**Title**: Lower extremity function during gait in participants with first time acute lateral ankle sprain compared to controls.

**Running title:** The effect of acute ankle sprain on gait

**Abstract word count:** 237

**Word count:** 3927

1 table

7 figures

**Authors:**

Cailbhe Doherty[1]

Chris Bleakley[3]

Jay Hertel[4]

Brian Caulfield[1]

John Ryan[5]

Eamonn Delahunt[1,2]

1. School of Public Health, Physiotherapy and Population Science, University College Dublin, Dublin, Ireland.
2. Institute for Sport and Health, University College Dublin, Dublin, Ireland.
3. Sport and Exercise Sciences Research Institute, Ulster Sports Academy, University of Ulster, Newtownabbey, Co. Antrim, Northern Ireland.
4. Department of Kinesiology, University of Virginia, Charlottesville, VA, United States.
5. St. Vincent's University Hospital, Dublin, Ireland.

**Address for Correspondence:**

Cailbhe Doherty

A101
School of Public Health, Physiotherapy and Population Science
University College Dublin
Health Sciences Centre
Belfield
Dublin 4
Ireland
Email: cailbhe.doherty@ucdconnect.ie
Telephone: 00 353 1 7166671
Fax: 00 353 1 716 6501

Conflicts of Interest and Source of Funding:

No conflicts of interest were associated with the authors and the results of this research. This study was supported by the Health Research Board (HRA_POR/2011/46) as follows: PI – Eamonn Delahunt; Co-investigators – Chris Bleakley and Jay Hertel; PhD student – Cailbhe Doherty).

## ABSTRACT

**Background**: Laboratory analyses of chronic ankle instability populations during gait have elucidated a number of anomalous movement patterns. However, no current research exists analysing these movement patterns in a group in the acute phase of lateral ankle sprain injury. It is possible that participants in the acute phase of lateral ankle sprain injury may display movement patterns continuous with their chronically impaired counterparts.

**Methods**: Sixty eight participants with acute first-time lateral ankle sprain injury and nineteen non-injured participants completed gait trials at a self-selected walking velocity. 3D lower extremity temporal kinematic (angular displacement) and kinetic (moment of force) data were collected from 200ms pre- to 200ms post- heel strike (period 1) and from 200ms pre- to 200ms post- toe off (period 2).

**Findings**: During period 1, the lateral ankle sprain group displayed increased knee flexion with increased net extensor pattern at the knee joint, increased ankle inversion and reduced ankle plantar flexion, with a greater inversion moment, compared to the non-injured control group.

During period 2, the lateral ankle sprain group displayed decreased hip extension with a corresponding decrease in the extensor moment at the hip, and decreased ankle plantar flexion with a decrease in the net plantar flexion moment, compared to the non-injured control group.

**Interpretation**: Acute lateral ankle sprain participants display coordination strategies potentially conducive to minimising specific joint loading following injury, and which may play a role in the onset of chronicity or recovery.



**Key words:** ankle joint [MEsH]; biomechanics [MEsH]; kinematics [MEsH]; kinetics [MEsH]; gait [MEsH]

## 1.0 Introduction

Ankle sprain injury has consistently been reported to be a significant risk for participants of a wide range of activity types [1,2]. Indeed, ankle sprain accounts for between 11-12% of injuries in activities such as soccer, field hockey, basketball, volleyball and orienteering, with a collective incidence of between 3.9 and 4.9 ankle sprains per 1000 hours of exposure [1] . The consequences of this injury extend beyond acute maladies however, as it has been reported that 32-74% of individuals with a history of ankle sprain report and endure a range of residual and chronic symptoms including episodes of "giving-way" of the ankle joint, perceived instability, as well as recurrent sprain [3,4]. Chronic ankle instability (CAI) is the encompassing term used to classify these symptoms [5].

Aberrancy of sensorimotor variables of neuromuscular control as determined using biomechanical analysis is a characteristic feature associated with the CAI [6]. Biomechanical measures of CAI populations are typically laboratory based, and involve active movements such as gait [7-9]. For example, previous biomechanical analyses have shown that participants with CAI display increased inversion [7,8] and increased plantar flexion [9] angular displacements, with a greater concentric evertor moment at the ankle joint, in the time period from 200ms pre- to 200ms post heel strike (HS), at the beginning of the stance phase of gait, and reduced plantar flexion [9] at toe-off (TO) at the end of stance. However, research utilizing biomechanical measures to evaluate neuromuscular control during gait in populations prior to the development of CAI, specifically those in the acute phase of a first-time lateral ankle sprain (LAS) injury is sparse. It is possible that a sample group of participants with acute LAS may display movement insufficiencies that are continuous with, and therefore contribute to, those observed in their counterparts in the chronic phase of injury. As such, an evaluation of active movement patterns during gait in a group with first-

time acute LAS injury may advance current understanding of potentially anomalous movement patterns that persist and contribute to the onset of the chronic sequalae of CAI. Therefore, the objective of the current study was to determine if participants with acute LAS display movement patterns continuous with their chronically impaired counterparts when compared to a non-injured control group, using kinetic (joint moment) and kinematic (angular displacement) laboratory measures during gait. It is plausible that these patterns may preclude long-term recovery, and thus, the current analysis stands to advance current understanding of the immediate adaptive motor control processes which develop following acute LAS, and which may contribute to the onset of chronicity.

## 2.0 Methods

### 2.1 Participants

Sixty eight participants (forty-six males and twenty-two females; age 23.26 ± 4.94 years; body mass 76.43 ± 14.33kg; height 1.74 ± 0.09m) were referred from a University-affiliated hospital Emergency Department with acute, first time, LAS injury. Nineteen uninjured participants (fifteen males and four females; age 22.5 ± 1.7 years; body mass 71.55 ± 11.30 kg; height 1.74 ± 0.1 m) were recruited from the hospital catchment area population using posters and flyers to act as a control group. The injured group were recruited within 2-weeks of sustaining their injury. The project was approved by the local ethics committee, and written consent was obtained from each subject prior to data collection. The following exclusion criteria were applied: (1) no previous history of ankle sprain injury (excluding the recent acute episode for the injured group); (2) no other lower extremity injury in the last 6 months; (3) no history of ankle fracture; (4) no previous history of major lower limb surgery; (5) no history of neurological disease, vestibular or visual disturbance or any other pathology that would impair their motor performance [10].

## 2.2 Questionnaires

The activities of daily living and sports subscales of the Foot and Ankle and Ability Measure (FAAMadl and FAAMsport), and the Cumberland Ankle Instability Tool (CAIT), were used to quantify self-reported function, patient reported symptoms and functional ability as measures of LAS severity [11], and overall ankle joint function and symptoms [12] respectively. All participants completed the subscales of the FAAM and CAIT on arrival to the laboratory.

## 2.3 Swelling

Ankle joint swelling was assessed using the figure-of-eight method [13]. High intra-rater and inter-rater reliability has been reported using this technique (ICC = 0.99) [14]. To determine the degree of swelling, the mean value (of 2 measures) was subtracted from the mean value of the non-injured ankle. For control participants the mean value of the non-dominant limb was subtracted from the mean value of the dominant limb.

## 2.4 Experimental procedures

Gait analysis data acquisition was made using 3 Codamotion cx1 units (Charnwood Dynamics Ltd, Leicestershire, UK). This system was fully integrated with two AMTI walkway embedded force plates (Watertown, MA); the Codamotion cx1 units were time synchronized with the force-plates. Kinematic data acquisition was made at 250 Hz and kinetic data at 1000 Hz. Ground reaction force (GRF) data were passed through a fourth-order zero phase Butterworth low-pass digital filter with a 6-Hz cut-off frequency [15]. Joint moments at the hip, knee and ankle were calculated from force plate, lower extremity kinematic, and anthropometric data using an inverse dynamic procedure [16].

