CI 4, 68%).

Finally, kinetic variables, kinematics and characteristics of the study sample were compared between Clusters (1, 2 and 3) and groups (GT and control); a 3 x 2 ANOVA and post-hoc LSD test was performed. No significant differences were found in pain, age, height, mass or leg length between clusters (all P>0.05). Significant differences were found in kinetic and kinematic (**Figure 3c**) variables between clusters (Supplementary Table). Notable features were that Cluster 1 demonstrated greater pelvic obliquity at heel strike than Cluster 2 (mean difference 5.0 degrees; 95% CI 2.8-7.3, P<0.001) and Cluster 3 (mean difference 2.9 degrees; 95% CI 0.7-5.1, P=0.01). Cluster 2 demonstrated greater lateral pelvic translation (foot placement closer to midline) Cluster 1 at heel strike (mean difference -14.8 FP: 1/2inter-ASIS%; 95% CI -28.0, -1.6, P=0.03). With respect to kinetics, Cluster 2 had a greater hip

flexion moment impulse during the first 50% stance than Clusters 1 (mean difference 0.9 Nm/BW.Ht%/s; 95%CI 0.5, 1.3, P<0.001) and 3 (mean difference 0.7Nm/BW.Ht%/s; 95% CI 0.2, 1.1, P=0.01). Comparison of individuals with and without GT within in each cluster (cluster x group interaction) revealed greater contralateral trunk lean throughout stance in individuals with GT in Clusters 1 and 3 (all P<0.05) but not for Cluster 2.

#### 4. Discussion

This first study to evaluate biomechanics during a step up task in GT revealed two principle findings. First, compared to pain-free controls, individuals with GT exhibited greater contralateral trunk lean, overall HADM impulse and internal rotation moment impulse during vertical thrust (first 50% of stance). Second, both groups exhibited substantial heterogeneity in the HADM waveform, which was explained by the presence of three subgroups. Individuals with GT were 4.5 times more likely than controls to be in the subgroup that exhibited: (1) the largest HADM impulse during the second 50% of stance (forward trajectory), and (2) greater lateral pelvic shift and less pelvic obliquity at heel-strike; and (3) greater flexion positive impulse during vertical thrust (first 50% stance) than the subgroup that was most frequent in controls. Together, these findings infer individuals with GT have greater demand for an internal moments generated by: (1) hip abductor muscles (including the gluteus medius and minimus via their insertions into the greater trochanter, and the TFL and UGM via their insertions into the ITB) throughout stance; and (2) hip external rotator and extensor muscles (including the gluteus maximus and posterior gluteus medius) during the first 50% stance when the hip is in the greatest position of adduction. These features are consistent with greater loads on the gluteal tendons in a hip position that is likely to increase tensile and compressive stress in these tendons.

The present findings of greater contralateral lean and indices of the HADM in individuals with GT than controls concur with between-group differences previously identified during walking on level ground (Allison et al., 2016b). Modelling studies suggest that: (1) ITB tension increases with HADM and hip adduction angle (Tateuchi et al., 2015) and (2) ITB tension and subsequent compressive forces between the ITB and greater trochanter (gluteal tendon insertion) increase with hip adduction angle (Birnbaum et al., 2004). The impact of greater HADM on gluteal tendon loading is likely to be greater during stair ascent than level walking for several reasons. First, stair ascent involves a greater range of hip adduction (and flexion) during weight acceptance than level walking (Nadeau et al., 2003). Second, ITB tension will also be influenced by contraction of the vastus lateralis which is activated in order to generate a large knee extensor moment in stair ascent (McFadyen and Winter, 1988; Nadeau et al., 2003); and potentially the greater degree of hip flexion (Nadeau et al., 2003) given the fascial relationship between the thoracodorsal, gluteus maximus fascia and ITB (Stern, 1972; Vieira et al., 2007). Third, individuals with GT were more likely to have a HADM waveform characterized by peak values that were almost three times greater than the peak of the moment pattern most frequent in controls, and two to three times higher than we have previously identified in individuals with and without GT during walking (Allison et al., 2016b). Taken together, we speculate this to imply greater compressive loading of the gluteal tendons at the greater trochanter in individuals with GT than controls. As excessive compressive load is accepted as a key mechanical factor in the aetiology of tendinopathy (see (Docking et al., 2013) for review), these findings have implications the development and/or perpetuation of GT. Further, large magnitude HADM during stair ascent implies requirement for generation of internal hip abductor moments that would be larger than those typically associated with stair ascent in pain-free individuals in this and previous studies (Kirkwood et al., 1999; Nadeau et al., 2003). This abductor demand, superimposed upon hip abductor

weakness in individuals with GT (Allison et al., 2016a; Woodley et al., 2008), may further contribute to gluteal tendon overload.

