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Foot hyperpronation alters lumbopelvic muscle function during the stance phase of gait

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ABSTRACT

Background: Although altered muscular control of the lumbopelvic region is one of the main risk factors for the development of low back pain and dysfunction, the influence of abnormal foot posture on lumbopelvic muscular function has not been investigated.
Research question: To determine possible functional changes due to hyperpronation in the main muscles that control the lumbopelvic segment.
Methods: Kinematic and kinetic data were collected from 15 persons with hyperpronated feet and compared to a control group of 15 persons with normally aligned feet during the stance phase of gait. A generic OpenSim musculoskeletal model was scaled for each participant. A computed muscle control approach was used to produce a forward dynamic simulation of walking to determine muscle function.
Results: In the hyperpronation group significantly greater peak forces were observed in the erector spinae, iliopsoas and abdominals compared to controls. The former group showed peak latencies for abdominal muscles during early stance, and for erector spinae muscles during both early and late stance. No significant between-group differences were found in gluteus maximus muscle activation in the stance phase of gait.
Significance: Abnormal foot pronation can change the timing and intensity of lumbopelvic muscle activation. These changes may predispose people to develop secondary dysfunctions.

1. Introduction

Among atypical foot mechanics, hyperpronation attracts much clinical attention because of its high incidence. Subtalar joint dysfunction as a consequence of hyperpronation alters normal biomechanics of the foot segment during the stance phase of gait. It is traditionally believed that biomechanical changes due to hyperpronation can be associated with lower extremity overuse injuries [1], as a result of altered force distribution and abnormal kinematic patterns. Moreover, it has been shown that in the normal population, bilateral and unilateral hyperpronation induced by a wedge can potentially propagate kinematic changes to the pelvis segment, generating anterior and lateral pelvic tilt [2], respectively. It is believed that abnormal anterior or lateral deviation of the pelvic segment may lead to lumbar hyperlordosis [3] or scoliosis [4], thereby resulting in low back dysfunction and pain. It has been also reported that patients with chronic low back pain tend to walk with excessive anterior pelvic tilt [5].

Although many studies have focused on the kinematic effects of hyperpronation on proximal segments, less attention has been given to possible changes in muscle function that can occur due to altered foot posture. There is limited evidence of increased electromyographic activity in the invertor muscles and decreased electromyographic activity in the evertor muscles in people with hyperpronated feet compared to those with normal foot alignment [6]. However, the available literature regarding the influence of foot hyperpronation on muscular function has not been adequately investigated.

Considering the role of the pelvis as a functional link between the lower extremities and lumbar spine, and the suggested interaction between lower extremity and trunk segment movements [7], a

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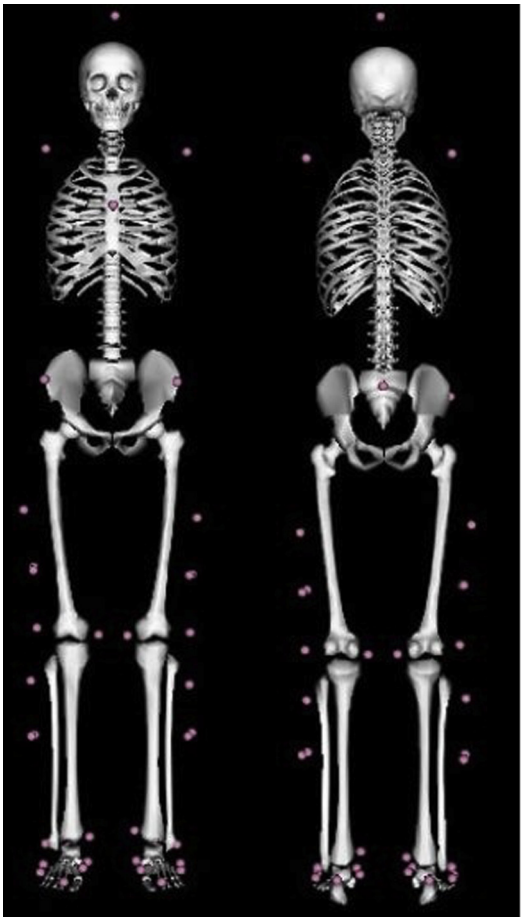