Participants were familiarised with testing procedures prior to commencement. Anthropometric data were obtained for the calculation of internal joint centres at the hip, knee, and ankle joints, after the participants' height and weight were recorded, and motion analysis system markers and marker wands were subsequently placed according to manufacturer guidelines by the same investigator in all participants as previously described [8]. A neutral stance trial was used to align the participant with the laboratory coordinate system and to function as a reference position for subsequent kinematic analysis [17]. During testing, participants walked barefoot across the 10 m walkway at a self-determined speed. Each participant was instructed to look at a distant mark to inhibit them from looking down at the floor. Five 'clean' gait cycles, defined by both the participant's feet landing fully on each of the force plates, were identified and saved for future analysis. Any data obtained whereby the participant did not strike the force plate fully was discarded. Prior to data analysis all values of force were normalised with respect to each subject's body mass (BM).

**2.5 Data analysis**

Kinematic data were calculated by comparing the angular orientations of the co-ordinate systems of adjacent limb segments. Kinetic data were calculated using a seven-segment rigid body model based on the principles of inverse dynamics. A vertical component GRF threshold of 10N was used to identify foot contact with the force plate. Kinematic and kinetic data relating to two periods for both limbs were analysed using the Codamotion software: period 1 extended from 200ms pre-HS to 200ms post-HS (coinciding with terminal swing, HS, loading response and mid-stance) and period 2 extended from 200ms pre-toe off (TO) to 200ms post-TO (coinciding with terminal stance, pre-swing, TO and initial swing). These time windows were chosen for analysis as they have previously been used to investigate CAI-associated movement pattern anomalies during gait [7-9], thus fulfilling our primary

objective. Furthermore, these time-windows, which chart the interaction between the motor apparatus and its external environment by quantifying the transitions between stance (closed kinetic chain) and swing (open kinetic chain), consummate an important period within which the motor control system must integrate afferent feedback with an appropriate efferent motor response [18]; accurate positioning at HS and TO are very important in the interest of maintaining safe locomotion of the motor apparatus as increased plantar flexion as well as inversion of the ankle joint stand to increase ground reaction force moments about the sub-talar joint with significant potential for re-sprain of the injured ankle [19,20].

See figure 1 for an illustration of period 1 and period 2.

The following axis conventions were utilised for kinematic and kinetic data: x axis = frontal-plane motion; y = sagittal-plane motion; z = transverse-plane motion. After analysis in the Codamotion software, data were then converted to Microsoft Excel file format with the number of output samples per trial set at 100 + 1 in the data-export option of the Codamotion software, which represented each gait period as 100%, for averaging and further analysis. Time-averaged 3-dimensional angular displacement profiles for hip, knee, and ankle joints were calculated for each limb of all participants in the specified gait periods. Time averaged, sagittal plane hip, knee and ankle moments, in addition to frontal plane ankle moments were identified from the kinematic and force plate data during the specified gait periods. A net sagittal-plane supporting moment profile of all three joints was then identified for each limb of all participants to identify the net-flexor/extensor pattern of all three joints [21]. Net internal moments are described and represent the body's reaction to the external load on each joint. The supporting moment, Ms, during gait was defined as Ms = Mk – Ma - Mh, where Mk, Ma and Mh are the moments at the knee, ankle and hip respectively [22].

**2.6 Statistics**

For the injured group, the injured limb was labelled as ‘‘involved’’ and the non-injured limb as ‘‘uninvolved’’. Limbs in the control group were side-matched to limbs in the injured group as “involved” and “uninvolved”.

Participant characteristics and swelling were compared between the LAS and control groups using multivariate analysis of variance. The dependent variables were age, mass, sex, height and ankle joint swelling. The independent variable was status (injured *vs* non-injured).The significance level for this analysis was set a priori with a bonferroni alpha level of $p < 0.01$.

To determine whether the injured group would demonstrate decreased function compared to the control group a multivariate analysis of variance was undertaken. The independent variable was group (injured *vs.* control). The dependent variables were CAIT score, FAAMadl score and FAAMsport score for the involved limb. The significance level this analysis was set a priori with a bonferonni adjusted alpha level of $p < 0.017$.

Between-group differences in involved and uninvolved limb 3-dimensional, time-averaged angular displacement profiles were tested for statistical significance using independent-samples t-tests for each data point for each period of gait. Similarly, between-group differences in involved and uninvolved limb sagittal plane time-averaged net supporting moment profiles with their hip, knee and ankle constituents, in addition to frontal plane ankle moment profiles, were tested for statistical significance using independent-samples t-tests for each data point for each period of gait. The significance level for these temporal analyses was set a priori at $p < 0.05$. Effect sizes were not calculated secondary to the number of separate comparisons for each variable.

All data were analyzed using Predictive Analytics Software (Version 18, SPSS Inc., Chicago, IL, USA).

## 3.0 Results

There was no statistically significant difference between the injured and control groups on the combined dependent variables of age, sex, height and body mass, $F(4, 82) = 1.639$, $p = 0.17$; Wilk's Lambda = 0.926; partial eta squared = 0.07.

Regarding participant characteristics and swelling there was a statistically significant difference between the injured and control groups on the combined dependent variables, $F(81,5) = 4.24$, $p = 0.002$; Wilk's Lambda = 0.79; partial eta squared = 0.21. When the results of the dependent variables were considered separately, swelling ($F[1, 85] = 17.34$, $p = 0.000$, partial eta squared = 0.17) was the only differences to reach statistical significance. An inspection of the mean scores indicated that injured participants had increased swelling on their involved limb compared to controls (11.09 [SD: 8.8cm] vs 2.47 [SD: 3.4cm]).

Regarding function a statistically significant main effect was observed for the combined dependent variables, $F(3, 77) = 75.147$, $p < 0.01$, Wilks' Lambda = 0.255, partial eta squared = 0.745. Questionnaire results are detailed in Table 1.

Time-averaged 3-dimensional kinematic profiles revealed that the injured displayed increased hip adduction on their involved limb, increased knee varus on their involved limb and bilateral increases in knee flexion and reductions in ankle plantar flexion compared to the control group in the time period from 200ms pre HS to 200ms post HS (specific details of angular displacement data for period 1 are presented in Figure 2).

In the period from 200ms pre TO to 200ms post TO, the injured group displayed reduced hip extension on their involved limb and bilateral increases in ankle inversion and reductions in ankle plantarflexion compared to the control group (specific details of angular displacement data for period 2 are presented in Figure 3).

Time-average sagittal plane moment of force profiles revealed that the injured group displayed reduced hip extension moment on their involved limb and bilateral increases in

knee extension moment and ankle flexor moment compared to control participants in the time period from 200ms pre HS to 200ms post HS (specific details of moment of force data for all lower extremity joints for period 1 are presented in Figure 4). The overall support moment profile for this period revealed a pattern of bilateral greater extension in injured participants compared to control participants. Furthermore, there was a bilateral increase in ankle inversion moment in this time period (specific details of moment of force data for the ankle joint during period 1 are presented in Figure 5).

In the period from 200ms pre TO to 200ms post TO, the injured group displayed bilateral reductions in hip flexor moment, increases in knee extension moment and reductions in ankle extension moment (specific details of moment of force data for all lower extremity joints for period 2 are presented in Figure 6). The overall support moment profile transitioned from reduced flexor moment to reduced extension moment in injured participants compared to control participants. Furthermore, there was a bilateral increase in ankle inversion moment in this time period (specific details of moment of force data for the ankle joint during period 2 are presented in Figure 7).

