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Angin, S, Mickle, KJ and Nester, CJ
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Contributions of foot muscles and plantar fascia morphology to foot posture

Salih Angin
School of Physical Therapy and Rehabilitation, Dokuz Eylul University, Inciralti, 35340 Izmir, Turkey

Karen J. Mickle
Institute of Sport, Exercise and Active Living (ISEAL), Victoria University, Melbourne, VIC 8001, Australia

Christopher J. Nester
School of Health Sciences, University of Salford, Salford, Manchester M6 6PU, United Kingdom

Corresponding author: Salih Angin
Dokuz Eylul University,
School of Physical Therapy and Rehabilitation
Inciralti, 35340 Izmir, Turkey

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Abstract

Background: The plantar foot muscles and plantar fascia differ between different foot postures. However, how each individual plantar structure contribute to foot posture has not been explored. The purpose of this study was to investigate the associations between static foot posture and morphology of plantar foot muscles and plantar fascia and thus the contributions of these structures to static foot posture.

Methods: A total of 111 participants were recruited, 43 were classified as having pes planus and 68 as having normal foot posture using Foot Posture Index assessment tool. Images from the flexor digitorum longus (FDL), flexor hallucis longus (FHL), peroneus longus and brevis (PER), flexor hallucis brevis (FHB), flexor digitorum brevis (FDB) and abductor hallucis (AbH) muscles, and the calcaneal (PF1), middle (PF2) and metatarsal (PF3) regions of the plantar fascia were obtained using a Venue 40 ultrasound system with a 5–13 MHz transducer.

Results: In order of decreasing contribution, PF3>FHB>FHL>PER>FDB were all associated with FPI and able to explain 69% of the change in FPI scores. PF3 was the highest contributor explaining 52% of increases in FPI score. Decreased thickness was associated with increased FPI score. Smaller cross sectional area (CSA) in FHB and PER muscles explained 20% and 8% of increase in FPI score. Larger CSA of FDB and FHL muscles explained 4% and 14% increase in FPI score respectively.

Conclusion: The medial plantar structures and the plantar fascia appear to be the major contributors to static foot posture. Elucidating the individual contribution of multiple muscles of the foot could provide insight about their role in the foot posture.

Keywords: Foot muscles, Plantar fascia, Morphology, Ultrasound, Pes planus, Foot Posture Index
Introduction

Forces produced by intrinsic and extrinsic foot muscles, and transmitted by the plantar fascia, act across the numerous rear, mid and forefoot joints and are thus assumed to contribute to foot posture. Differences in foot posture are associated with altered plantar pressure patterns [1] with likely alteration of external joint moments as well as kinaesthesia inputs [2]. Motor responses to the altered sensory inputs could thereafter affect muscle function and the foot mechanics associated with that foot posture [2, 3]. Indeed, muscle strength and function have been shown to be related to foot posture [4] and different foot kinematics exhibited between cavus, planus and normal foot postures [5].

Muscle morphology (cross sectional area (CSA) and thickness) can be indicative of muscle performance, including strength [6], and has been used to investigate relationships between foot muscles and foot posture. Murley et al. [7] reported an association between flat-arched feet and thicker peroneus longus muscle and tibialis anterior tendon, and thinner Achilles tendon.

Furthermore, increased navicular drop, indicative of a more pronated foot posture, has been shown to occur after impairing intrinsic muscles using anaesthesia and a fatigue protocol. Consequently, electrically stimulated plantar intrinsic muscles have shown to produce sufficient forces to reduce longitudinal arch deformation under load [8]. This suggests variation in foot posture may be due to variation in muscle function [9].

