Title

Foot stiffening during the push-off phase of human walking is linked to active muscle contraction, and not the windlass mechanism

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Media Summary

The human foot has evolved to support our ability to walk upright on two legs. The foot's function has historically been considered highly dependent on a passive link between our toes and the foot's arch. In this paper we provide evidence that these so-called passive mechanisms rely on supporting muscle contraction, and that foot muscles are required for applying force through our feet to the ground. Therefore, maintaining strong and healthy foot muscles may be key to our ability to walk as we do.

Abstract

The rigidity of the human foot is often described as a feature of our evolution for upright walking, and is bolstered by a thick plantar aponeurosis that connects the heel to the toes. Previous descriptions of human foot function consider stretch of the plantar aponeurosis via toe extension ('windlass mechanism') to stiffen the foot as it is levered against the ground for push-off during walking. In this study we applied controlled loading to human feet in vivo, and studied foot function during the push-off phase of walking, with the aim of carefully testing how the foot is tensioned during contact with the ground. Both experimental paradigms revealed that plantar aponeurosis strain via the 'windlass mechanism' could not explain the tensioning and stiffening of the foot that is observed with increased foot-ground contact forces and push-off effort. Instead, electromyographic recordings suggested that active contractions of ankle plantar flexors provide the source of tension in the plantar aponeurosis. Furthermore, plantar intrinsic foot muscles were also contributing to the developed tension along the plantar aspect of the foot. We conclude that active muscular contraction, not the passive windlass mechanism, is the foot's primary source of rigidity for push off against the ground during bipedal walking.

Keywords

Foot, bipedalism, plantar aponeurosis, windlass mechanism, intrinsic muscles, plantar flexors

Background

The evolution of bipedalism and an upright posture in humans is reflected in adaptations to the structure and function of our feet [1-4]. Pronounced arches, a robust calcaneus, short toes, and an adducted hallux distinguish human feet from those of other apes [1]. A more substantial plantar aponeurosis is also present in the human foot (Fig 1), which is considered important for bipedalism in two-ways. First, the plantar aponeurosis has been described as a tie between the heel and the toes that affords rigidity to the foot [5]. A rigid foot hypothetically benefits bipedal gait by providing a stiff lever for exerting push-off forces against the ground [6]. However, other work has emphasised that elastic stretch and recoil of the plantar aponeurosis during each footfall of gait helps the long arch function as an energy saving spring [7]. It is unclear how an elastic 'spring-like' plantar aponeurosis can simultaneously act as a rigid tie. Therefore, the mechanisms by which human foot mechanics are optimised for push-off during bipedal gait require further investigation.

The plantar aponeurosis arises from the calcaneus, traverses the plantar aspect of the foot, and wraps around the underside of the five metatarsal heads, before inserting on the proximal phalanges (Fig 1). Therefore, extension of the toes exerts a pull on the calcaneus via the plantar aponeurosis (Fig 1). Hicks [5] coined this a 'windlass mechanism' whereby toe extension winds the aponeurosis around the metatarsal heads. The resulting force applied to the

calcaneus and metatarsal heads draws the foot's longitudinal arch together, raising its midpoint. In gait, extension of the toes occurs as the heel rises and we push off against the ground to transition from one step to the next (Fig 1). This is when the windlass mechanism is thought functional, as it has been suggested to stiffen the foot for more effective leverage [6]. However, during this phase, the longitudinal arch is actually recoiling with declining force rather than behaving as a rigid lever, and recent work shows that raising the longitudinal arch by extending the toes actually decreases the arch's stiffness [8]. Also, simulations show that plantar aponeurosis tension in late stance does not increase with the greater push-off forces required for increasing walking speed, despite increased toe extension [9]. Furthermore, non-human apes can adequately push-off for bipedal walking despite exhibiting minimal arch rise [3, 10, 11]. Therefore it is unclear how the windlass mechanism alone which, in gait coincides with a rising arch and decreasing tension in the aponeurosis, could serve to stiffen the foot during push-off. Physical simulations on human cadaver feet correlate plantar aponeurosis tension with ankle plantar flexor muscle forces (Fig 1) that tend to flatten the long arch during gait and peak before push-off [12, 13]. It seems more feasible that tension in the plantar aponeurosis, and across the plantar aspect of the foot (including the metatarsal-phalangeal joints), is not simply dependent on extension of the toes [9]. Extension of the toes should not be assumed to result in increased aponeurosis tension or foot stiffening in late stance. This questions a key part of the traditional understanding of the link between human foot structure and function during gait.