## 4.0 Discussion

The current investigation has elucidated injury-associated movement patterns in a group reporting significant functional impairment (as determined by the CAIT and subscales of the FAAM) following first-time acute LAS during gait by comparing them to a non-injured control group. This analysis was exploratory in nature in that we considered participants with acute LAS may display movement patterns similar to their chronically impaired counterparts. Monaghan et al. [23], Drewes et al. [24] and Delahunt et al. [7] have previously reported that participants suffering recurrence following an acute LAS display increased inversion at the ankle joint around HS (the equivalent of period 1 in the current study), while Chinn et al.[25]

previously reported no differences in this time window in the same group. In the current analysis, we did not observe any significant differences between LAS and control participants for frontal plane ankle motion around HS, however the injured group did display a significant increase in ankle joint inversion (bilaterally) around TO which is in agreement with the findings of Drewes et al. [24]. This coincided with a motor pattern of increased inversion moment at the ankle joint in both limbs prior to, and following, TO. LAS participants also displayed a bilateral reduction in ankle joint plantar flexion during both period 1 and period 2 of the current analysis, which is in contrast with the findings of Spaulding et al. [9] and Chinn et al.[25], who evaluated movement patterns on the involved limb only, both finding increased plantar flexion in CAI participants during the stance phase of gait. To the authors' knowledge to date, changes in inversion and plantar flexion at the ankle joint during the stance phase of gait are the only laboratory-analysis derived movement pattern differences unique to the gait of participants with CAI compared to non-injured controls, based on previous research [6].

These movement patterns may be adopted in the interest of minimising perceived risk during task performance; the net displacement of the body constitutes a summation of all the forces and motions acting upon, and concerned with, its safe translation during locomotion [26]. These forces however are limited by the anatomical constraints within which they operate [27]. Hence, this translation is potentially altered by acute LAS, and can be seen to be evident in the kinematic and kinetic findings of the current investigation.

The disruption caused by acute insult to the foot and ankle in injured participants can be seen to have been compensated for by local and global modifications which may have been adopted in the aim of minimising risk of further injury; to protect the vulnerable ankle joint, there developed an increased reliance on more proximal structures to absorb impact force

with a concurrent decrease in the propulsive forces that have the potential to augment the magnitude of these impacts [26]. Specifically, with regards to period 1, functional impairment at the ankle joint may have stimulated the knee joint to exaggerate its primary role in the attenuation of impact forces, as displayed by the bilateral increase in LAS participants' knee flexion, and greater knee extensor moment pattern at HS (≃ 0.2 in LAS participants vs ≃ -0.17 Nm/kg in controls, at HS); the ankle plantar flexors, hip flexors, and hip extensors are the main muscle groups that contribute to energy generation in the sagittal plane [28-30], while the knee joint muscles act mainly eccentrically in the absorption of impact energy [26,29]. The implications of the exaggerated motor pattern of absorption at the knee are two-fold: first, increased knee flexion will amplify the descent of the body's centre of gravity, thus limiting gait efficiency, and this must be accounted for using compensatory mechanisms that heighten it: a decrease in the hip adduction and knee varus angular displacement, with a coinciding reduced extensor pattern at the hip (≃ 0.1 in LAS participants vs ≃ 0.2 Nm/kg in controls, at HS) on the involved limb may be the manifestation of this appropriated compensatory mechanism. Indeed frontal plane hip motion (with its corollary of frontal plane knee motion) is one of the primary determinants of the rhythmic displacement of the body's centre of gravity during gait [26], and a reduced extensor pattern at the hip facilitates collapse and thus lowering of the extremity [22]. The bilateral greater net extensor pattern of the lower extremity joints in this period (≃ 0.4 in LAS participants vs ≃ -0.4 Nm/kg in controls, at HS) could reflect the motor pattern at the knee and may also give an overall indication of the compensatory tendency of injured participants to prevent collapse of the total lower extremity (which has connotations of the recently endured injury event). Secondly, the increase in the knee flexion was precipitous to ankle motion, with a decrease in the observed plantar flexion, and the net extensor pattern of the ankle joint in LAS participants (≃ -0.4 in LAS participants vs ≃ 0.01 Nm/kg in controls, at

HS). This resultant ‘closed-pack’ position of reduced plantar flexion, which is closer to sagittal plane neutral, provides the ankle joint of LAS participants with greater stability, secondary to an increase in the congruity between the inferior aspects of the tibia and fibula, and the superior aspect of the talus [31]. Overall, this could reflect a compensatory mechanism to maintain the integrity of the vulnerable ankle joint by using a global strategy of attenuating impact forces using joints proximal to the ankle. Muscle model driven computer simulations have previously shown that an increased HS plantar flexion may cause an increased likelihood of an ankle sprain [32]. Thus, the motor apparatus of LAS participants may have organised its proximal component (in this case, the knee joint), to adjust to the injury constraint.

With regards to period 2, the propulsion of body through space is achieved primarily via the output of the hip and ankle [26,29,33]. LAS participants displayed movement and motor patterns conducive to a reduction in propulsion: the bilateral decrease in hip extension observed in LAS participants compared to control participants, with a coinciding reduction in the net extensor pattern at the hip joint ($\simeq$ -0.4 in LAS participants vs $\simeq$ -0.6 Nm/kg in controls, 100ms pre-TO), and ankle plantar flexion, with a coinciding reduction in the net extensor pattern at the ankle joint prior to TO ($\simeq$ 0.75 in LAS participants vs $\simeq$ 1.2 Nm/kg in controls) combine to indicate a reduction in propulsive motor patterns during terminal stance in injured participants. This shift may be part of a strategy to reduce ankle joint loading by reducing the forces associated with locomotion. The increase in ankle inversion moment at TO previously described could be a compensatory mechanism to restore normal propulsive patterns at the ankle joint: the reduction in propulsive sagittal hip and ankle function may have been compensated for using the rigidity of inversion in the supinating ankle joint, as an assistant to push-off [20]. Combined with a more inverted positioning prior to TO, this may

indicate that injured participants load the lateral column of their foot to a greater extent during the latter part of stance [31,34]. Recent research has demonstrated that CAI participants activate their peroneus longus (PL) prior to HS which is in contrast to healthy controls, thus preventing sufficient medial displacement of the centre of pressure during loading response [34,35]. This may manifest in CAI participants bearing weight more laterally on their foot during stance, which then persists into TO [36]. The current study has elucidated an increase in inversion moment with a coinciding increase in ankle inversion around TO; one could speculate that the increased inversion positioning seen in late stance and early swing is a consequence of the abnormal loading response, and may be predicated by early activation of the PL in the swing phase of gait, which has been demonstrated in CAI participants [25]. Greater inversion around the sub-talar joint axis is likely to produce an external load that further forces the foot into inversion, with increasing the potential risk of lateral ligamentous trauma[20]. The reduction in overall propulsion during TO can be seen to be reflected in the overall extensor pattern, where the flexion-to-extension range in injured participants is significantly reduced compared to that of control participants.

The bilateral nature of the observed results is in agreement with previous findings in participants with recurrance following LAS [37,38], and are the first indication that these deficits may present immediately following the acute injury during gait. That unilateral injury had bilateral manifestations may be linked to an alteration in alpha motoneuron pool excitability [39], suggesting that spinal-level motor control mechanisms have changed. We concur with the speculations of previous researchers who have theorised that because in vivo measures of sensoriotor function require conscious perception of peripheral joint and muscle information, supraspinal aspects of motor control are also altered [37,40]. From a rehabilitation perspective, the findings of the current investigation imply that it may be

pertinent to concentrate on the restoration of proximal neuromuscular control strategies using gait-retraining programmes that are bilaterally applied.