Flexor hallucis longus (FHL) and flexor digitorum longus (FDL) are known contributors to the shape of the medial longitudinal arch and act by resisting midfoot dorsiflexion associated with foot pronation [10]. We have previously shown that the CSA of these two extrinsic muscles is greater in pes planus than normal foot posture [11], although the intrinsic supinator muscles were smaller in pes planus. These extrinsic and intrinsic muscles might be expected to change morphology in a similar way since they create the same moments around many foot joints. According to Hintermann et al. [10], and using the tibialis posterior moment arm as reference (1.00), average invertor moment arms were 0.75 for flexor digitorum longus, and 0.62 for flexor hallucis longus. Perhaps in pes planus the different foot posture reduces the FHL and FDL moment arm at the rearfoot [12] so that they need to generate greater forces to contribute the required moments to resist external pronation moments and facilitate normal sagittal plane ankle function. This may result in hypertrophy as seen in posterior tibial tendon dysfunction induced pes planus [13], and reduced demand for forces from intrinsic supinators, hence the CSA of extrinsic muscles would be greater and that of intrinsic muscles reduced. Murley et al. [14] reported decreased peroneal muscle activity in flatfeet which would be complementary to greater invertor activity associated with their greater CSA in pes planus.
The plantar fascia, particularly the forefoot portion, was also reported to be thinner in pes planus foot types [11], perhaps via a similar mechanism. However, its function is also coupled to transmission of Achilles tendon forces to the forefoot during walking and thus is not solely concerned with foot posture [15] and also toe flexion/extension.

There is emerging evidence that intrinsic and extrinsic muscles and plantar fascia differ between different foot postures. The multiple muscles of the foot differ from each other in terms of size (longus/brevis) but also location (intrinsic/extrinsic, medial/lateral) and are therefore likely to contribute to foot posture in different ways. However, how individual plantar structures contribute to foot posture has not been explored. Understanding major and minor contributors could be relevant in the design and evaluation of interventions for foot muscle strength and clinical pathologies associated with specific foot types. The purpose of this study, therefore, was to investigate any associations between foot posture and measures of intrinsic and extrinsic foot muscles and plantar fascia and thus the contributions of these structures to foot posture.

Methods

Participants
A total of 111 subjects (61 males, 50 females) aged between 18 and 47 years were recruited from university communities. They were free of lower extremity injuries in the past 12 months and had no history of lower extremity surgery and visual or vestibular disorders. The study was approved by the institutional ethical committee. Each participant provided informed consent before participating in the study.

Foot posture assessment
The Foot Posture Index (FPI) was employed for quantitative assessment of foot posture by an experienced physiotherapist (worked in musculoskeletal care for 8 years). Both feet of each participant were assessed for the six FPI criteria. The six individual scores were then combined to give a composite score between -12 and +12. A composite score between 0 and 5 indicated a normal foot posture, ≥ 6 a pes planus posture.

Measurement of the muscle cross-sectional area and plantar fascia thickness
Ultrasound can be used to reliably measure foot muscle and plantar fascia features [16, 17] and was the method chosen for this study. Muscle CSA and PF thickness were scanned by the Chief Investigator (SA), who has had extensive training on foot and ankle musculoskeletal ultrasound scanning. The scanning took place one week after the FPI assessment and the assessor was blind to the FPI Score. A Venue 40 musculoskeletal ultrasound system (GE Healthcare, UK) with a 5–13 MHz wideband linear array probe with 12.7 mm to 47.1 mm surface area was used to image CSA of
each structure and thickness of the plantar fascia in the right foot of each participant. Details of probe position and orientation for each structure, and all other aspects of the protocol are explained elsewhere [11, 16].

Each participant lay in the prone position for scanning PF, FHB and FDB muscles, and in the supine position for scanning the AbH, FDL, FHL, and PER muscles. The medial part of the PF was scanned longitudinally at three different regions: calcaneal part (PF1); middle part (PF2); and metatarsal part (PF3) attached to the second MTP joint based on where the highest pressure was previously found during push-off [18]. All scans were performed with the ankle joint in the neutral position. The CSA and thickness measures were taken by the ultrasound user (SA), who remained blind to the FPI scores, using Image J software (National Institute for Health, Bethesda, USA) and as described in the previous studies. The mean value was derived from three images.