In contrast to previous data of pain-free controls (Nadeau et al., 2003), we identified large variability in the HADM in both GT and control groups. The variability identified here was explained by three moment waveforms verified by cluster analysis. Cluster 1 exhibited a negative HADM during the second 50% of stance and Cluster 3 a large positive HADM. Similar variability in polarity of the hip flexor moment was reported in pain-free individuals a small study by McFayden and Winter (McFadyen and Winter, 1988). Those authors suggested that, unlike the knee and ankle, hip moments during stance phase could not be stereotypical due to variations in trunk and pelvic angular accelerations between individuals during stair ascent (McFadyen and Winter, 1988). Although variation in trunk lean was evident amongst participants in both groups in the present study, post-hoc analysis did not reveal a significant interaction between pelvic and trunk position and the HADM as identified previously during walking in this cohort (Allison et al., 2016b). It must be considered that the nature of our stair ascent task, which started and ended from a static position with parallel feet, would induce a different pattern of accelerations of the centre of mass than walking. These accelerations would likely differ in the frontal plane with variable manifestation of HADM waveform patterns. Further, although we identified subgroups based on HADM waveforms that were associated with increased or reduced relative risk of GT, these subgroups included participants with and without GT. It is possible that biomechanical mechanisms contributing to tendon overload differ in individuals with GT, and are influenced by other factors such as bony morphology of the proximal femur and pelvis (Fearon et al., 2012) and/or hip abductor muscle activation patterns influencing tension within the ITB (Allison et al 2015 unpublished data). Longitudinal studies are required to ascertain whether

controls with HADM waveforms characteristic of a large HADM impulse are at risk of developing GT. However, this is unlikely to be a simple relationship, as it would likely depend on exposure to loading and individual tissue properties.

Although stair ascent has not been studied in participants with GT, previous studies have included individuals with intra-articular hip pathology, including hip OA (Meyer et al., 2015), femoroacetabular impingement (FAI) (Rylander et al., 2013). While hip abductor muscle weakness is common to GT (Allison et al., 2016a; Woodley et al., 2008), hip OA (Loureiro et al., 2013) and FAI (Casartelli et al., 2011), its relationship with kinematics of stair ascent may differ. No frontal plane differences in stair kinematics have been identified between individuals with and without FAI, but this analysis excluded evaluation of the trunk (Rylander et al., 2013). Conversely, a recent study by Meyer et al.(2015) of individuals with mostly advanced hip OA (mean age 49.9 years) identified lower HADM and greater ipsilateral trunk lean during stair ascent (step height - 184 mm). The lower step height than that used in the present study (240mm) implies a lower demand for the hip OA group. Although individuals with advanced hip OA exhibit disability (Fearon et al., 2014) and hip abductor strength deficits (Loureiro et al., 2013) similar to that reported in GT, the findings for individuals with hip OA contrast those of the present study, where individuals with GT exhibited greater HADM and contralateral lean away from the stance limb. Previous studies evaluating walking in hip OA suggest a compensatory ipsilateral trunk lean can develop with disease progression (Thurston, 1985; Watelain et al., 2001; Zeni et al., 2015), representing a strategy to reduce the HADM, demand on the hip abductor muscles and provocative joint contact forces. The present data imply individuals with GT (and no evidence of intra-articular pathology) do not alleviate load on the weak and painful lateral hip structures using this compensatory strategy. Whether this develops later in the course of the disease, develops if

pain is elevated or whether these patterns are distinct disease-specific adaptations requires further consideration.

The between-group differences in trunk lean during the stance phase of stair ascent was small on average (range of 2-3 degrees). This may be challenging to detect visually in a clinical setting and in isolation such a small magnitude of trunk lean may not have clinical relevance. Trunk lean was defined as the angle of the thorax (T2-T12) relative to vertical, and although thorax-to-vertical can be observed visually, the minimal detectable difference is not known. As trunk lean relative to the pelvic segment has also been suggested as a method for clinicians to identify trunk lean and/or lateral shift of the pelvis (Grimaldi, 2011); lateral translation of the pelvis over the stance foot (as associated at heel strike in the GT-dominant subgroup) together with a contralateral trunk lean (greater in GT group), may provide an optimal method for assessment in a clinical setting. Together these identified patterns in GT in this study might be characteristic of the 'shunting' ('abnormal') pelvic pattern described in clinical gait commentary of those with GT (Bird et al., 2001; Grimaldi, 2011; Woodley et al., 2008). Targeting trunk and pelvic control in conservative treatment might be appropriate to address biomechanics of step-up in individuals with GT.