While our results are important, this study was not without its limitations. Due to the design of the current study, it is unknown as to whether the movement patterns observed preceded or occur as a result of LAS, and whether they actually have any implication for the onset of CAI in the longer term recovery of these participants. Future studies would benefit from a longitudinal follow-up period to determine the gait-related movement risk factors for LAS, and subsequently the movement characterisitcs that are associated with recovery or chronicity.

## 5.0 Conclusion

The findings of this study advance current understanding of the effects of LAS on gait. The results suggest that participants with acute LAS demonstrate bilateral differences in gait coordination strategies to non-injured controls that adhere intuitively to the fundamental goals of safe and efficient locomotion, with potential links to the progression of chronic sequalae.

## Acknowledgements

This study was supported by the Health Research Board (HRA_POR/2011/46) as follows: PI – Eamonn Delahunt; Co-investigators – Chris Bleakley and Jay Hertel; PhD student – Cailbhe Doherty). The results of the present study do not constitute endorsement by ACSM.

399 References


400 [1] Doherty C, Delahunt E, Caulfield B, Hertel J, Ryan J, Bleakley C. The Incidence and
401 Prevalence of Ankle Sprain Injury: A Systematic Review and Meta-Analysis of
402 Prospective Epidemiological Studies. Sports Med 2014;44 (1) 123-140
403 [2] Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle
404 injury and ankle sprain in sports. Sports Med 2007;37:73-94.
405 [3] Anandacoomarasamy A, Barnsley L. Long term outcomes of inversion ankle injuries.
406 British journal of sports medicine 2005;39:e14; discussion e14.
407 [4] Konradsen L, Bech L, Ehrenbjerg M, Nickelsen T. Seven years follow-up after ankle
408 inversion trauma. Scand J Med Sci Sports 2002;12:129-135.
409 [5] Delahunt E, Coughlan GF, Caulfield B, Nightingale EJ, Lin CW, Hiller CE. Inclusion
410 criteria when investigating insufficiencies in chronic ankle instability. Med Sci Sports
411 Exerc 2010;42:2106-2121.
412 [6] Hiller CE, Nightingale EJ, Lin CW, Coughlan GF, Caulfield B, Delahunt E.
413 Characteristics of people with recurrent ankle sprains: a systematic review with meta-
414 analysis. British journal of sports medicine 2011;45:660-672.
415 [7] Delahunt E, Monaghan K, Caulfield B. Altered neuromuscular control and ankle joint
416 kinematics during walking in subjects with functional instability of the ankle joint.
417 The American journal of sports medicine 2006;34:1970-1976.
418 [8] Monaghan K, Delahunt E, Caulfield B. Ankle function during gait in patients with
419 chronic ankle instability compared to controls. Clin Biomech (Bristol, Avon)
420 2006;21:168-174.
421 [9] Spaulding SJ, Livingston LA, Hartsell HD. The influence of external orthotic support
422 on the adaptive gait characteristics of individuals with chronically unstable ankles.
423 Gait Posture 2003;17:152-158.

[10] Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Postural control strategies during single limb stance following acute lateral ankle sprain. Clinical Biomechanics.

[11] Carcia C, Martin R, Drouin J. Validity of the Foot and Ankle Ability Measure in athletes with chronic ankle instability. J Athl Train 2008;43:179-183.

[12] Hiller C, Refshauge K, Bundy A, Herbert R, Kilbreath S. The Cumberland ankle instability tool: a report of validity and reliability testing. Arch Phys Med Rehabil 2006;87:1235-1241.

[13] Esterson PS. Measurement of ankle joint swelling using a figure of 8*. The Journal of orthopaedic and sports physical therapy 1979;1:51-52.

[14] Tatro-Adams D, McGann S, Carbone W. Reliability of the figure-of-eight method of ankle measurement. The Journal of orthopaedic and sports physical therapy 1995;22:161-163.

[15] Winter D. Biomechanics and motor control of human movement. 4th ed. Hoboken, NJ: John Wiley and Sons; 2009.

[16] Gagnon D, Gagnon M. The Influence of Dynamic Factors on Triaxial Net Muscular Moments at the L5/S1 Joint during Asymmetrical Lifting and Lowering. J Biomech 1992;25:891-901.

[17] Wu G, Siegler S, Allard P, Kirtley C, Leardini A, Rosenbaum D, et al. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion—part I: ankle, hip, and spine. Journal of Biomechanics 2002;35:543-548.

[18] McVea D, Donelan J, Tachibana A, Pearson K. A role for hip position in initiating the swing-to-stance transition in walking cats. J Neurophysiol 2005;94:3497-3508.

[19] Wright I, Neptune R, van den Bogert A, Nigg B. The influence of foot positioning on ankle sprains. J Biomech 2000;33:513-519.

[20] Tropp H. Commentary: Functional Ankle Instability Revisited. J Athl Train 2002;37:512-515.

[21] Winter D. Moments of force and mechanical power in jogging. J Biomech 1983;16:91-97.

[22] Winter D. Overall principle of lower limb support during stance phase of gait. J Biomech 1980;13:923-927.

[23] Monaghan K, Delahunt E, Caulfield B. Ankle function during gait in patients with chronic ankle instability compared to controls. Clinical Biomechanics 2006;21:168-174.

[24] Drewes LK, McKeon PO, Paolini G, Riley P, Kerrigan DC, Ingersoll CD, et al. Altered ankle kinematics and shank-rear-foot coupling in those with chronic ankle instability. J Sport Rehabil 2009;18:375-388.

[25] Chinn L, Dicharry J, Hertel J. Ankle kinematics of individuals with chronic ankle instability while walking and jogging on a treadmill in shoes. Phys Ther Sport 2013;14:232-239.

[26] Saunders J, Inman V, Eberhart H. The major determinants in normal and pathological gait. J Bone Joint Surg Am 1953;A:543-558.

[27] Winter D. Knowledge base for diagnostic gait assessments. Med Prog Technol 1993;19:61-81.

[28] Chen IH, Kuo KN, Andriacchi TP. The influence of walking speed on mechanical joint power during gait. . Gait Posture 1997;6:171-176.

[29] Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. Phys Ther 1994;74:872–885.

[30] Winter DA. The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological. Waterloo: University of Waterloo Press; 1991.

[31] Willems T, Witvrouw E, Delbaere K, De Cock A, De Clercq D. Relationship between gait biomechanics and inversion sprains: a prospective study of risk factors. Gait Posture 2005;21:379-387.

[32] Wright IC, Neptune RR, van den Bogert AJ, Nigg BM. The influence of foot positioning on ankle sprains. J Biomech 2000;33:513-519.

[33] Teixeira-Salmela LF, Nadeau S, McBride I, Olney SJ. Effects of muscle strengthening and physical conditioning training on temporal, kinematic and kinetic variables during gait in chronic stroke survivors. J Rehabil Med 2001;33:53-60.

[34] Schmidt H, Sauer LD, Lee SY, Saliba S, Hertel J. Increased in-shoe lateral plantar pressures with chronic ankle instability. Foot Ankle Int 2011;32:1075-1080.

[35] Nyska M, Shabat S, Simkin A, Neeb M, Matan Y, Mann G. Dynamic force distribution during level walking under the feet of patients with chronic ankle instability. British journal of sports medicine 2003;37:495-497.

[36] Feger M, Donovan L, Hart J, Hertel J. Lower Extremity Muscle Activation in Patients With and Without Chronic Ankle Instability. J Athl Train 2014;[In press].

[37] Hass C, Bishop M, Doidge D, Wikstrom E. Chronic ankle instability alters central organization of movement. The American journal of sports medicine 2010;38:829-834.

[38] Wikstrom EA, Bishop MD, Inamdar AD, Hass CJ. Gait termination control strategies are altered in chronic ankle instability subjects. Med Sci Sports Exerc 2010;42:197-205.