Data analysis

Rasch transformation of the raw FPI values described by Keenan et al. [27] was used for conversion of the raw FPI categorical data to continuous data for parametric statistical tests. Data from the right foot was analyzed in order to satisfy the independence assumption of statistical analysis [19]. Variables with skewed distributions were log transformed.

Univariate Pearson correlation coefficients were calculated for transformed FPI and the ultrasound variables. The ultrasound variables that significantly correlated with transformed FPI were input as independent variables into a multiple regression analysis to find major contributors to the FPI. The linear regression analysis was run following the backward stepwise elimination procedure based on the probability of F determined as a stepping method criteria. A significance level of P<0.05 was required for entry into the model, and P>0.06 was the criterion for removal. The maximum value of variance inflation factor (VIF) was determined as 5.0 for multicollinearity. All statistical analysis was performed using IBM SPSS software version 20.0 (IBM Corporation, Armonk, NY, USA).

Results

Forty-three individuals (38%) had pes planus (18 females) with mean FPI of 7.86 ± 1.58 (range 6–11) and Rasch transformed means of 5.55 ± 1.21 (range 3.81–7.77). The remaining 68 had normal feet (32 females) with mean FPI of 1.41 ±1.44 (range 0–5) and Rasch transformed mean FPI of 0.78 ± 0.97 (range -0.21 – 3.81). Demographics of the participants are shown in Table 1.

As a result of the correlation analysis (see Table 2), PF1 was identified as the only variable that was not significantly correlated to FPI and therefore excluded from the regression analysis. All other
variables were included in the multiple regression analysis. Higher transformed FPI scores (i.e. a more pes planus foot type) were correlated with smaller CSA of the AbH (r = -0.42, p < 0.0001), FHB (r = -0.44, p < 0.0001) and PER (r = -0.28, p = 0.003) muscles. Higher transformed FPI scores were also correlated with thinner PF2 (r = -0.54, p <0.0001) and PF3 (r = -0.72, p <0.0001). Higher transformed FPI scores were also correlated with larger CSA of FDB (r = 0.19, p = 0.045), FDL (r = 0.35, p <0.0001) and FHL (r = 0.37, p <0.0001). Distribution of PF thickness and cross-sectional area of the muscles are represented in Table 3.

A total of eight variables that significantly correlated to FPI were narrowed to five as AbH, FDL and PF2 were excluded from the final model based on the stepping method criteria [28]. The resulting five-variable model (F = 47.48; p <0.0001) had an r =0.83, \( r^2 = 0.69 \), and variance inflation factor (VIF) <3.0 (Table 4). The five variables in the final model accounted for 69% of variance in the FPI score. Of the individual independent variables, decreased thickness of PF3 was the highest contributor explaining 52% (\( \beta = -0.51 \)) of increases in FPI. Smaller CSA in FHB and PER muscles explained 20% (\( \beta = -0.23 \)) and 8% (\( \beta = -0.16 \)) of increases in FPI respectively. Larger CSA of FDB and FHL muscles explained 4% (\( \beta = 0.33 \)) and 14% (\( \beta = 0.32 \)) of increases in FPI respectively.

Measured mean FPI was 2.63 ± 2.56 and the predicted FPI mean based on the CSA of the muscles and PF3 thickness in the model was 2.63 ± 2.13 (Figure 1).

Discussion
We have found, in order of decreasing contribution, PF3>FHB>FHL>PER>FDB were all associated with FPI (r = 0.83), and were able to explain 69% of the change in FPI scores (Figure 1). Among these variables, plantar fascia was the main contributor to change in FPI scores, contributing more than the other four factors combined. The role of these five variables in foot posture agrees with prior studies that have investigated the function of these structures [11, 20] but their relative contributions have not been described before.