Clearly it is challenging to interpret the influence of the windlass mechanism and function of the human plantar aponeurosis for gait. This is compounded by the need to consider muscular contributions to foot mechanics, which based on indirect evidence, have been proposed to contribute to plantar tension in late stance [9]. Several plantar intrinsic foot muscles course a parallel path to the plantar aponeurosis from the heel to the toes (Fig. 1). These muscles are active during the late stance phase of gait [14, 15], and appear to be important for modulating foot mechanics [16-19]. In a recent study, anaesthetically blocking plantar intrinsic muscle activation during gait lead to a decreased stiffening of the metatarsal-phalangeal joint (MTP_j – Fig. 1) and reduced push-off power [20]. Cadaveric experiments also support the notion that great-toe flexor muscles make important contributions to forces across the MTP_j [21]. Therefore we must consider the influence of active muscle contraction from toe flexors when interpreting the importance of the windlass mechanism for the human specialisation for bipedal gait.

This study aimed to deconstruct the interplay between ankle plantar flexor activation, plantar intrinsic foot muscle activation, and the windlass mechanism in human feet. In doing so, we expected to show that the passive windlass mechanism cannot simply be applied based on MTP joint kinematics when interpreting dynamic foot function during gait. In a first experiment, we manually extended the toes of individuals performing seated isometric plantar flexor contractions of varied intensity. We hypothesised that with greater plantar flexor muscle activation, the foot's long arch would be loaded more, stretching the plantar aponeurosis to a greater degree, and resisting the engagement of the windlass mechanism. We also hypothesised that plantar intrinsic muscle activation would increase synergistically with activation of the ankle plantar flexors, and this would stiffen the MTP_j to further resist the windlass mechanism and limit plantar aponeurosis strain. To extend the findings to walking mechanics, in a second experiment participants walked with normal and voluntarily increased push-off against the ground. We hypothesised that with greater push-off, greater plantar intrinsic foot muscle activation would explain stiffening of the MTP_j, not the windlass mechanism.

Methods

Experiment 1 - Ten individuals (sex: 9M and 1F; mean ± sd age = 27 ± 7; body mass = 76 ± 10 kg) free from leg or foot injuries, provided written informed consent to participate in this study that was approved by the institutional human research ethics committee of The University of Queensland. Sample size was determined from a large effect size (Cohen's f = 0.5) observed in a prior study of muscular responses to loading (experiment 1 in [20]) and a desired power of 0.95. Participants sat with their knee flexed, and the ball of their foot placed on a platform atop a force plate (Figure 1B). As per previous experiments [19, 20], a linear actuator (PS10-70x400U-BL-QJ, NTI AG, Linmot, Switzerland) mounted on a shop press was used to apply controlled loads to the distal thigh of participants. Loads of 0.5 (low), 1.0 (mid), and 1.5 (high) body weights (BW) were applied, each repeated three times. As the load was applied, participants were instructed to activate their plantar flexor muscles sufficiently to prevent dorsiflexion of the ankle. Thus, the purpose of the increasing loading conditions was to elicit increasing levels of plantar flexor muscle activation and Achilles tendon force. Once the target load on the thigh was reached, a hinged plate under the

toes was rotated to extend the toes by means of a cord that was tensioned via a winch until a target force of 250 N was measured in a load cell (Tedea Huntleigh 614, Vishay Precision Group, USA) in series with the cord (Figure 1B). The winch was then released allowing the toes to flex back to neutral, and the load on the thigh released.

A force plate (OR6-7-1000, AMTI, USA) measured applied forces that were sampled at 4000 Hz by a 14-bit analogue-digital converter and logged by QTM software (Qualysis, Sweden) synchronously with motion capture and electromyography (EMG) data. Six motion capture cameras (Oqus, Qualysis, Sweden) captured 3D positions of markers placed on the foot and shank in accordance with the IOR model described by Leardini et al. [22]. The foot model was implemented in Visual 3D software (C-motion Inc., USA), and used to construct shank, calcaneus, mid-foot, metatarsal, and great toe segments. Joint angles were defined as distal segment orientations in the proximal segment's coordinate system and were: ankle – calcaneus relative to shank; cal-met – metatarsal relative to calcaneus (representing longitudinal arch deformation); and metatarsal-phalangeal joint (MTP_j) – great toe relative to metatarsal. Ankle joint plantar flexion moment was calculated using ground reaction forces and a rigid foot segment via inverse dynamics analysis, but only for the period prior to tensioning of the cord. During toe rotation, a quasi-static calculation of MTP_j moment was performed. The component of cord force perpendicular to the great toe's long axis was multiplied by its lever arm to the proximal end of the great toe. MTP_j quasi-stiffness was calculated as the slope of the MTP_j moment vs angle relationship. The length of the plantar aponeurosis (PA) was estimated via a geometric model (similar to one previously described for plantar intrinsic muscle paths [15, 18]). The length of the PA was estimated as the distance between its attachments on the calcaneus and first phalanx, wrapped about a point immediately inferior to the MTP_j. This was intended to be a largely conceptual model of the PA, capable of capturing important changes in length for comparison across experimental conditions.

