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Lower Extremity Joint Stiffness during Walking Distinguishes Children With and Without Autism

Jeffrey D. Eggleston, Ph.D.^{a,*}, John R. Harry, Ph.D.^b, and Janet S. Dufek, Ph.D.^c

^aDepartment of Kinesiology, University of Texas at El Paso, 500 University Avenue, El Paso, TX 79968, USA

^bDepartment of Kinesiology and Sport Management Texas Tech University, 2500 Broadway, Box 43011, Lubbock, TX 79409, USA

^cDepartment of Kinesiology and Nutrition Sciences, University of Nevada, Las Vegas 4505 South Maryland Parkway, Las Vegas, NV 89154, USA

Abstract

How children with Autism Spectrum Disorder (ASD) and peers with typical development (TD) modulate lower extremity stiffness during walking could identify a mechanism for gait differences between groups. We quantified differences in lower extremity joint stiffness and linear impulses, along the vertical and anterior/posterior axes during over-ground walking in children with ASD compared to age- and gender-matched children with TD. Nine age- and gender-matched pairs of children, aged 5–12 years, completed the current study. Joint stiffness and linear impulses were computed in four sub-phases of stance: loading response, mid-stance, terminal stance, and pre-swing. The Model Statistic technique ($\alpha=0.05$) was used to test for statistical significance between the matched-pairs for each variable and sub-phase. Furthermore, dependent *t*-tests ($\alpha=0.05$) were utilized, at the group level, to determine whether significant differences existed between sub-phases. Results indicate that children with ASD may exhibit greater stiffness in pre-swing, and thus, produce inefficient propulsive forces during walking. We attribute these differences to sensory processing dysfunction previously observed in children with ASD.

Keywords

Autism Spectrum Disorder; Joint Stiffness; Locomotion; Pediatric; Sensory Processing

1. Introduction

Autism Spectrum Disorder (ASD) is clinically characterized by core features such as deficits in social and language skills, movement stereotypy, restricted interests and hyper- and

*Corresponding Author Phone: 915-747-7208, jdeggleston@utep.edu.

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hyposensitivity to sensory stimuli (American Psychiatric Association, 2013; Baoi, 2010; Kim & Lord, 2013). However, recent hypotheses suggest movement quality should also be considered a core feature of the disorder (Dufek, Eggleston, Harry, & Hickman, 2017; Eggleston, Harry, Hickman, & Dufek, 2017; Hocking & Caeyenberghs, 2017; Moran, Foley, Parker, & Weiss, 2013) since motor dysfunction is present in as many 90% of children with ASD (David et al., 2009; Floris et al., 2016; Miyahara et al., 1997). Specifically, children with ASD were shown to exhibit both different movement patterns and lesser movement control over a variety of movements in comparison to children with typical development (TD) (Calhoun, Longworth, & Chester, 2011; Cook, 2016; Dufek et al., 2017; Eggleston et al., 2017; Fournier et al., 2010; Kindregan, Gallagher, & Gormley, 2015; May et al., 2016; Rinehart, Tonge, Bradshaw et al., 2006; Rinehart, Tonge, Iansek et al., 2006). Not only are these movement abilities different than their peers with TD, the degree of physical performance impairments is quite heterogeneous among children with ASD (Dufek et al., 2017; Eggleston et al., 2017). It has been stated that continued study of movement quality in this population could help explain the root cause of movement dysfunction in individuals with ASD (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; Hocking & Caeyenberghs, 2017; Kindregan et al., 2015; Mari, Castiello, Marks, Marraffa, & Prior, 2003; Moran et al., 2013; Weiss, Moran, Parker, & Foley, 2013) and provide a more comprehensive understanding of the disorder. Furthermore, continued experimentation relative to unique motor abilities may provide more detailed insight into specific intervention-responses among individuals with the disorder to improve overall treatment outcomes.