Several methodological issues in the present study require consideration. Although we present *external hip adduction* moment data as an indicator of *internal hip abductor* moments, further research is required to understand the muscle activation patterns of the hip abductors during stair ascent. Our groups were not matched for anthropometric characteristics with the GT group having a greater BMI and pelvic width. A primary reason was that greater BMI and adiposity has been shown to be associated with GT (Fearon et al., 2012) and our aim was to investigate individuals as they present clinically. Moment data was normalized to

body weight times height, thus the non-normalized between-group differences are greater than those presented here. Technical considerations when comparing our data to others and inferring these findings to practice include: step height, analysis of the stance leg on the first step (others have analysed the second step (Kirkwood et al., 1999; Nadeau et al., 2003)), and the number of stairs in the task. Two studies have previously reported no difference between the peak HADM during stair ascent and walking in healthy individuals aged 55 to 75 (Kirkwood et al., 1999) and 40 to 71 (Nadeau et al., 2003) years, age ranges comparable to our study. This does not agree with our work, which shows a higher peak HADM in stair ascent (8.1 Nm/kg) than what we have previously reported during walking (5.6 Nm/kg) in the same cohort of pain-free controls (Allison et al., 2016b). This might be explained by differences in task demands; previous studies used lower step heights (215mm (Kirkwood et al., 1999) and 170mm (Nadeau et al., 2003)), than the 240mm step height of the present study. Stair ascent in day-to-day function typically involves a greater number of stairs, and this might be more profoundly affected by muscle weakness, fatigue or lateral hip pain than the reduced task we evaluated. An additional consideration is that only 17 participants reported pain during testing and pain levels were low, despite the report by 32 participants that stair ascent was a provocative task during initial screening (median (IQR) 5(5) on the NRS). Finally, our study was not powered for subgroup analysis, thus we present these subgroups as exploratory findings.

#### 5. Conclusion

In conclusion, the present study showed that during stair ascent, individuals with GT exhibit greater contralateral lean and a greater total positive HADM moment and impulse and internal rotation impulse during the first 50% of stance than pain-free controls. Further longitudinal research is needed to evaluate whether these movement patterns contribute to the development or perpetuation of GT and its symptoms. Whether modification of biomechanics

of stair ascent with conservative interventions has relevance for GT also warrants investigation.

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	Gluteal Pain-fre		Mean	<i>P</i> -value			
	tendinopathy	control	difference				
	(n=35)	(n=35)	(95% CI)				
Age, years	54 (8)	53 (9)	1 ( -4, 5)	0.71			
Height, m	1.68 (0.09)	1.67 (0.10)	0.00 (-0.04,0.05)	0.85			
Weight, kg	73.8 (14.6)	67.9 (13.1)	5.8 (-0.8, 12.4)	0.08			
Body mass index, kg/m <sup>2</sup>	26.1 (4.3)	24.1 (2.7)	2.0 (0.3, 3.8)	0.02			
Inter-ASIS width, mm	th, mm 264 (26)		33 (22, 44)	<0.001			
Sex, n (%)	2						
Female	26 (74%)	26 (74%)		$1.0^{\text{¥}}$			
Male	9 (26%)	9 (26%)		$1.0^{\text{¥}}$			
Symptomatic (Test)	Right = 14	Right = 17					
hip*	Left = 21	Left = 18					
Dominant limb	Right $= 31$	Right $= 33$					
$\mathbf{G}$	Left = 4	Left $= 2$					
Symptom duration,	18 (28)	0 (0)					
months, median (IQR)							
Lateral hip pain severity, (0-10), median $(IQR)^{\infty}$							
Average over past week <sup>‡</sup>	4 (1)						
Worst over past week <sup>‡</sup>	7 (1)						
Walking (normal pace) <sup>‡</sup>	3 (2)						
Walking (fast pace) <sup>‡</sup>	4 (4)						
Stair climbing <sup>‡</sup>	5 (5)						

Table 1. Descriptive characteristics of the study sample.