Experiment 2 – Nine participants (sex: 5M and 4F; mean ± sd age = 27 ± 4; body mass =73 ± 15 kg) provided written informed consent to participate in this study that was approved by the departmental ethical review committee at the University of Exeter. Sample size was determined from a large effect size (Cohen's f = 0.53) observed in a prior study of foot kinematic changes with increasing running speed [15] and a desired power of 0.95. Each participant completed walking trials in which they either walked across the laboratory (including one step on a force plate) at their self-selected preferred walking pace (control - CON), or approached the force plates at their preferred speed, but then produced a more forceful push-off with the right leg during the measured step (PUSH). Participants were instructed to maximise the strength of their push-off, without initiating running gait. A more forceful push-off was confirmed by visualisation of vertical and anterior ground reaction forces. This was not intended to simulate a specific form of natural gait, but was effectively a step in which the participant increased their walking speed considerably. Motion capture data were recorded for the same marker placements as Experiment 1 using a four-scanner Codamotion system (Charnwood Dynamics, UK). The same kinematic modelling process was followed as for Experiment 1 using Visual 3D software (C-motion, USA). Ground reaction force data were recorded synchronously using an in-ground force plate (BP400600HF, AMTI, USA) and sampled at 4000 Hz with a 16-bit analogue-to-digital convertor (NI PXI6224, National Instruments Ltd., USA). As per our prior work [20], the net moment of force generated about the MTPj was calculated quasi-statically once the ground reaction force vector passed anterior to the MTPj.

Electromyography measurements (both experiments) – Ankle plantar flexor muscle activation was assessed by surface EMG recordings, made using a bipolar electrode configuration (Ag–AgCl electrodes, 24 mm diameter, Tyco Healthcare Group) over the belly of the soleus (SOL) muscle. Fine wire EMG recordings were made from two major plantar intrinsic foot muscles using bipolar fine-wire electrode pairs, with a detection length of 2 mm (0.051 mm stainless steel, Teflon coated, Chalgren, USA). Wires were inserted under sterile conditions using a delivery needle (0.5 x 50 mm) and ultrasound guidance, into flexor digitorum brevis (FDB) and abductor hallucis (AH), as per our prior work [15, 16]. The fine-wires and surface electrodes were connected via preamplifiers operating at a gain of 20 (MA-411, Motion Lab Systems, USA) to a wired EMG system (MA300, MotionLab Systems, USA) with a low pass analogue filter set to 2000 Hz. EMG data were sampled at 4000 Hz by the respective analogue-to-digital convertors described above for each experiment. EMG data processing was performed in Matlab (The Mathworks, USA) and included removal of any DC offset, followed by band-pass digital filtering (Butterworth filter:20-400 Hz for surface EMG, 40-1000 Hz for intramuscular), before being enveloped using a rolling root-mean-square (RMS) calculation over windows of 100 ms. For Experiment 1, the RMS signal for each muscle was then normalised to the maximum RMS value during the 1.5 BW load condition. For Experiment 2, normalisation was performed to the maximum from the PUSH condition. The normalised EMG signals were each integrated with respect to time over the period from the

onset of actuator loading to the time of maximum force applied to the toes (Experiment 1), or over the stance phase of walking (Experiment 2), to yield an integrated EMG (iEMG) value.

Statistics - Linear mixed models, and the maximum likelihood ratio test (MLRT) were used to test the hypotheses for Experiment 1. To test if increasing plantar flexor contraction level affected the compression of the MLA after initial loading, raising of the MLA during toe extension, or plantar aponeurosis stretch, a model was fit for each variable using the Matlab (The Mathworks, USA) function *fitlme*. The model included fixed effects of intercept and soleus iEMG, and random effects of participant on intercept and slope for iEMG of soleus (to account for between-participant variability). To test for significance, the fixed effect of soleus iEMG was removed from a second model, and the MLRT was run on the two models using the Matlab (The Mathworks, USA) *compare* function. Because the plantar intrinsic muscles and the PA are the two main structures able to resist toe extension, to test what determined the quasi-stiffness of the MTP_j, another linear mixed model was fit with MTPj quasi-stiffness as the outcome variable. The model included fixed effects for plantar intrinsic muscle iEMG and final PA length, and random effects of participant on both (intercept and slope). The MLRT was run comparing this model and models where each of fixed effects had been removed. In experiment 2, the effect of condition (control vs. PUSH) on outcome measures of iEMG for each muscle, and peak MTPj moment were tested with paired t-tests. An alpha level of 0.05 was set for statistical significance for all tests.