Opensim marker set up

TopHead	Acromium R/L
Sternum	ASIS R/L
V.Sacral	Thigh.Upper R/L
Thigh.Front R/L	Thigh.Rear R/L
Knee.Lat R/L	Knee.Med R/L
Shank.Upper R/L	Shank.Front R/L
Shank.Rear R/L	Ankle.Lat R/L
Ankle.Med R/L	Heel R/L
Midfoot.Sup R/L	Midfoot.Lat R/L
Toe.Lat R/L	Toe.Med R/L
Toe.Tip R/L	

Fig. 1. OpenSim marker set up; R = right, L = left, ASIS = anterior superior iliac spine, Med = medial, Lat = lateral, Sup = supetior, Tip = tip toe.

relationship can be hypothesized between abnormal foot posture and changes in lumbopelvic muscle function. Moreover, anteriorly tilted pelvic position may significantly increase the strain on the lumbopelvic musculature. Additionally, it was suggested that any dysfunction in the lumbopelvic musculature due to impaired force generation capacity and neuromuscular activation maybe associated with low back pain and injuries [8]. In this connection, it has been shown that patients with low back pain have altered motor control [9] and abnormal trunk muscle activities [10]. An association has also been postulated between hyperpronated foot posture and intermittent low back pain [1], although the exact etiology for this pathophysiological connection is not clear.

To the best of our knowledge, no previous research has been conducted to evaluate muscle function in the lumbopelvic region during gait in the population with true pronation. Because walking is a routine daily living activity with a high motor control demand, we believed that possible abnormal lumbopelvic muscle functioning due to hyperpronation may be detectable during this dynamic task. Thus the purpose of this study was to seek an explanation for possible changes in lumbopelvic muscular function due to foot hyperpronation during the stance phase of gait in participants with hyperpronated feet compared to a control group of participants with normally aligned feet. We hypothesized that there would be a significant between-group difference in muscle function variables in the lumbopelvic region. In-depth evaluation of lumbopelvic muscle behavior in persons with hyperpronated feet may yield further insights into the possible effect of subtalar dysfunction on the development of low back pain and dysfunction.

2. Materials and methods

2.1. Participants

Thirty asymptomatic women aged between 18 and 30 years (15 with neutrally aligned feet and 15 with flexible hyperpronated feet) participated in this cross-sectional study. Prior to participation, all participants were informed about the nature of the study and were asked to sign an informed consent form, approved by the Shiraz University of Medical Sciences Ethics Research Committee. The reason for recruiting sex-specific participants in the present study was the reported gender difference in pelvic–hip complex kinematics [11] and muscle force generation capacity [12], which may confound the results.

The six-item Foot Posture Index (FPI-6) [13] which is a valid and reliable rating system for multiple-segment assessments of foot posture was used for group allocation. The participants were considered either hyperpronated or normal according to reference values provided for the FPI-6, with a score of 0 to +5 indicating normal foot posture, a score of +6 to +9 indicating pronated foot and a score of 10 or higher indicating hyperpronation. The single heel raise test [14] was used to select flexible hyperpronated feet subjects among volunteers with hyperpronation.

Participants in the normal group were matched to participants in the hyperpronation group for anthropometric variables. In both groups the right lower limb was dominant. The dominant lower limb was determined by asking participants to kick a ball, and whichever leg they used for the kick was considered the dominant limb.

The exclusion criteria were any history of surgery or injuries to the lumbopelvic complex or the lower limbs, functional or structural orthopedic conditions that could interfere with the normal walking, such

Table 1
Participants' demographic characteristics.