Gait abnormalities in children with ASD have been related to muscular weakness (Kindregan et al., 2015), hypotonia, akinesia, and bradykinesia (Damasio & Maurer, 1978; Kohen-Raz, Volkman, & Cohen, 1992). However, the specific neuro-musculo-skeletal parameter underlying the uniqueness of each child's developed movement pattern remains unknown. A potential variable to explain such unique physical presentations is lower extremity joint stiffness due to its importance during musculoskeletal movement performance (Butler, Crowell, & Davis, 2003) and its involvement in all human gait patterns. Joint stiffness is often quantified using a mass-spring model which describes the interaction between the body and the ground (Farley & Morgenroth, 1999) by dividing the torque about the joint by the angular displacement of the joint during. As joint angular position changes during walking, the stiffness of the joint also changes (Farley & Morgenroth, 1999). Individuals with TD voluntarily modify joint stiffness in response to changes within the environment (Ferris & Farley, 1997; Ford, Myer, & Hewett, 2010) during a movement task. However, individuals with ASD might not possess the ability to appropriately modulate lower extremity joint stiffness due to deficits in movement planning and spatial awareness (dyspraxia) (Dziuk et al., 2007; MacNeil & Mostofsky, 2012), as well as sensory perception (Kern et al., 2006). Examining lower extremity joint stiffness may provide insight into the neurophysiological underpinnings of motor planning and control associated with this disorder. Understanding how children with ASD, relative to children with TD, modulate lower extremity stiffness could identify a mechanism for gait differences observed between children with ASD and their peers with TD.

Increased joint stiffness is thought to be a compensatory strategy employed to maintain stability about the joint, as observed in individuals with knee osteoarthritis (Gustafson,

Gorman, Fitzgerald, & Farrokhi, 2016). However, insufficient joint stiffness can lead to excessive and unnecessary joint motion (Butler et al., 2003) due to a lack of dynamic joint stability (Ford et al., 2010). Increased stiffness could also lead to increased ground reaction force (GRF) impulse magnitudes, due to either increased torque or decreased angular motion about a joint. The combination of increased GRF impulse magnitudes and excessive joint motion could lead to issues relating to balance and stability challenges (Ament et al., 2015; Bugnariu et al., 2013; Memari et al., 2013) which may result in a trip or fall. Specific to ASD, increased joint stiffness might be employed at distal joints, such as the ankle, as a protective mechanism to reduce the risk of falling when the foot is in direct contact with the ground. Moreover, increased distal joints stiffness during the early portion of stance may provide evidence for a lesser ability to control the distal segments in anticipation for impact with the ground. The large number of significant differences previously observed in both vertical and anterior/posterior GRF trajectories during stance phase between children with ASD and their peers with TD (Dufek et al., 2017) indicates children with ASD might modulate their lower extremity joint kinematics prior to, and immediately upon ground contact in comparison to their peers with TD who adapt stiffness to dynamic changes in terrain or motor task. Because the foot is the only body part contacting the ground during bipedal walking, children with ASD may have a neurologic strategy to cautiously modulate the ankle joint stiffness because it is the most distal larger joint in the lower extremity which is responsible for ankle strategy balance response. These anticipated occurrences could explain the greater number of significant differences previously observed between children with ASD and matched peers with TD in hip joint angular positions versus ankle joint angular positions (Dufek et al., 2017).

The purpose of this investigation was to compare lower extremity joint stiffness between children with ASD and children with TD during the stance phase of over-ground walking using a matched-pair design. A secondary purpose was to determine whether differences in lower extremity joint stiffness coincided with different GRF magnitudes, as defined by GRF impulse. It was hypothesized that (a) greater magnitudes of joint stiffness would be observed in children with ASD, and (b) greater GRF impulse magnitudes would coincide with greater joint stiffness magnitudes. These hypotheses were based upon scientific speculation and the known presence of dyspraxia (Dziuk et al., 2007; MacNeil & Mostofsky, 2012), decreased coordination and smoothness during walking (Rinehart et al., 2006), and deficits of sensory perception (Kern et al., 2006) among children with ASD in comparison to children with TD.

2. Methods

2.1. Participants

Nine children with ASD and nine children with TD (5–12 years of age) participated in this study (14 males, 4 females; 9.0 ± 2.3 years, 1.4 ± 0.2 m, and 34.2 ± 14.0 kg, ASD; and 8.9 ± 2.1 years, 1.4 ± 0.2 m, and 36.3 ± 10.2 kg, TD). Participants were recruited from the community population through recruitment flyers which were dispersed to therapeutic clinical offering services to individuals with ASD. Further recruitment was conducted through the host university list-serve email list soliciting participants with ASD as well as children with TD. Each child with ASD was required to have a clinical diagnosis of the

disorder, and each child's parent(s) verbally verified the diagnosis. Children with TD were age- and gender-matched to a study-enrolled child with ASD. Varied gait patterns were not excluded to best replicate the heterogeneous presentations among children with ASD (Calhoun et al., 2011; Eggleston, Landers, Bates, Nagelhout, & Dufek, 2018; Eggleston et al., 2017; Kindregan et al., 2015). Parental consent and child assent were obtained prior to completing any study related activities as approved by the Institutional Review Board (protocol number: 710824), and in accordance with the Declaration of Helsinki. An *a priori* sample-size estimation was not performed due to the matched-pair study design in which a single child with ASD was compared to a single matched control with TD. In such a design, a greater number of trials or observations afford greater statistical power independent of the sample size (Bates, Dufek, James, Harry, & Eggleston, 2016).