This cross-sectional analysis describes static foot posture and relates it to muscle features that are assumed to infer the dynamic function of the muscle e.g. larger CSA equates to greater muscle
strength and therefore greater forces during gait [21]. The muscle forces in standing and thus during our foot posture measures would be different than those during gait. We cannot ascertain whether a change in any of the structures evaluated would lead to a change in foot posture and therefore cannot infer a cause-and-effect relationship between the foot structures and foot posture. However, within the context of this limitation, we have identified apparent different contributions of the selected muscles and plantar fascia to foot posture.

Plantar fascia thickness at the metatarsal region (PF3) was the greatest contributor to change in FPI (52%). That fascia was found to contribute more than muscles could perhaps relate to the fact we assessed posture statically, during which perhaps passive structures rather than muscle forces are relied upon. However, if this were true then PF1 and PF2 might have also been significant contributors and they were not. The plantar fascia has been reported to contribute as much as 80% of the force resisting lowering of the medial arch [20]. In their cadaveric study, Huang et al. [22] found that the plantar fascia was highest contributor (55.6%) to arch stability among the other static structures, and their simulated model showed that there was little muscle activity during standing posture.

That both extrinsic (FHL, PER) and intrinsic (FHB, FDB) muscles were contributors in the final regression model perhaps reflects their shared function in determining foot posture. However, there was no pattern in contribution in terms of muscle size and thus assumed muscle forces and foot posture. FHB was second greatest contributor yet is smaller in muscle volume and tendon thickness (a surrogate measure of forces born) than FHL and PER. Whilst the shortening capacity of FHB is certainly smaller than that of extrinsic muscles [23], Hashimoto et al. [4] found increased medial longitudinal arch (MLA) height after use of exercises strengthening intrinsic flexor muscles including FHB. Decreased FPI scores and increased MLA height with exercises targeting intrinsic muscles have also been reported [24]. However, muscles associated with the hallux and medial side of the foot (FHB and FHL) were ranked 2nd and 3rd contributors, and the main contributor, PF3, was measured on the medial side of the foot too. FHL and FHB together contributed 34% to the FPI scores whereas PER and FDB contributed only 12%. FHB contributed 14% whereas the more lateral FDB contributed 4% to the FPI scores. The contribution from medial structures might therefore be more important to foot posture. Measures of lateral plantar fascia and flexor digiti minimi muscle would be required to clarify relative contributions of other lateral/medial structures.

Thinner fascia could mean higher loads if those loads lengthened the fascia. However, it could be speculated that the PF could not stretch uniformly throughout its length. Morphologically, PF3 is thinner and could be more sensitive to tensile forces compared to other regions (PF1 and PF2). As the highest tension load was found at the PF3 region during the push off [25] this may indicate elongation...
[26, 27] and further decrease thickness of the plantar fascia at the metatarsal region (PF3). However, given the weakness of some correlations it is not clear why there may be increased CSA in some muscles with apparently contradictory smaller CSA in muscles with similar function. This could be related to the so called windlass mechanism. Hicks concluded that the toes are forced into an extended position in toe-standing and walking by the action of body weight, and the arch is caused to rise by the windlass mechanism (tensile forces in the plantar fascia) without direct action of any muscle [28]. Other studies have also revealed that whilst plantar fascia provides passive stiffness to the longitudinal arch, plantar intrinsic and extrinsic muscles continuously regulate this stiffness [26, 29].

The windlass mechanism also works in a reverse direction when the foot is loaded. As the MLA flattens in pes planus foot, tensional forces increases in the plantar fascia [29], the reverse windlass mechanism therefore pulls the metatarsophalangeal (MTP) joints into flexion [29, 30]. This action is normally shared by plantar fascia and plantar intrinsic muscles. This could also mean that a reverse windlass mechanism lessens the intrinsic muscle activity required for MTP joint flexion.