	Group	N	Mean (SD)	Range	P value
Age (years)	NL	15	21.86 (3.48)	18-30	0.88
	HP	15	21.62 (3.34)	18-30	
Height (cm)	NL	15	157.46 (4.67)	153-168	0.78
	HP	15	156.67 (4.31)	151-165	
Body mass (kg)	NL	15	59.72 (3.34)	54.54-66.07	0.71
	HP	15	58.21 (3.02)	53.21-65.72	
Walking speed (m/s)	NL	15	1.27 (0.15)	0.99-1.45	0.78
	HP	15	1.28 (0.16)	0.98-1.45	

N: number of participants in each group; SD: standard deviation; NL: normal; HP: hyperpronation.

as abnormal knee varus or valgus, chronic pain due to structural or functional problems in the lower limb and lumbopelvic complex, neurological ailments affecting gait [15], limb length discrepancy greater than 10 mm [16].

All objective measurements used in this study were obtained by the same experienced physical therapist in all participants to avoid possible inter-examiner discrepancies.

2.2. Measurements and procedures

Kinematic data were collected with an eight-camera motion analysis system (Proreflex, Qualisys Track Manager®Ltd., Gothenburg, Sweden) at a sampling rate of 120 Hz. Ground reaction force (GRF) data were collected with a single force plate (Kistler Instrument®, Winterthur, Switzerland) placed underneath an 8-m walkway, at a sampling rate of 240 Hz.

To obtain anthropometric data and build a biomechanical model, retroreflective calibration markers 19 mm in diameter were placed on each participant in accordance with the OpenSim marker set on the anatomical points (Fig. 1).

After multiple practice trials, five acceptable barefoot walking trials at a comfortable self-selected speed were recorded for each participant. Only trials in which the participant's dominant foot landed on the force plate were used for further analysis.

2.3. Data reduction

Data were synchronously recorded with Qualisys Track Manager (QTM) software (Qualisys Track Manager®Ltd., Gothenburg, Sweden) and then imported into the OpenSim software. A generic OpenSim musculoskeletal model with 23 degrees of freedom and 92 muscles [17] was scaled for each participant to match her anthropometric characteristics.

OpenSim inverse kinematic analysis was used to calculate joint angles across the stance phase. Then the inverse dynamics problem was solved to determine external joint torques. Next, a residual reduction algorithm was used to improve dynamic consistency between kinematic parameters of the model and measured GRFs. Muscle forces were then computed with the computed muscle control (CMC) approach. This technique found a set of muscle excitations that closely tracked participants' motions within a forward dynamic simulation via feedback and feedforward control [17].

Force patterns of the five trunk muscles (primary controllers of the lumbopelvic segment in the sagittal plane) were determined based on musculoskeletal simulation: erector spinae, iliopsoas, external oblique abdominal, internal oblique abdominal and gluteus maximus. The vertical component of GRF was used to accurately determine stance phase. Muscle force time series were normalized by body weight, and were interpolated to 101 data points, each point representing one percentage of the stance phase. Peak normalized muscle forces and timing of these peaks were extracted from each time series during the discrete subphases of the stance: first double support (DS1), single leg support (SLS) and second double support (DS2).

In addition to the force plate data, the subphases were defined with a high-pass algorithm to detect toe off and heel strike events in the left foot. These events were visually verified to prevent any possible incorrect event detection.

2.4. Statistical analysis

The normality of data set distribution was confirmed with Shapiro–Wilk's test. Group differences in peak normalized muscle forces and peak times were compared with independent-sample *t*-tests. All statistical analyses were done with IBM® SPSS version 21.0 (IBM Corp, Armonk, NY, USA) with a significance level of 0.05.

3. Results

There were no statistically significant differences between groups in any of the demographic characteristics (Table 1). No significant difference was found in FPI scores between the dominant (10.6 ± 0.7) and non-dominant limb (10.3 ± 0.8) in the hyperpronation group ($p = 0.2$).

According to Hicks et al. [18] low average root mean square of marker tracking error across inverse kinematic solutions (7.6 mm) and low average root mean square of residual forces were detected (less than 5 N) during simulation steps.