2.2. Instrumentation

An eight-camera motion capture system (120 Hz, Vicon Motion Systems Ltd., Oxford, UK) was used to obtain three-dimensional kinematic data. Three-dimensional force data were synchronously collected with kinematic data via one Kistler (480 Hz; Kistler Instrument Corp., Amherst, NY, USA) and two AMTI (480 Hz, Advanced Mechanical Technology Inc., Watertown, MA, USA) force platforms mounted flush with the floor.

2.3. Procedures

Retro-reflective markers were adhered bilaterally on the following anatomical locations: base of the second toe, base of the fifth metatarsal, calcaneus, medial and lateral malleoli, medial and lateral knee joint line, anterior superior iliac spine, and posterior superior iliac spine. An additional marker was placed on the sacrum to aid in tracking pelvis motion. Four three-marker clusters were placed bilaterally at the lateral aspect of the thigh and leg. Participants completed 20 over-ground walking trials at a self-selected velocity. Walking velocity was not directly monitored to avoid masking the uniqueness among children with ASD (Calhoun et al., 2011; Dufek et al., 2017; Eggleston et al., 2017) that could occur using a controlled walking speed. To assist with participant management during procedures, the research team maintained casual conversations with participants to invoke a low-stress environment. During assent, research team members also described what would occur during the testing, in age-appropriate language, and told participants to ask any questions they wanted at any point. Furthermore, the assent document also contained images of what would be occurring during testing. If a participant had a question, we would refer back to the image and also use age-appropriate language to answer their question. When applying the retroreflective markers, research team members allowed participants to touch and feel the markers as we described how we would apply them. Furthermore, during testing, research team members offered praise to participants as they completed trials. This was performed to ensure participants completed testing to the best of their abilities and followed instructions from the research team.

2.4. Data Reduction and Analysis

Raw data were processed in the Visual 3D biomechanical software suite (C-Motion, Inc., Germantown, MD, USA). Marker trajectories and ground reaction force data were filtered with a low-pass, bi-directional Butterworth digital filter with cutoff frequencies of 6 and 25

Hz, respectively. Joint moments were calculated using inverse dynamics procedures. Joint angular positions of the hip, knee, and ankle joints were calculated such that positive magnitudes represented a flexed/dorsiflexed position. GRF data were extracted along the vertical (vGRF) and anterior-posterior (yGRF) axes. Data were then normalized to 101 data points to represent the stance phase of gait. To define the stance phase, heel strike and toe-off events were identified as the time when the vGRF increased above 20 N and the subsequent time when the vGRF decreased below 20 N, respectively. Data were then exported to Gnu Octave for further analysis using custom code.

Joint stiffness was calculated as the change in the joint moment divided by the change in joint angular displacement, as described by Farley and colleagues (1998). The stance phase of gait was divided into the following four sub-phases expressed as a percentage of stance (Rancho Los Amigos National Rehabilitation Center, 2001): loading response (LR; 0–17%), mid-stance (MS; 18–50%), terminal stance (TS; 51–83%), and pre-swing (PSw; 84–100%). Thus, stiffness values were calculated to represent the average stiffness throughout each sub-phase. GRF impulse magnitudes were calculated from the normalized vGRF and yGRF profiles for each sub-phase. Specifically, the integral of the vGRF and yGRF curves was calculated bilaterally with respect to the normalized time of the stance phase. Thus, four vGRF impulse and four yGRF impulse magnitudes were calculated per participant per limb representing the change in vertical and anterior-posterior momentum of the center of mass during each sub-phase.

2.5. Statistical Analysis

Data were analyzed on a matched-pair basis (Dufek et al., 2017). Mean and standard deviation values for each parameter were calculated per participant for each stance sub-phase. The Model Statistic technique (Bates, James, & Dufek, 2004) was used to test for statistical differences ($\alpha = 0.05$) in the joint stiffness values between each matched pair at each sub-phase. Thus, four statistical tests were conducted per variable per pair. The Model Statistic technique ($\alpha = 0.05$) was also used to quantify differences between the matched-pairs for vGRF and yGRF impulses at each sub-phase.