Mean (standard deviation) unless otherwise stated

<sup>‡</sup> Measured using a self-reported 11 point-numerical rating scale (0 = no pain; 10 = worst

pain imaginable),  $^{\infty}$  Data not normally distributed

¥ P<0.05 using Pearson Chi-Square test

\* 'Symptomatic Hip' designated in control participants by a coin toss

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# Table 2. Kinematics and kinetics during the stance phase of stair ascent in individuals with and without GT

	Gluteal	Pain-free	Mean difference	<i>P</i> -value			
	tendinopathy	control	(95%CI)				
			$\leq$				
Kinematic Variables							
Maximum hip adduction	on angle, degrees	G					
Heel strike (HS)	5.1 (7.2)	5.2 (6.2)	-0.1 (-3.3, 3.2)	0.96			
HS to Reciprocal	12.4 (5.8)	12.8 (6.1)	-0.5 (-3.2, 2.6)	0.77			
toe off (RTO)							
RTO to end of stance	12.4 (5.8)	12.5 (5.6)	-0.1 (-2.9, 2.7)	0.97			
Maximum hip internal	rotation angle, degr	rees					
HS	-3.7 (9.2)	-4.2 (7.5)	0.5 (-3.5, 4.5)	0.81			
HS to RTO	-3.6 (7.4)	-3.0 (7.6)	-0.6 (-4.2, 3.1)	0.75			
RTO to end of stance	-0.7 (7.1)	-0.1 (8.0)	-0.6 (-4.3, 3.1)	0.76			
Maximum hip flexion a	Maximum hip flexion angle, degrees						
HS <sup>#</sup>	77.1 (10.2)	74.5 (5.8)	2.6 (-1.4, 6.6)	0.24			
HS to RTO <sup>#</sup>	79.3 (10.2)	75.4 (5.7)	3.8 (-0.1, 7.8)	0.12			
RTO to end of stance	82.3 (10.4)	80.3 (6.4)	2.0 (-4.2, 5.1)	0.31			
Maximum pelvic obliq	uity <sup>a</sup> , degrees						
HS O	6.5 (5.3)	7.7 (3.4)	-1.1 (-3.2, 1.0)	0.31			
HS to RTO	9.8 (4.5)	10.3 (3.5)	-0.5 (-2.4, 1.4)	0.59			
RTO to end of stance	7.9 (4.2)	8.7 (3.7)	-0.9 (-2.8, 1.1)	0.38			
Lateral translation pelvis <sup>b</sup> , foot placement from midline:1/2 Inter-ASIS distance (%)							
HS	74.4 (28.2)	75.8 (23.6)	-1.5(-13.8, 10.9)	0.82			
HS to RTO	30.5 (13.7)	33.1 (17.7)	-2.6 (-10.3, 5.0)	0.49			
RTO to end of stance	10.23 (16.6)	11.39 (15.2)	-1.2 (-8.0, 5.7)	0.74			
Maximum ipsilateral trunk lean <sup>c</sup> , degrees							
HS	-2.4 (3.7)	0.7 (3.2)	-3.1 (-4.8, 1.4)	0.001*			
HS to RTO	2.7 (2.9)	4.8 (2.5)	-2.0 (3.4, -0.7)	0.003*			
RTO to end of stance	3.8 (3.9)	5.2 (2.6)	-1.5 (-3.1, 0.2)	0.08			