In the hyperpronation group, greater normalized peak forces were found in erector spinae muscle at DS1, SLS and DS2 ($P = 0.02$, $P = 0.02$ and $P = 0.03$ respectively). Moreover, significant between-group differences were found for time to peak values in this muscle at DS1and DS2 ($P < 0.01$, $P = 0.01$ respectively). In other words, in the hyperpronation group, peak values in this muscle were significant delayed compared to the control group (Fig. 2) (Table 2).

Peak iliopsoas muscle forces were greater in the hyperpronation group during DS2 compared to controls ($P = 0.01$). No significant between-group differences were found for the time to peak variables (Fig. 2) (Table 2).

Not significant between-group differences were found in normalized gluteus maximus muscle forces and time to peak variables in the stance phase of gait (Fig.2) (Table 2).

Participants with hyperpronated feet also produced greater normalized peak internal oblique abdominal muscle forces during the entire stance phase compared to the control group. During DS1, this muscle reached its peak force value with a significant delay compared to the control group ($P = 0.02$) (Fig. 2) (Table 2).

During DS1, both peak force and time to peak variables were significantly greater for external oblique muscle in the hyperpronation group ($P < 0.01$, $P = 0.01$ respectively) (Fig. 2) (Table 2).

4. Discussion

Although altered muscular control in the lumbopelvic region is one of the main putative risk factors for the development of low back pain and dysfunction, the influence of hyperpronation– a highly prevalent malalignment of the foot – on lumbopelvic muscular function has not been investigated. The purpose of the present study was to compare muscle function variables in the lumbopelvic region in participants with hyperpronation vs. normal feet in the stance phase of gait. We hypothesized that functioning of the muscles that act as the main controllers of the lumbopelvic segment in the sagittal plane would differ between the two groups. Our results documented several between-group differences in peak force per se and peak force timings in all muscles studied here except the gluteus maximus. The patterns of muscle forces in our participants are consistent with previous studies which investigated trunk muscle functions during walking with musculoskeletal simulation methods [19,20].