The joint stiffness magnitudes were also evaluated on a group level to determine if any of the stance sub-phases (LR, MS, TS, or PSw) were statistically significant from the others, when collapsed across participants and joints. Specifically, dependent *t*-tests ($\alpha = 0.05$) were utilized to test for statistically significant differences between the number of significant differences collapsed across all pairs for each of the four sub-phases.

3. Results

The Model Statistic analyses revealed statistically significant different magnitudes of joint stiffness between all children with ASD and their peers with TD. These differences were observed at all three lower extremity joints for all four sub-phases of stance. The children with ASD also exhibited highly individualized joint stiffness magnitudes, Tables 1 and 2 display the joint stiffness values for two exemplar pairs representing the greatest (Table 1) and fewest (Table 2) numbers of significant differences observed among the matched pairs.

A greater number significant differences were detected during PSw in comparison to the other three sub-phases when collapsed across the joints and pairs ($p = 0.001$, $p < 0.001$, and $p = 0.003$, for PSw in comparison to LR, MS, TS, respectively). No differences among any of the other sub-phases were detected. Table 3 illustrates the number of significant differences observed per joint and sub-phase.

Significant vGRF impulse differences were detected in no fewer than 33% of the matched pairs for any sub-phase and limb, with percentages for the left and right limbs, respectively, of 56% and 78%, 44% and 56% (MS and TS), and 56% to 33% (PSw). Significantly different vGRF impulse magnitudes were detected in a majority of the matched pairs (Table 4), with little consistency relative to the polarity of the mean differences. The yGRF impulse magnitudes were significantly different in no fewer than 22% of the matched pairs for any sub-phase and limb, with percentages for the left and right limbs, respectively, of 56% and 56% (LR), 56% and 22% (MS), 33% and 44% (TS), and 56% and 22% (PSw). Similar to the vGRF impulse data, significant differences in yGRF impulse were detected among the majority of the matched pairs (Table 5), with little consistency relative to the polarity of the mean differences.

4. Discussion

The purposes of this study were to examine lower extremity joint stiffness in children with ASD in comparison to children with TD during over-ground walking and to determine if different stiffness magnitudes coincided with different GRF impulse magnitudes. It was hypothesized that children with ASD would have both significantly greater lower extremity joint stiffness magnitudes in comparison to children with TD and significantly greater GRF impulse magnitudes. While the data support our hypotheses, PSw was the only sub-phase in which significantly greater stiffness was consistently observed for all joints for children with ASD.

Specifically, these findings revealed motor impairment during the latter portion of stance (when the foot interacts with the ground to complete the propulsive phase of gait). Recent research suggests increased stiffness magnitudes could be expected to coincide with an altered ability to generate appropriate magnitudes of propulsive force to produce forward locomotion (Dufek et al., 2017). As displayed in Table 5, five children in the current sample of children with TD exhibited increased yGRF impulse during PSw in comparison to their matched-pairs, whereas only one child with ASD exhibited an increase yGRF impulse, suggesting that children with ASD may demonstrate impairments during the latter portion of stance, leading to an inadequate generation of propulsive force. This increased stiffness likely precedes impaired movement patterns and control shortly after PSw during the swing phase of gait (Dufek et al., 2017; Eggleston et al., 2017). Interestingly, different stiffness values for the loading response, mid-stance, and terminal stance sub-phases were not a characteristic identified in this sample of children with ASD. Implementing gait training interventions aimed at improving propulsive mechanism efficiency for children with ASD could ensure that appropriate anatomical structures and mechanisms are utilized such that the risk of chronic over-use injuries and/or musculoskeletal conditions to structures not made for the specific task decreases.

When these data are considered alongside those of Rinehart and colleagues (Rinehart et al., 2006), Fournier et al. (2010), and Kanner (1943), children with ASD exhibit uncoordinated movement during toe off. This assumption is supported by the vGRF and yGRF impulse magnitudes from the current study which identify how the body produces (*e.g.* propulsive toe-off force) force over a specific time. Due to the lack of propulsive force generation during PSw, other mechanical mechanisms must compensate to facilitate forward locomotion and precipitate altered gait mechanics throughout the remainder of the gait cycle. The markedly different gait mechanics of individuals with ASD compared to individuals with TD has been attributed to dysfunction in motor cortex segregation (Nebel et al., 2014) and the function of the cerebellum (D'Mello, Moore, Crocetti, Mostofsky, & Stoodley, 2016; Mosconi, Wang, Schmitt, Tsai, & Sweeney, 2015) and basal ganglia (Leisman, Braun-Benjamin, & Melillo, 2014; Radonovich, Fournier, & Hass, 2013; Shetreat-Klein, Shinnar, & Rapin, 2014; Subramanian et al., 2017). Furthermore, it is likely that one, or a combination of the brain structures affected by ASD, leads to these individuals not generating optimal propulsive force, due to a lack of motor planning.