Maximum contralateral trunk lean, degrees						
HS to RTO	-2.6 (3.7)	0.6 (3.1)	-3.1 (-4.8, 1.5)	0.001*		
RTO to end of stance	-3.9 (3.3)	-1.7 (3.2)	-2.2 (-3.8, -0.6)	0.01*		
			6			
Kinetic Variables						
External hip adduction mon	nent (HADM), Nm/	(BW.Ht)%				
HS	-0.7 (5.4)	0.2 (3.4)	-0.6 (-3.1, 1.3)	0.43		
RTO	3.4 (2.8)	2.9 (2.9)	0.7 (-0.9, 2.1)	0.42		
Overall maximum	10.7 (3.9)	8.1 (3.8)	2.6 (0.8, 4.5)	0.01*		
Positive HADM impulse, Nr	n.sec/(BW.Ht)%					
1st 50% stance	2.3 (1.1)	1.9 (0.8)	0.4 (-0.1, 0.1)	0.09		
2nd 50% stance	4.1 (3.4)	2.3 (2.8)	1.9 (0.4, 3.4)	0.01*		
Overall stance	6.5 (3.1)	4.1 (2.7)	2.3 (0.9, 3.8)	0.03*		
External hip flexion moment, Nm/(BW.Ht)%						
HS	-2.8 (5.7)	-3.2 (3.6)	0.4 (-2.0, 2.8)	0.74		
RTO	5.5 (3.1)	6.0 (2.1)	-0.5 (-1.8, 0.9)	0.49		
Overall maximum	7.1 (2.2)	6.5 (1.9)	0.6 (-0.4, 1.7)	0.23		
Positive external hip flexion impulse, Nm.sec/(BW.Ht)%						
1 <sup>st</sup> 50% stance	2.1 (0.8)	1.8 (0.6)	0.3 (-0.1, 0.6)	0.15		
2 <sup>nd</sup> 50% stance#	0.4 (0.6)	0.4 (0.4)	0.0 (-0.3, 0.3)	0.88		
Overall stance	2.8 (2.4)	2.1 (0.7)	0.7 (-0.2, 1.5)	0.72		
External hip internal rotation moment, Nm/(BW.Ht)%						
HS	0.1 (1.8)	-0.2 (1.3)	0.3 (-0.5, 1.0)	0.51		
RTO	-1.4 (1.1)	-1.4 (1.2)	-0.0 (-0.8, 0.5)	0.95		
Overall maximum	1.3 (0.8)	1.0 (0.6)	0.3 (-0.1, 0.6)	0.10		
Positive external hip internal rotation impulse, Nm.sec/(BW.Ht)%						
1 <sup>st</sup> 50% stance	0.2 (0.2)	0.1 (0.1)	0.1 (0.0-0.2)	0.01*		
2 <sup>nd</sup> 50% stance	0.3 (0.1)	0.2 (0.1)	0.1 (-0.01, 0.2)	0.12		
Overall stance	0.3 (0.1)	0.2 (0.1)	0.1 (-0.01, 0.14)	0.09		

Reciprocal - contralateral leg

<sup>#</sup>Data not normally distributed

\* significant between group difference

<sup>a</sup> Positive pelvic obliquity indicates the contralateral pelvis is dropped relative to the stance limb

 $^{b}$  0% = position of the calcaneus directly under the midline, 100% = position of the calcaneus directly under the ASIS

<sup>c</sup> Negative values indicate trunk lean away from the stance limb (ie, Contralateral trunk lean)

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	<b>CLUSTER 1</b>	CLUSTER 2	CLUSTER 3	Total
GT				
Incidence	11	12	9	32
Estimated	34.8%	37.5%	28.13%	
Proportion	(20.4, 51.7)	(22.9, 54.8)	(15.56, 45.37)	
(95%CI)			2	
CONTROL				
Incidence	21	4	9	34
Estimated	61.7%	11.8%	26.5%	
Proportion	(45.0, 76.1)	(4.7, 26.6)	(14.6, 43.1)	
(95%CI)	L.			
<b>ODDS Ratio</b>	0.32	4.5	1.08	
(95% CI)	(0.12, 0.89)	(1.27, 15.95)	(0.37, 3.21)	
Relative Risk	44%	-22%	6.3%	
Reduction	(4, 68)	(-79, -14)	(-13, 5)	
(95%CI)				
Absolute Risk	27%	-26%	2%	
Reduction	(3-47)	(-44,-5)	(-23, 19)	
(95%CI)				

Table 3. C	Comparison	of cluster	frequencies	s in each	participant	group
						0

Evaluated using the Newcombe-Wilson method



Figure 1. Participant performing reciprocal step up task. Analysis was restricted to the period

of stance on the test leg, i.e. middle 3 panels.

**Figure 2.** Group ensemble averages for kinematic and kinetic variables during the stance phase of stair ascent. Data are shown for GT (red) and control (black) participants as mean (solid line) and standard deviation (dashed line). The grey vertical lines represent the range of time of reciprocal toe off (RTO) in participants (beginning of single support on first step).



**Figure 3.** (A) Ensemble averages of Cluster 1 (black), Cluster 2 (red) and Cluster 3 (grey). Cluster 1 included more controls; Cluster 2 included more GT participants; Cluster 3 included similar numbers of GT and control participants. (B) Cluster centres are plotted for the 3 groups for GT and control participants. (C) Kinematics at heel strike, heel strike to reciprocal toe off and reciprocal toe off to end of stance are shown for each.



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