[39] Sedory EJ, McVey ED, Cross KM, Ingersoll CD, Hertel J. Arthrogenic muscle response of the quadriceps and hamstrings with chronic ankle instability. J Athl Train 2007;42:355-360.

[40] Hertel J. Sensorimotor deficits with ankle sprains and chronic ankle instability. Clin Sports Med 2008;27:353-370, vii.

519 Figure legends

520 Figure 1.Illustrative depiction of a link segment model completing period 1 (200ms pre-HS to
521 200ms post-HS), and period 2(200ms pre-TO to 200ms post-TO) of the gait cycle.
522 Abbreviations: HS = heel strike; TO = toe off.
523 Stance limb = limb in bold.
524

525 Figure 2. Hip-joint adduction-abduction, knee joint varus-valgus and flexion-extension, and
526 ankle joint plantarflexion-dorsiflexion angular displacement during period 1 of the gait
527 cyclefrom 200ms pre-HS to 200ms post-HS for the involved and uninvolved limbs of LAS
528 and control groups. Adduction, varus, flexion and dorsiflexion are positive; Abduction,
529 valgus and plantarflexion are negative. Black line with arrow=initial contact. Shaded area =
530 area of statistically significant difference between LAS and control groups. Abbreviations:
531 HS = heel strike; LAS = lateral ankle sprain.
532

533 Figure 3. Hip-joint flexion-extension, ankle joint inversion-eversion and plantarflexion-
534 dorsiflexion angular displacement during period 2 of the gait cyclefrom 200ms pre-TO to
535 200ms post-TO for the involved and uninvolved limbs of LAS and control groups. Flexion,
536 inversion, dorsiflexion are positive; Extension,eversion and plantarflexion are negative. Black
537 line with arrow=initial contact. Shaded area = area of statistically significant difference
538 between LAS and control groups. Abbreviations: TO = toe off; LAS = lateral ankle sprain.
539

540 Figure 4. Sagittal plane joint moment-of-force profiles for the hip, knee and ankle during
541 period 1 of the gait cycle from 200ms pre-HS to 200ms post-HS for the involved and
542 uninvolved limbs of the LAS and control groups. Extension moments are positive; flexion
543 moments are negative. Black line with arrow=initial contact. Shaded area = area of

544 statistically significant difference between LAS and control groups. Abbreviations: HS = heel
545 strike; LAS = lateral ankle sprain; Mh = Hip moment; Mk = Knee Moment; Ma = Ankle
546 moment; Ms = Support moment (Mk-Mh-Ma).
547
548 Figure 5.Frontal plane joint moment-of-force profiles for the ankle joint during period 1 of
549 the gait cycle from 200ms pre-HS to 200ms post-HS for the involved and uninvolved limbs
550 of the LAS and control groups. Inversion moments are positive; eversion moments are
551 negative. Black line with arrow=initial contact. Shaded area = area of statistically significant
552 difference between LAS and control groups. Abbreviations: HS = heel strike; LAS = lateral
553 ankle sprain.
554
555 Figure 6. Sagittal plane joint moment-of-force profiles for the hip, knee and ankle during
556 period 2of the gait cycle from 200ms pre-TO to 200ms post-TO for the involved and
557 uninvolved limbs of the LAS and control groups. Extension moments are positive; flexion
558 moments are negative. Black line with arrow=initial contact. Shaded area = area of
559 statistically significant difference between LAS and control groups. Abbreviations: TO = toe
560 off; LAS = lateral ankle sprain; Mh = Hip moment; Mk = Knee Moment; Ma = Ankle
561 moment; Ms = Support moment (Mk-Mh-Ma).
562
563 Figure 7.Frontal plane joint moment-of-force profiles for the ankle joint during period 2of the
564 gait cycle from 200ms pre-TO to 200ms post-TO for the involved and uninvolved limbs of
565 the LAS and control groups. Inversion moments are positive; eversion moments are negative.
566 Black line with arrow=initial contact. Shaded area = area of statistically significant difference
567 between LAS and control groups. Abbreviations: TO = toe off; LAS = lateral ankle sprain.
568

***Potential Reviewers (please suggest at least 2 potential reviewers)**

**Suggested reviewer #1: Dr Cathleen Brown.**

Dr Brown is an associate professor at the Department of Kinesiology in the University of Georgia.

She has published a large number of articles pertaining to the effects of ankle sprain injury on coordination strategies during a wide range of dynamic tasks.

These articles have established her as a leading researcher in the field of ankle sprain injury and neuromuscular control.

We believe Dr Brown would be a suitable potential reviewer for the current manuscript.

**Suggested reviewer #2: Dr Claire Hiller**

Dr Hiller has published a wide range of articles on the deficits associated with Chronic Ankle Instability, many of which are considered landmark papers in the field.

She is widely regarded as an expert in the field of ankle sprain research, with a particular focus on objective measures of CAI, and the aberrant movement characteristics of this population.

As the current paper is an exploratory analysis of participants with acute ankle sprain injury during gait to determine if their movement patterns are continuous with their chronically impaired counterparts, and as we incorporated several objective measures commonly used in CAI investigations to quantify acute dysfunction, we believe Dr Hiller would be well-placed to review the current article.

**Table 1**

Table 1. Questionnaire scores (mean ± SD with 95% CIs) for the LAS and control groups.

| | CAIT | FAAMadl | FAAMsport |
|---|---|---|---|
| Injured | 11.60 ± 7.21[a] | 56.77 ± 13.85%[a] | 39.80 ± 28.71%[a] |
| Control | 30 ± 0[b] | 100 ± 0%[b] | 100 ± 0%[b] |

[a] significantly different from control group;

[b] significantly different from injured group;

**Figure 1**

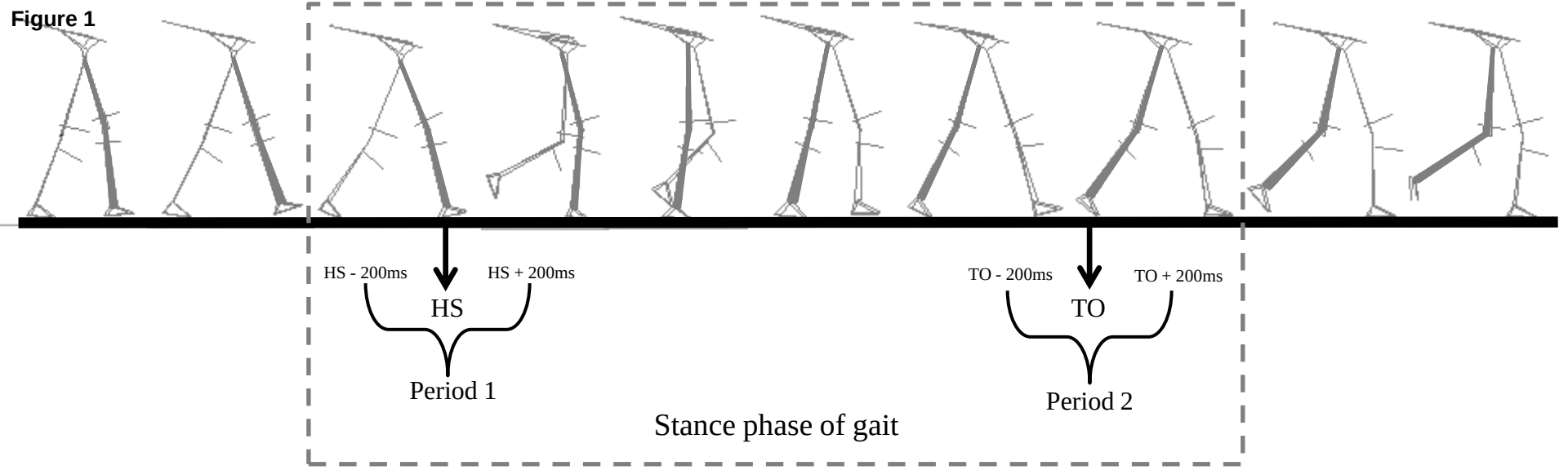

HS - 200ms
HS + 200ms
HS
Period 1
TO - 200ms
TO + 200ms
TO
Period 2
Stance phase of gait