While joint stiffness is continually modulated during gait, excessive stiffness can lead to bony tissue injuries during locomotion (Butler et al., 2003). Although the children in the current study were performing a fundamental movement (walking), walking requires many consecutive foot strikes on the ground over time. Based on the vGRF impulse magnitudes, only two children with TD exhibited statistical increases in impulse magnitudes, whereas five children with ASD exhibited statistical increases in vGRF impulse magnitudes. Additionally, six children with ASD exhibited greater vGRF impulse magnitudes during mid-stance, when body weight is located directly over the ground-contacted foot. This may indicate that a large magnitude of force is still being applied to and through the body during mid-stance. In mid-stance, only one child with TD exhibited a statistical increase in mid-stance vGRF impulse in comparison to their matched-peer. Furthermore, continued impact with the ground while experiencing higher-than-needed vGRF impulse and employing increased stiffness about the lower extremity joints places greater stress of the musculoskeletal system to attenuate the excess impact forces. Attenuating unnecessarily high impact forces could be a catalyst for damage to the involved musculoskeletal structures (Collins & Whittle, 1989). However, the individualized stiffness and vGRF impulse values observed suggests children who exhibit decreased joint stiffness may be at lesser risk for bony tissue injuries (Butler et al., 2003). However, more direct evaluations of injury risk and/or occurrence are needed to determine whether different magnitudes of joint stiffness increase the risk for soft or bony tissue injuries.

Despite the current results, it remains unclear whether individuals with ASD can effectively modulate lower extremity stiffness in response to environmental stimuli similarly to neurotypical individuals (Ford et al., 2010). A hallmark feature of children with ASD is the lack of accurate sensory perception, which can be either hyper- or hyposensitive relative to internal or external stimuli (Kern et al., 2006). Thus, differences in joint stiffness may be a consequence of inaccurate processing of proprioceptive stimuli (Kern et al., 2006) in children with ASD. This assumption may be supported by the differences observed herein during mid-stance and terminal stance for the vGRF impulse magnitudes. If a child with ASD is incapable of accurately processing proprioceptive information, the neurological

system may not have the ability to make appropriate alterations to the movement pattern when their center of mass is moving forward in response to the movement of the lower-limbs. However, proprioceptive sensory processing might improve as a child with ASD grows older such that processing becomes more similar to that of individuals with TD (Kern et al., 2006) and the ability to modulate joint stiffness is refined. Nevertheless, there may be a continued inability to adequately modify joint stiffness when faced with novel stimuli. Various training modalities could be tested to offer additional insight into the improvement motor performance through the potential variety of kinematic and kinetic motor outputs, and thus, provide a more comprehensive understanding of ASD, and more importantly, the motoric impairments associated with the disorder. Additionally, it is also recommended that tactile and proprioceptive differences be addressed with interventions, which may further assist in improving motor performance and sensory processing.

4.1. Limitations

The limitations of this study include a small sample size, which has the potential to threaten the findings of the study. However, the authors believe this limitation is mitigated due to the single-subject study design utilized. The statistical methods utilized in the current study rely more on the number of observations completed by each participant rather than the total number of participants in the sample. Nevertheless, the authors acknowledge the challenges of generalizing these findings to the greater population. Still, it is important for future research to build upon these findings and use larger sample sized to determine if the findings in the current sample do represent other children with ASD. An additional limitation of the current study is not controlling for walking velocity. While other studies examining gait in children with ASD did not control for walking velocity, inter-trial velocity variability may have influenced the observed results. It is possible, however, that controlling for walking velocity might alter an individuals' walking mechanics, thus influence the outcomes as well.

5. Conclusion

This analysis revealed that children with ASD may exhibit differences in lower extremity joint stiffness during the PSw phase of gait when compared to matched-peers with TD. Greater stiffness coincided with an inefficient generation of propulsive impulse. Together, these outcomes may trigger many of the observed gait related differences in children with ASD (Dufek et al., 2017; Eggleston et al., 2017; Kindregan et al., 2015). Additionally, it is plausible that many of the observed differences are due to known dysfunction in the motor cortex, cerebellum, and basal ganglia, causing ineffective movement patterns. These findings support the movement that motor dysfunction should be included as a core symptom of ASD (Dufek et al., 2017; Eggleston et al., 2017; Fournier et al., 2010; Hocking & Caeyenberghs, 2017; Moran et al., 2013; Weiss et al., 2013). Additionally, these findings suggest a need for interventions to improve propulsive force generation and address other motor and sensory impairments observed during walking in hopes of providing improved quality of life of individuals with the disorder (Provost, Lopez, & Heimerl, 2007) , although further research is needed to determine effective interventions which may assist in providing such therapy to children with ASD.