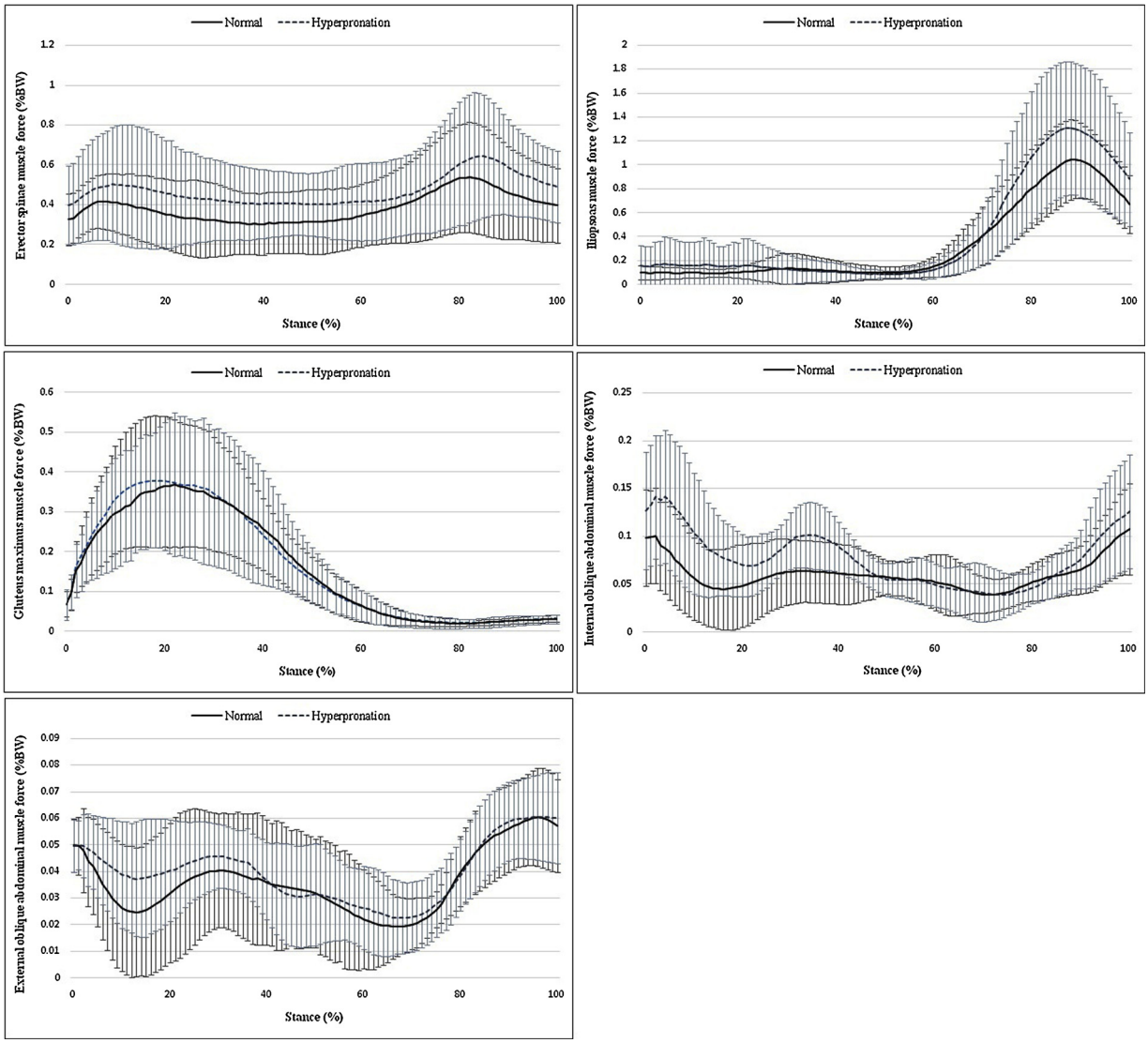

Fig. 2. Ensemble average curves and SD of normalized muscle forces of the selected muscles of the right body side (erector spinae, iliopsoas, gluteus maximus, internal oblique abdominal and external oblique abdominal) during the stance phase of gait in the normal (solid) and hyperpronation (dotted) groups.

In the lumbar region, the erector spinae muscles have two distinct bursts of activity during the gait cycle, both related to opposite heel strike. The first burst occurs between heel strike and 20% of the gait cycle, and the second occurs from 45% to 70% of the gait cycle. These bursts of activity serve mainly to counteract forward bending of the trunk relative to the hips at heel strikes. The greater erector spinae muscle force we observed in the hyperpronation group during the stance phase maybe due to increased anterior inclination [21] of their pelvic segment, which in turn may increase the external flexor moment applied on the pelvic segment. In this situation, greater activity of the erector spinae muscles would be needed to modify postural changes in the pelvic segment.

In participants with hyperpronated feet, the erector spinae muscle reached its peak values with a significant delay compared to the control group. Significant delays in electromyographic activation of some trunk muscles were previously reported in patients with low back pain during voluntary movement of their lower extremity [22]. Further to this, Aruin and Latash [23] noted that in patients with a history of low back pain, delayed trunk muscle response to perturbations (rapid movement of the upper extremity) was a sign of altered motor control. Together with the above findings, our results support alterations in motor control functions as a possible factor in the development of lumbopelvic pain and dysfunction.

The iliopsoas was also evaluated in the present study. This muscle is concentrically activated from slightly before toe off until initial swing. The purpose of this activation is to produce lumbar stabilization, in addition to moving the pelvic segment in the sagittal plane. We found that during late stance, peak iliopsoas muscle forces were greater in the hyperpronation group than the control group. During the stance phase of walking, lack of full knee extension in abnormal foot pronation [24] offers a mechanical explanation for this finding. People with hyperpronation may increase anterior tilt in their pelvic segment as a result of iliopsoas muscle-induced shift in the line of gravity toward the anterior of the knee joint, which would passively result in further knee extension. However, in view of the suggested role of the iliopsoas in spinal stabilization, increased activity of this muscle may be a response to possible decreased stability in the lumbar region.