**Figure 2**

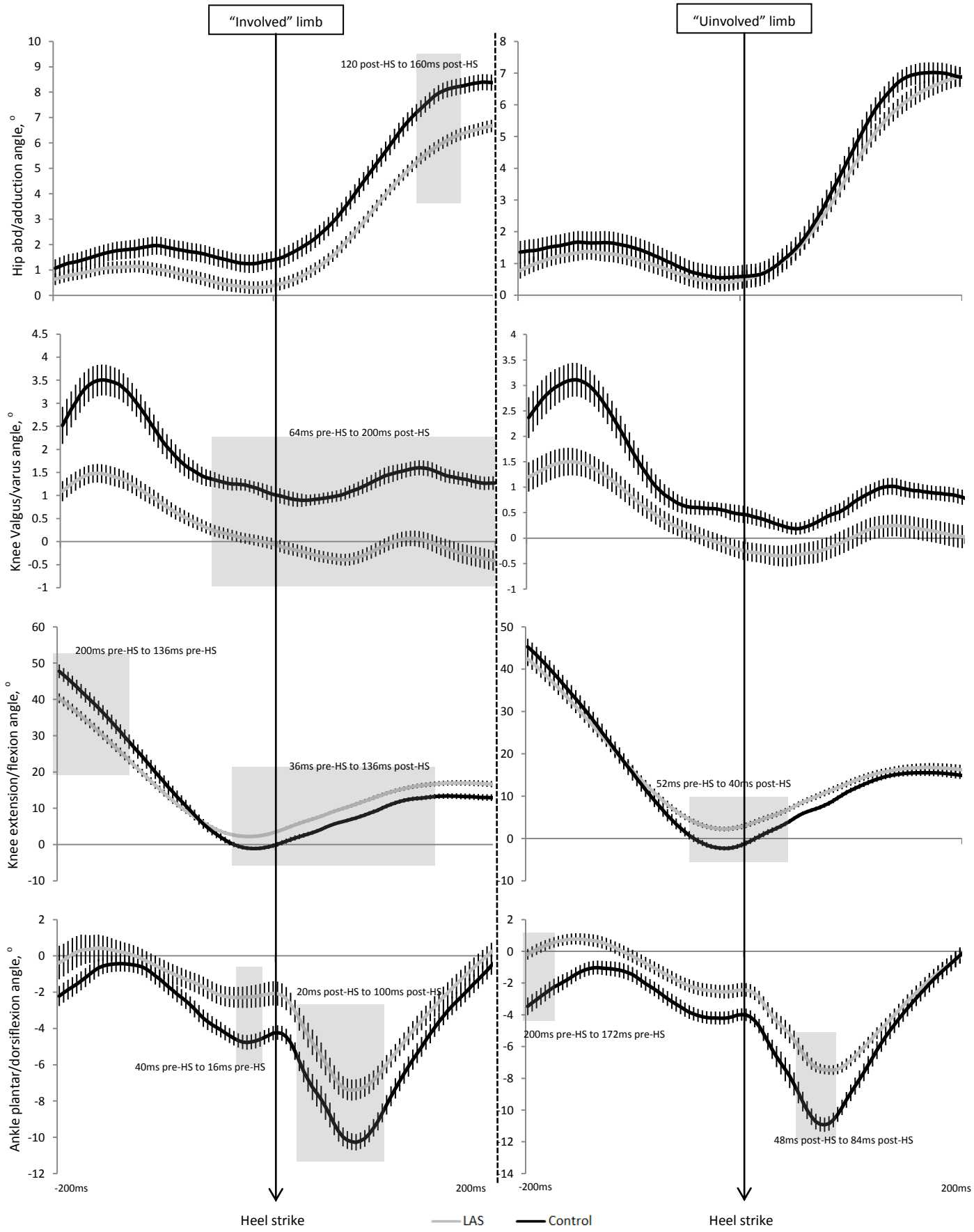

"Involved" limb
"Uinvolved" limb
Hip abd/adduction angle, °
120 post-HS to 160ms post-HS
Knee Valgus/varus angle, °
64ms pre-HS to 200ms post-HS
Knee extension/flexion angle, °
200ms pre-HS to 136ms pre-HS
36ms pre-HS to 136ms post-HS
52ms pre-HS to 40ms post-HS
Ankle plantar/dorsiflexion angle, °
20ms post-HS to 100ms post-HS
40ms pre-HS to 16ms pre-HS
200ms pre-HS to 172ms pre-HS
48ms post-HS to 84ms post-HS
-200ms
200ms
Heel strike
LAS
Control
Heel strike