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Highlights

- Children with ASD exhibit differences in lower extremity joint stiffness.
- Stiffness differences contribute to inefficient push-off during gait.
- Differences may be due to sensory processing dysfunction in children with ASD.

Table 1.

Joint Stiffness Values for a Pair of Children with ASD and TD Exhibiting the Greatest Number of Statistically Significant Differences.

Hip	Left		Right	
	ASD	TD	ASD	TD
LR	0.057±0.047	0.553±1.477	0.054±0.411	0.458±0.662
MS	0.019±0.007	0.014±0.008	0.022±0.004	0.024±0.007
TS	0.037±0.017	0.053±0.026	0.053±0.022	0.059±0.033
PSw	10.579±3.227	17.591±2.305 *	10.319±2.136	18.496±2.331 *
Knee	left		Right	
	ASD	TD	ASD	TD
LR	0.082±0.021	0.126±0.051 *	0.052±0.012	0.115±0.068 *
MS	0.051±0.008	0.135±0.075 *	0.049±0.011	0.106±0.059 *
TS	0.064±0.198	-0.097±0.240	0.037±0.073	-0.062±0.180
PSw	-12.368±3.120	-18.930±1.916 *	-6.175±2.931	-8.897±2.829
Ankle	left		Right	
	ASD	TD	ASD	TD
LR	0.053±0.105	-0.179±0.315 *	0.069±0.052	0.005±0.042 *
MS	0.074±0.362	0.177±0.113	-0.005±0.115	0.118±0.063 *
TS	0.011±0.234	0.903±3.755	-0.073±0.050	-1.890±6.395
PSw	7.311±3.236	-7.514±2.509 *	9.476±2.530	-4.312±2.257 *

Note. All stiffness values are in Nm/deg. LR = loading response, MS = mid-stance, TS = terminal stance, and PSw = pre-swing.

* Asterisks denote statistically significant differences between the matched-pair. Increased values indicated greater stiffness in the respective joint, at the respective sub-phase.

Table 2.

Joint Stiffness Values for the Pair of Children with ASD and TD Exhibiting the Fewest Number of Statistically Significant Differences.

Hip	Left		Right	
	ASD	TD	ASD	TD
LR	0.087±0.157	0.404±1.232	0.817±0.998	0.247±1.394
MS	-0.001±0.020	0.001±0.011	0.007±0.012	0.019±0.011
TS	0.012±0.029	0.029±0.024	0.020±0.014	0.036±0.017
PSw	-1.292±4.445	7.744±0.921 *	-2.167±2.294	4.101±1.624 *
Knee	left		Right	
	ASD	TD	ASD	TD
LR	0.134±0.131	0.092±0.052	0.051±0.010	0.070±0.042
MS	0.17±0.187	0.247±0.210	0.676±1.034	0.299±0.474
TS	-0.011±0.110	-0.041±0.161	0.008±0.047	-0.458±1.902
PSw	-20.998±2.530	-16.838±2.525 *	-27.028±3.457	-21.685±3.167 *
Ankle	left		Right	
	ASD	TD	ASD	TD
LR	-0.032±0.180	-0.097±0.282	0.052±0.023	-0.091±0.391
MS	-0.125±0.870	0.193±0.121	0.060±0.083	0.150±0.102
TS	-0.003±0.207	0.022±0.535	-0.002±0.031	-0.016±0.543
PSw	-3.901±2.351	-9.090±1.799	-7.806±2.642	-9.146±3.141 *

Note. All stiffness values are in Nm/deg. LR = loading response, MS = mid-stance, TS = terminal stance, and PSw = pre-swing.

* Asterisks denote statistically significant differences between the matched-pair. Increased values indicated greater stiffness in the respective joint, at the respective sub-phase.

Table 3.