Our results showed no significant between-group differences in normalized gluteus maximus muscle forces or timing of peak variables in the stance phase of gait. Previous studies suggested possible decreased hip extension [25] associated to decreased dorsiflexion of the first metatarsophalangeal joint as a consequence of foot hyperpronation [26]. Therefore, increased activity of the hip extensor muscles would be expected. However, we found no difference between groups in gluteus

Table 2
Mean(SD) peak normalized erector spinae, iliopsoas, gluteus maximus, internal oblique abdominal and external oblique abdominal muscle forces and timings during the stance phase of gait in the normal and hyperpronation groups.

	Subphase	Variable	Normal group Mean (SD)	Hyperpronation group Mean (SD)	P value
Erector spinae	**DS1**	Peak force (%BW)	0.35(0.17)	0.57(0.28)	0.02*
		Timing (% stance)	8.06(1.9)	11.86(4.05)	0.003*
	SLS	Peak force (%BW)	0.33(0.15)	0.46(0.13)	0.02*
		Timing (% stance)	54.86(9.52)	53(12.04)	0.64
	DS2	Peak force (%BW)	0.49(0.24)	0.69(0.27)	0.03*
		Timing (% stance)	84.26(3.97)	88.20(4.14)	0.01*
Iliopsoas	**DS1**	Peak force (%BW)	0.10(0.04)	0.17(0.20)	0.2
		Timing (% stance)	15.26(4.74)	12.60(5.66)	0.17
	SLS	Peak force (%BW)	0.79(0.25)	1.07(0.47)	0.07
		Timing (% stance)	79.93(0.25)	79.86(0.51)	0.65
	DS2	Peak force (%BW)	1.10(0.15)	1.43(0.43)	0.01*
		Timing (% stance)	89.93(3.34)	87.66(0.11)	0.86
Gluteus maximus	**DS1**	Peak force (%BW)	0.39(0.16)	0.42(0.08)	0.4
		Timing (% stance)	23.46(3.79)	21.2(4.09)	0.1
	SLS	Peak force (%BW)	0.36(0.15)	0.41(0.07)	0.3
		Timing (% stance)	31.30(2.39)	29.26(2.4)	0.07
	DS2	Peak force (%BW)	0.02(0.009)	0.03(0.006)	0.4
		Timing (% stance)	99.86(0.35)	99.8(0.56)	0.7
Internal oblique abdominal	**DS1**	Peak force (%BW)	0.10(0.05)	0.16(0.06)	0.01*
		Timing (% stance)	3.6(1.05)	4.8(1.56)	0.02*
	SLS	Peak force (%BW)	0.07(0.03)	0.15(0.06)	0.00*
		Timing (% stance)	36.66(13.86)	41.66(10.84)	0.28
	DS2	Peak force (%BW)	0.10(0.03)	0.13(0.04)	0.04*
		Timing (% stance)	98(3.27)	99.46(1.24)	0.12
External oblique abdominal	**DS1**	Peak force (%BW)	0.04(0.005)	0.05(0.01)	0.001*
		Timing (% stance)	2(1.01)	5.13(3.02)	0.01*
	SLS	Peak force (%BW)	0.03(0.01)	0.03(0.02)	0.6
		Timing (% stance)	40.86(8.88)	41.26(9.35)	0.9
	DS2	Peak force (%BW)	0.05(0.008)	0.06(0.01)	0.1
		Timing (% stance)	95.66(3.75)	95.2(3.38)	0.1

DS1: first double support; SLS: single leg support; DS2: second double support; SD: standard deviation.
* Significant difference in mean values, $P < 0.05$.

maximus muscle force. This could be explained by the findings in Lieberman et al. [27] who reported that this muscle is almost silent during level walking. It is thus not surprising that this muscle played no significant role in compensating for decreased hip extension. A possibility that should be considered is that compensatory changes occur in other hip extensor muscles, but this is speculative at this point, since we did not evaluate the activity of these muscles.