**Figure 3**

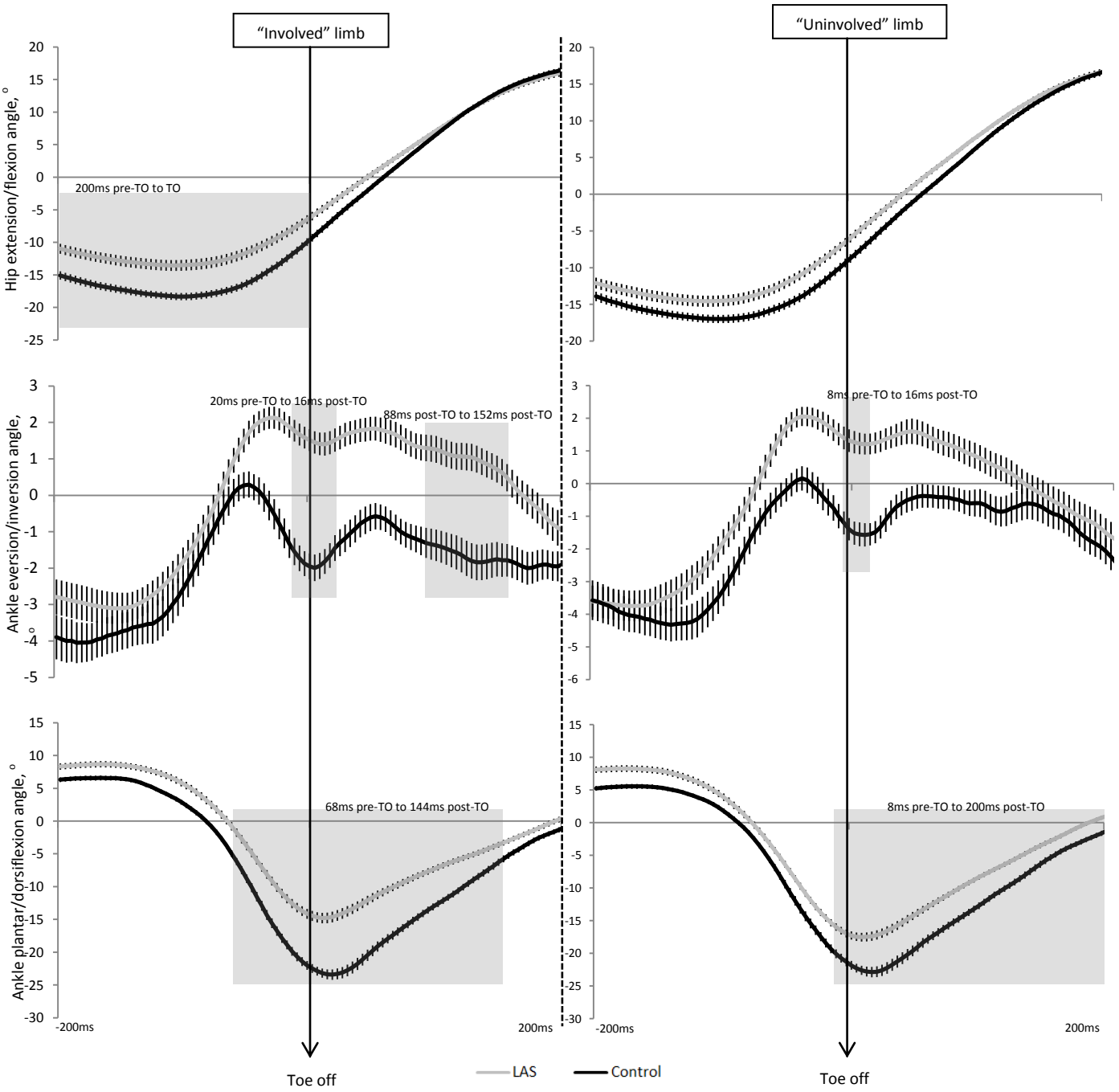

"Involved" limb
"Uninvolved" limb
Hip extension/flexion angle, °
200ms pre-TO to TO
Ankle eversion/inversion angle, °
20ms pre-TO to 16ms post-TO
88ms post-TO to 152ms post-TO
8ms pre-TO to 16ms post-TO
Ankle plantar/dorsiflexion angle, °
68ms pre-TO to 144ms post-TO
8ms pre-TO to 200ms post-TO
-200ms
200ms
Toe off
LAS
Control

**Figure 4**

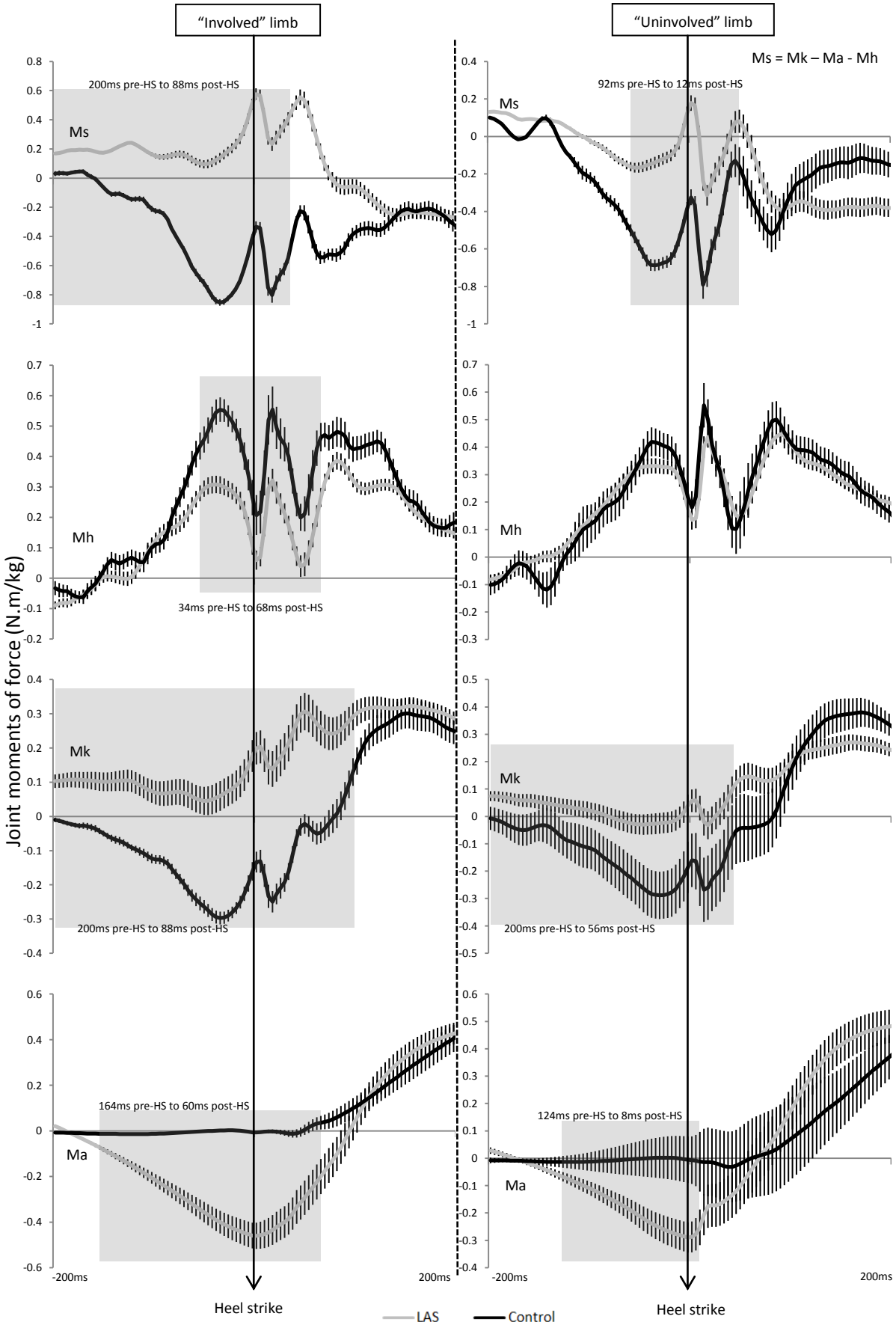

"Involved" limb
"Uninvolved" limb
Ms = Mk – Ma - Mh
Joint moments of force (N.m/kg)
Ms
Mh
Mk
Ma
200ms pre-HS to 88ms post-HS
92ms pre-HS to 12ms post-HS
34ms pre-HS to 68ms post-HS
200ms pre-HS to 88ms post-HS
200ms pre-HS to 56ms post-HS
164ms pre-HS to 60ms post-HS
124ms pre-HS to 8ms post-HS
-200ms
200ms
Heel strike
Heel strike
LAS
Control

**Figure 5**

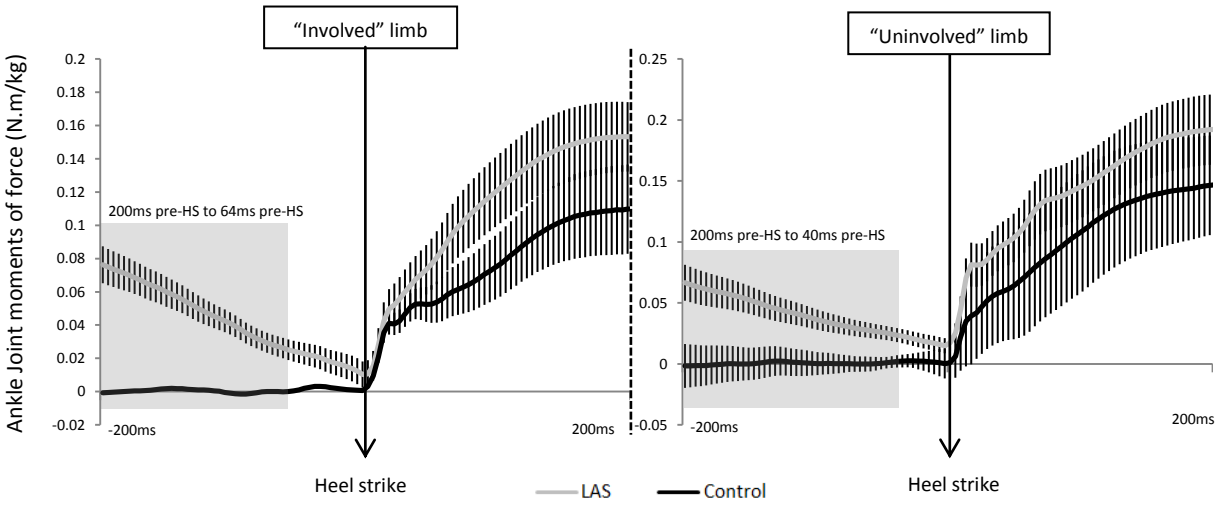

"Involved" limb
"Uninvolved" limb
Ankle Joint moments of force (N.m/kg)
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
-0.02
200ms pre-HS to 64ms pre-HS
-200ms
200ms
0.25
0.2
0.15
0.1
0.05
0
-0.05
200ms pre-HS to 40ms pre-HS
-200ms
200ms
Heel strike
Heel strike
LAS
Control