Number of Significant Differences per Lower Extremity Joint and Stance Sub-Phase

Sub-phase	Hip		Knee		Ankle		Total
	Left	Right	Left	Right	Left	Right	
Loading Response	0	1	4	2	1	1	9*
Mid-stance	2	3	2	1	2	2	12*
Terminal Stance	4	4	1	0	0	0	9*
Pre-swing	7	6	6	6	7	8	40
Joint Sub-total	13	14	13	9	10	11	

Note. All values are the number of pairs that exhibited statistically significant differences in stiffness values, based upon the Model Statistic technique ($\alpha = 0.05$).

* The asterisk denotes a statistical difference, revealed by dependent t -tests, between the total number of significant differences, collapsed across limbs and joints, between pre-swing and the remaining three stance sub-phases.

Table 4. vGRF Impulse Magnitudes for Each Participant and Matched Pair During the Stance Sub-Phases.

Sub Phase	Matched Pair	Left Strides			Right Strides			TD	
		Me an	S D	ASD	Me an	S D	ASD		
L R	1	6.5	1.5	9.1*	1.2	6.2	1.0	7.9*	2.4
	2	8.9	6	1.7	3	2.9	3	7	0.8
	3	9.7*	0	1.6	0	1.9	6	9	1.2
	4	11.4*	8	1.7	2	0.1	10.5	7	1.4
	5	8.2	4	2.1	2	1.5	5.6	5	1.4
	6	7.5	9	0.7	8	0.6	6.8	1	2.7
	7	13.2*	6	1.7	1	1.1	13.7	2	1.2
	8	13.4*	5	2.8	9	0.1	14.3	1	2.9
M S	9	6.5	4	2.9	2	1.8	8.4	7	1.1
	1	17.7	8	0.1	18.4	0	1.1	17.8	8
	2	16.9	0	1.1	17.7	1	1.1	16.8	3
	3	19.0	3	2.1	6	0.1	19.0	3	1.0
	4	23.9*	1	3.1	1	2.1	21.9	9	1.0
	5	17.5	9	1.2	2	1.1	19.9	0	2.3
	6	19.2*	1	1.1	5	0.1	17.4	1	2.9
	7	17.8	9	0.1	17.1	4	1.1	18.5	1
8	20.5*	0	3.1	7	0.1	20.4	1	1.4	

Sub Phase	Match ed Pair	Left Strides				Right Strides							
		ASD		TD		ASD		TD					
		Me an	S D	Me an	S D	Me an	S D	Me an	S D				
TS	9	17.4	4	4.	18.4	7	0.	21.3 *	6	1.	17.9	1	1.
	1	19.1	1	1.	18.7	7	1.	18.7	9	0.	17.2	2	5.
	2	19.2	8	0.	18.0	1	1.	18.2	7	0.	18.4	0	1.
	3	16.1	9	2.	18.2	6	0.	15.8	6	1.	18.7 *	6	0.
	4	14.5	4	2.	16.5 *	9	1.	14.2	0	3.	17.9 *	2	1.
	5	17.3	6	0.	17.0	5	1.	16.5	6	1.	16.9	5	1.
	6	17.5	7	0.	18.6 *	0	1.	16.8	2	2.	18.2	3	3.
	7	14.5	3	1.	16.7 *	9	0.	14.6	7	0.	16.6 *	6	0.
	8	22.6 *	7	0.	18.6	7	0.	22.6 *	4	1.	18.3	9	0.
PS w	9	15.8	9	2.	16.5	6	1.	16.3	6	1.	17.5	8	0.
	1	3.6	3	1.	4.5	0	1.	4.9*	2	1.	3.4	8	1.
	2	3.3	2	1.	3.7	1	1.	2.9	9	0.	4.1	5	1.
	3	2.2	7	1.	5.1*	6	1.	4.9	1	1.	5.4	1	1.
	4	3.5	4	2.	2.5	2	0.	3.9	1	2.	4.2	6	1.
	5	3.6	6	2.	2.1	0	1.	4.5*	3	1.	2.1	0	1.
	6	3.5	0	1.	5.2*	3	1.	4.5	3	2.	4.5	8	0.
	7	2.0	7	0.	4.1*	5	1.	1.3	8	0.	3.9*	9	0.
	8	1.9	0	1.	4.5*	6	1.	3.0	1	2.	3.5	8	0.
9	3.4	1	2.	2.1	6	0.	3.1	9	1.	4.6	7	1.	

Note: All magnitudes are in body weight * seconds (BW-s). LR = loading response; MS = mid-stance; TS = terminal stance; PSw = pre-swing; Mean = average across trials; SD = $\pm$ standard deviation across trials;

* = significantly greater than ASD/TD for the respective limb (left strides/right strides) and sub-phase ($p < 0.05$).

Table 5.