With respect to the abdominal muscles, the results of our study showed significantly greater peak normalized internal oblique abdominal force during the entire stance phase in the hyperpronation group. In the external oblique abdominal muscle, the magnitude of peak force was greatest during initial stance. In both abdominal muscles, the timing of peak force in the early stance phase was significantly delayed in the hyperpronation group compared to the normal group. It is believed that these muscles, as part of a larger system, play an important role in controlling gross spinal movements and thus providing sufficient spinal stability. However, evidence is lacking to clearly define the function and activity patterns of these muscles during walking. McGill [28] believed that abdominal muscles together with the thoracolumbar fascia form a “hoop” around the trunk. This author suggested that the role of this hoop is mainly to provide lumbopelvic stability. Furthermore, Cholewicki et al. [29] demonstrated that greater activation of the hoop muscles compensates for the loss of stiffness in the spinal column. On the other hand, increased abdominal muscle activity may be a mechanical response to counteract the increased anterior pelvic tilt in persons with hyperpronated feet.

Considering the increased level of activity of the abdominal and erector spinae muscles, the results of the present study support the existence of “guarding” in the lumbopelvic region in persons with abnormal foot pronation. The guarding hypothesis was previously proposed in patients with low back pain. Simultaneous increases in the activity level of the anterior and posterior trunk muscles were reported in people with low back pain compared to healthy individuals [30]. Some authors proposed that guarding of the superficial trunk muscles may overload these muscles [31,32], and consequently lead to excessive loading of the spinal structures.

Taken together, our findings showed that foot hyperpronation altered lumbopelvic muscle function during the stance phase of gait. Deficits in the lumbopelvic musculature in terms of neuromuscular activation and force distribution imbalance may predispose the structure to injury [8], which may in turn lead to the development of low back pain. In this connection, it was reported that increased iliopsoas muscle activity may put the intervertebral disc at risk of injury due to cyclic overloading [33].

This study has limitations. Although the model gait2392 is one of the most widely used models in the literature, this generic model features a rigid single spine segment and the muscle architecture is derived from cadaver studies or from anthropometrics of a small number of healthy participants and may not represent the exact muscle-tendon architecture of every individual participant either. Moreover, there still is not a consensus about the muscle force changes that might be considered as risk factor for development of lumbopelvic disorders in the literature. However, our findings add to clinical reasoning information related to claim linking foot malalignment to symptoms at low back region. In this light, the evaluation of foot posture should be considered an essential part of rehabilitation in patients who suffer from lumbopelvic pain.

We suggest that more conclusive results could be obtained in studies designed to investigate the influence of foot morphology on muscle function in the lumbopelvic region using more specific models that may be developed in the future. We recommend further research designed to examine the treatment options for foot posture aimed at regaining normal lumbopelvic muscular function in persons with foot hyperpronation.

5. Conclusion

This study is the first to identify the effect of abnormal foot alignment on lumbopelvic muscle function during walking. The importance of changes in muscle activation patterns becomes salient when we consider that walking is a routine task that is repeated thousands of times a day. In this situation any biomechanical abnormality may lead to the development of pathology.

We provide evidence of significantly greater peak muscle forces in the erector spinae, iliopsoas, internal oblique abdominal and external oblique abdominal muscles in women with hyperpronation than in women with normal foot alignment. Moreover, in our hyperpronation group we observed significant delays in peak force values in the abdominal muscles during early stance, and in the erector spinae muscles during both early and late stance.

The results of this study suggest that in persons with abnormal pronation, muscular control of movement in sagittal plane is disturbed. From a clinical perspective, altered muscle activation and function is one of the risk factors predisposing individuals to dysfunction and injuries. Therefore, assessment of foot posture should be considered a part of rehabilitation programs for lumbopelvic dysfunction. In addition, persons with abnormal pronation may benefit from muscle coordination exercises to prevent lumbopelvic symptoms.

Declaration of Competing Interest

The authors declare no conflict of interest.

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