**Figure 6**

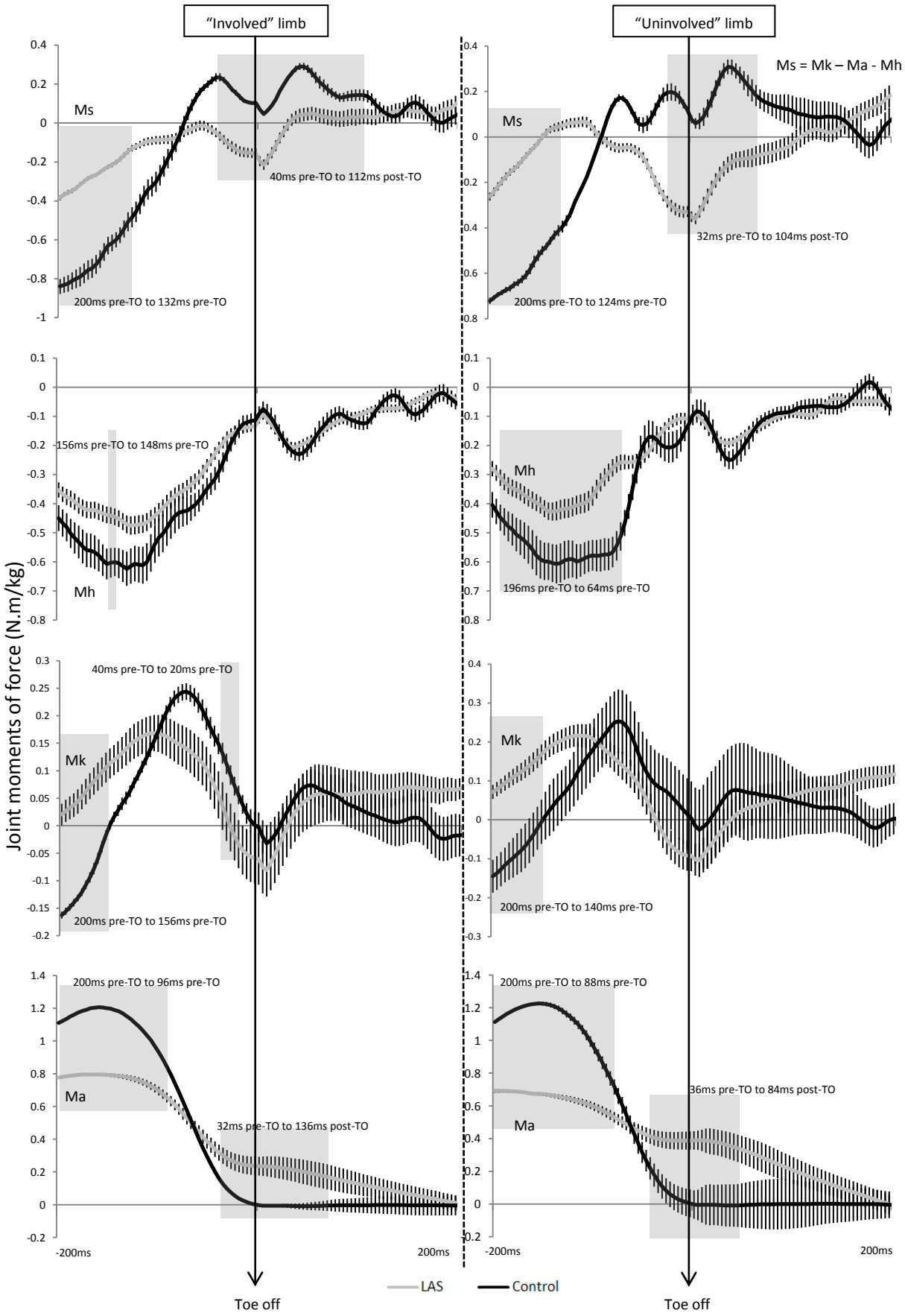
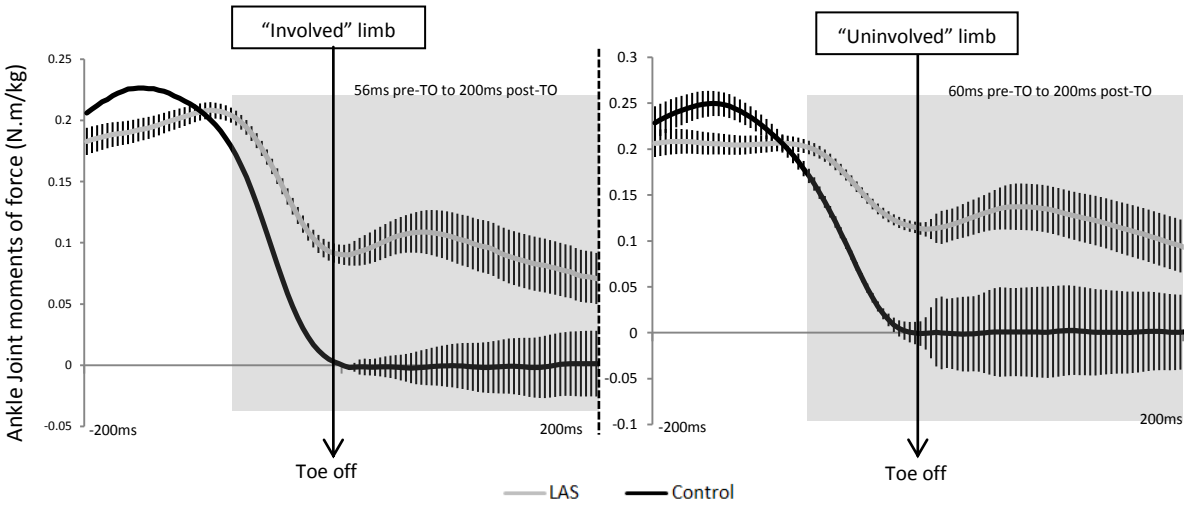

"Involved" limb
"Uninvolved" limb
Ms = Mk – Ma - Mh
Ms
40ms pre-TO to 112ms post-TO
200ms pre-TO to 132ms pre-TO
32ms pre-TO to 104ms post-TO
200ms pre-TO to 124ms pre-TO
156ms pre-TO to 148ms pre-TO
Mh
196ms pre-TO to 64ms pre-TO
40ms pre-TO to 20ms pre-TO
Mk
200ms pre-TO to 156ms pre-TO
200ms pre-TO to 140ms pre-TO
200ms pre-TO to 96ms pre-TO
Ma
32ms pre-TO to 136ms post-TO
200ms pre-TO to 88ms pre-TO
36ms pre-TO to 84ms post-TO
Joint moments of force (N.m/kg)
-200ms
200ms
LAS
Control
Toe off
Toe off

**Figure 7**


"Involved" limb
"Uninvolved" limb
Ankle Joint moments of force (N.m/kg)
56ms pre-TO to 200ms post-TO
60ms pre-TO to 200ms post-TO
-200ms
200ms
Toe off
LAS
Control