This work poses several new questions. Does increased load in the plantar fascia lead to thinner fascia or hypertrophy? Thicker fascia in cases of heel pain may suggest the latter, but this is equally likely to be the effects of inflammation as much as tissue hypertrophy. Also, the rationale for using extrinsic rather than intrinsic muscles is not clear, nor is the use of lesser toe rather than hallux muscles. How and why these mechanisms are used to control foot behaviour remains unclear and points to the need for research that explains how the body uses the duplication in foot and ankle musculature and plantar fascia to vary foot stiffness and how this leads to differences in static foot posture. A mechanism clearly exists since we were able to explain 69% of variation in the FPI scores by a combination of 5 measures of muscle and fascia structure. However, PF data explained more than 50% of the variance in FPI scores and so is clearly the starting point for any explanation. The relationship between plantar fascia morphology and its dynamic behaviour requires further clarification.

There are several limitations that need to be acknowledged. The age and BMI differences between our groups were not considered in the analysis values were similar in both groups. However, these factors may influence the muscle morphology. The gender balance in each group was not equal, and may also affect muscle size. Whilst ultrasound has a good to excellent inter-rater reliability it is user dependent. We did not directly test the intra-rater reliability of the operator, although the values for all structures measured are in line with prior literature. Finally, cavus foot types have not been included in the study and thus one end of the foot posture spectrum is absent. It is also acknowledged that only FPI values for normal and planus feet have been included in the multiple regression analysis compared to total range from planus foot (-12) to cavus foot (+12). Further research is required to confirm our findings over the full range of foot postures.
In conclusion, we have demonstrated the contribution of the plantar muscles and fascia structure to FPI scores. The medial plantar structures appear to be the major contributors to foot posture with the PF alone contributing 52% of changes in FPI. Elucidating the individual contribution of multiple muscles that differ from each other in terms of size and location, and plantar fascia structure, provide insight about their role in foot posture. Further studies are warranted to explore the interactions between the individual structures and how they each and collectively contribute to differences in dynamic foot function and static foot posture.

References


Table 1: Demographic features of the groups

<table>
<thead>
<tr>
<th></th>
<th>Pes Planus</th>
<th>Normal Feet</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Mean ±SD</td>
<td>Mean ±SD</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>23.74 ±4.87</td>
<td>24.79 ±6.38</td>
<td>0.331</td>
</tr>
<tr>
<td>Body Weight</td>
<td>69.30 ±13.16</td>
<td>69.84 ±13.70</td>
<td>0.838</td>
</tr>
<tr>
<td>Body height</td>
<td>171.65 ±8.31</td>
<td>171.66 ±8.38</td>
<td>0.995</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>23.36 ±3.25</td>
<td>23.60 ±3.67</td>
<td>0.725</td>
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Table 2: Correlation coefficients between FPI and cross-sectional area of the muscles and plantar fascia thickness

<table>
<thead>
<tr>
<th>Variables</th>
<th>r</th>
<th>p</th>
</tr>
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<tr>
<td>AbH</td>
<td>-0.42</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>FDB</td>
<td>0.19</td>
<td>= 0.045</td>
</tr>
<tr>
<td>FDL</td>
<td>0.35</td>
<td>= 0.0002</td>
</tr>
<tr>
<td>FHB</td>
<td>-0.44</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>FHL</td>
<td>0.37</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>PER</td>
<td>-0.28</td>
<td>=0.003</td>
</tr>
<tr>
<td>PF1</td>
<td>-0.01</td>
<td>=0.925*</td>
</tr>
<tr>
<td>PF2</td>
<td>-0.54</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>PF3</td>
<td>-0.72</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*Not significant

AbH, Abductor hallucis; FDB, flexor digitorum brevis; FDL, Flexor digitorum longus; FHB, Flexor hallucis brevis; FHL, Flexor hallucis longus, PER, peroneus longus and brevis; PF1, plantar fascia (calcaneal part); PF2 plantar fascia (middle part); PF3, plantar fascia (metatarsal part)
Table 3: Distribution of the PF thickness and CSA of the muscles based on the FPI.