Anterior-Posterior Impulse Magnitudes for Each Participant and Matched Pair During the Stance Sub-Phases.

Sub Phase	Match ed Pair	Left Strides				Right Strides			
		ASD		TD		ASD		TD	
		Me an	S D	Me an	S D	Me an	S D	Me an	S D
L R	1	- 0.76	0.13	- 1.73 *	0.42	- 1.13	0.29	- 1.75 *	0.61
	2	- 1.30	0.49	- 1.38	0.55	- 1.75 *	0.36	- 1.38	0.09
	3	- 1.48 *	0.23	- 0.84	0.26	- 1.76 *	0.57	- 0.83	0.27
	4	- 1.57 *	0.61	- 1.07	0.40	- 1.75	0.74	- 1.53	0.27
	5	- 2.08	0.93	- 2.06	0.23	- 0.96	0.36	- 2.03 *	0.54
	6	- 1.38	0.31	- 1.26	0.18	- 1.29	0.34	- 1.32	0.27
	7	- 1.95 *	0.29	- 1.43	0.25	- 2.51 *	0.35	- 1.59	0.48
	8	- 2.67 *	0.61	- 1.84	0.30	- 2.59	1.00	- 2.01	0.44
	9	- 1.18	0.72	- 1.77	0.34	- 1.72	0.72	- 1.53	0.24
M S	1	- 1.01	0.24	- 1.72 *	0.47	- 1.86	0.66	- 1.94	0.90
	2	- 0.55	0.39	- 1.48 *	0.27	- 1.31	0.71	- 1.53	0.41
	3	- 1.16	0.64	- 1.18	0.30	- 1.76 *	0.65	- 1.14	0.23
	4	- 1.13 *	0.61	- 0.62	0.02	- 1.66	0.86	- 1.77	0.64
	5	- 1.87	0.35	- 1.87	0.56	- 1.93	0.88	- 1.54	0.53
	6	- 2.24 *	0.53	- 1.48	0.28	- 1.31	0.44	- 1.31	0.43
	7	- 1.05	0.27	- 1.54 *	0.43	- 1.54	0.41	- 1.32	0.54
	8	- 1.53	0.88	- 1.46	0.45	- 0.77	0.87	- 1.30	0.30
	9	- 1.61	1.11	- 1.71	0.37	- 2.51 *	0.80	- 1.70	0.33
T S	1	1.40	0.45	1.94	0.61	0.87	0.53	1.22	0.91
	2	1.50	0.28	2.04	0.57	1.86	0.67	1.36	0.57
	3	1.99 *	0.22	0.67	0.45	1.88 *	0.35	0.74	0.35

Sub Phase	Match ed Pair	Left Strides				Right Strides			
		ASD		TD		ASD		TD	
		Me an	S D	Me an	S D	Me an	S D	Me an	S D
	4	1.64 *	0.33	1.57	0.25	1.56	0.68	1.08	0.77
	5	1.74	0.88	1.71	0.60	1.41	0.19	2.40 *	0.58
	6	1.40	0.46	1.13	0.23	1.35	0.81	1.07	0.48
	7	2.73 *	0.43	1.67	0.59	2.32 *	0.55	1.64	0.37
	8	2.16	0.95	1.56	0.53	2.04 *	0.69	1.50	0.27
	9	1.62	0.59	1.78	0.32	1.54	0.51	1.51	0.40
P S	1	0.97	0.33	1.57 *	0.39	1.12	0.21	1.03	0.55
	2	1.18	0.31	1.27	0.38	1.00	0.23	1.24	0.41
	3	0.63	0.48	1.05	0.28	1.40 *	0.20	1.16	0.32
	4	0.62	0.47	0.79 *	0.13	0.84	0.51	1.07	0.20
	5	0.88	0.55	0.75	0.34	1.07	0.42	0.92	0.36
	6	1.05	0.29	1.33 *	0.31	1.05	0.33	1.14	0.23
	7	0.81	0.29	1.36 *	0.42	0.47	0.30	1.35 *	0.23
	8	0.67	0.34	1.30 *	0.34	0.96	0.65	0.96	0.21
	9	1.12	0.61	0.68	0.20	0.94	0.69	1.33	0.40

Note: All magnitudes are in body weight * seconds (BW-s). LR = loading response; MS = mid-stance; TS = terminal stance; PS = pre-swing; Mean = average across trials; SD = $\pm$ standard deviation across trials;

* = significantly greater than ASD/TD for the respective limb (left strides/right strides) and sub-phase ($p < 0.05$).