<table>
<thead>
<tr>
<th>FPI</th>
<th>N</th>
<th>AbH-CSA Mean±SD</th>
<th>FDB-CSA Mean±SD</th>
<th>FDL-CSA Mean±SD</th>
<th>FHB-CSA Mean±SD</th>
<th>FHL-CSA Mean±SD</th>
<th>PER-CSA Mean±SD</th>
<th>PF1-T Mean±SD</th>
<th>PF2-T Mean±SD</th>
<th>PF3-T Mean±SD</th>
</tr>
</thead>
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<tr>
<td>0-5</td>
<td>68</td>
<td>2.71±0.36</td>
<td>2.06±0.55</td>
<td>2.43±0.62</td>
<td>3.20±0.47</td>
<td>2.84±0.67</td>
<td>3.68±0.82</td>
<td>0.33±0.05</td>
<td>0.19±0.03</td>
<td>0.13±0.01</td>
</tr>
<tr>
<td>6-12</td>
<td>43</td>
<td>2.28±0.43</td>
<td>2.19±0.48</td>
<td>2.75±0.60</td>
<td>2.69±0.44</td>
<td>3.31±0.69</td>
<td>3.21±0.66</td>
<td>0.33±0.05</td>
<td>0.16±0.02</td>
<td>0.10±0.02</td>
</tr>
<tr>
<td>Total</td>
<td>111</td>
<td>2.54±0.44</td>
<td>2.11±0.53</td>
<td>2.56±0.64</td>
<td>3.00±0.52</td>
<td>3.03±0.71</td>
<td>3.50±0.79</td>
<td>0.33±0.05</td>
<td>0.17±0.03</td>
<td>0.12±0.02</td>
</tr>
</tbody>
</table>

FPI: Foot Posture Index; Mean±SD: Mean (cm²) ± Standard Deviation; AbH: Abductor hallucis; FDB: Flexor digitorum brevis; FDL: Flexor digitorum longus; FHB: Flexor hallucis brevis; FHL: Flexor hallucis longus; PER: Peroneal muscles; PF (1,2,3): plantar fascia (calcaneal portion, middle portion, metatarsal portion); CSA: Cross-sectional area; T: Thickness
Table 4: Multiple regression between FPI and cross-sectional area of the muscles and plantar fascia thickness remained in the final model ($F = 47.48; r = 0.83, p < 0.0001, r^2 = 0.69$), and rank of contribution to FPI score.

<table>
<thead>
<tr>
<th>Dependent</th>
<th>Independents</th>
<th>β-coefficient</th>
<th>$r^2$</th>
<th>$p$</th>
<th>VIF</th>
<th>Rank</th>
</tr>
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<tbody>
<tr>
<td>FPI</td>
<td>FDB</td>
<td>0.33</td>
<td>0.04</td>
<td>=0.0006</td>
<td>1.20</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>FHB</td>
<td>-0.23</td>
<td>0.20</td>
<td>&lt;0.0001</td>
<td>2.05</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>FHL</td>
<td>0.32</td>
<td>0.14</td>
<td>&lt; 0.0001</td>
<td>2.28</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>PER</td>
<td>-0.16</td>
<td>0.08</td>
<td>= 0.001</td>
<td>1.52</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>PF3</td>
<td>-0.51</td>
<td>0.52</td>
<td>&lt; 0.0001</td>
<td>2.96</td>
<td>1</td>
</tr>
</tbody>
</table>

FPI, Foot Posture Index; FDB, flexor digitorum brevis; PER, peroneus longus and brevis; PF3, plantar fascia (metatarsal part); VIF, variance inflation factor.
Figure 1: Regression plot displaying the association ($F = 47.48$, $r = 0.83$; $r^2 = 0.69$) between observed FPI and predicted FPI using cross-sectional area of the muscles and plantar fascia thickness from the group of normal and pes planus feet.
- The medial plantar structures appear to be the major contributors to static foot posture.
- Plantar fascia is the main contributor among the plantar structures.
- Plantar muscles have less contribution on static foot posture.