Results

Experiment 1. Participants responded to initial loading of the leg with a burst of EMG activity from SO, compressing the MLA (Fig. 2A, C). There was a significant effect of increasing SO activation on increasing MLA compression (MLRT p=0.01), and expressed by loading condition the MLA angle increased (MLA was more compressed) at higher actuator loads (mean ± SD, low = 182 ± 8° mid = 183 ± 8° high = 184 ± 9°). Furthermore, the rise of the MLA due to toe rotation was less at higher actuator loads, reaching mean ± SD final angles of: low = 176 ± 9° mid = 181 ± 9° high = 183 ± 8°. The MLRT revealed a significant (p=0.03) effect of SO iEMG for restricting MLA rise upon toe rotation.

As a force was applied to rotate the toes, both AH and FDB experienced large bursts of EMG activity (Fig.2 B, C) and iEMG for both muscles was greater at higher loads (Fig. 3). The iEMG for AH and FDB significantly increased the quasi-stiffness of the MTPj (Fig. 4B, MLRT p = 0.04), which lead to reduced extension of the MTPj at higher actuator loads (Fig 4A). Plantar aponeurosis final length was unaffected by SO iEMG and did not change across actuator loading condition (Fig 5B).

Experiment 2. The peak anterior GRF values were significantly higher for PUSH than CON (179 ± 79 N vs. 119 ± 29 N, t-test p = 0.01), confirming a more forceful push-off. FDB and AH iEMG increased significantly with increased push-off effort (FDB: CON = 0.062 ± 0.05, PUSH = 0.10 ± 0.05, t-test p = 0.036; AH: CON = 0.08 ± 0.02, PUSH = 0.15 ± 0.03, t-test p <0.001; Fig. 6B). This occurred concurrently with greater peak MTPj moments (CON = 4.3 ± 2.3 N·m, PUSH= 8.9 ± 4.4 N·m, t-test p=0.004). MTPj angles and plantar aponeurosis lengths followed similar patterns of change over the latter half of stance in CON and PUSH (Fig. 6D).

Discussion

Providing tension across the long arch of the foot via the windlass mechanism has been considered an integral component of the human foot's role in bipedal walking and running mechanics. However, this interpretation is based on a passive-inelastic description of foot mechanics. Here we have shown that implementing the windlass mechanism in-vivo depends on active control of ankle and intrinsic foot muscles. As such, assumptions about tension across the plantar aspect of the foot, or rigidity of the foot during walking and running, should not be based only on the kinematics of the windlass mechanism. In fact, we demonstrate that greater extension of the toes only occurs when foot and ankle muscles are more relaxed, and tension across the plantar aspect of the foot is lower. We therefore suggest that extension of the MTPj in late stance should not be considered to increase tension across the long arch, but serve as an indication that active loading of the PA by plantar flexors is being released. Furthermore, plantar intrinsic foot muscles can tune MTPj moments to adjust MTPj mechanics, and limit strain of the plantar aponeurosis.

In Experiment 1 the participants matched increased loading from the actuator with greater soleus muscle activation, leveraging the forefoot against the supporting surface, and resulting in greater compression of the MLA prior to the

MTPj being extended. We consider this to mimic the level of loading during a more forceful push against the ground during the mid-to-late stance phase of gait. During this phase of gait, the foot begins to rotate over the metatarsal heads, extending the MTPj and engaging the windlass mechanism (Fig. 6A), which we simulated by forcibly extending the MTPj. It is sometimes considered that MTPj extension tensions the plantar aponeurosis and increases the rigidity of the foot as a propulsive lever (e.g. [23]). However in Experiment 1, toe extension was limited in higher loading conditions where soleus activity was higher, suggesting that greater toe extension is actually associated with a decline in push-off force, and reduced loading of the MLA. The same MTPj extension torque was applied in all conditions, so it is unsurprising that the final plantar aponeurosis length was unchanged between conditions (Figure 5B). However, with greater soleus activation (higher loading) this plantar aponeurosis strain was achieved with less MTPj extension and a more compressed MLA than at lower loads. This indicates that toe extension occurs only as ankle plantar flexor contraction is released, and tension across the plantar aspect of the foot becomes less. This observation agrees with the correlation between Achilles tendon force and plantar aponeurosis tension found by Erdemir et al. [12]. It also highlights that greater toe extension does not equate to the foot is becoming 'stiffer' or more rigid, in concordance with Welte et al.'s work [8], and modelling of dynamic foot mechanics by Caravaggi et al. [9]. The traditional conceptualisation of the windlass mechanism has the toes as an input crank arm that rotates to wind the plantar aponeurosis tight. We suggest that the input to the mechanism in gait is the force from the ankle plantar flexors pulling on the calcaneus and compressing the arch to tension the plantar aponeurosis, which in turn pulls the toes into the substrate and creates an internal flexion moment about the MTPj. The original paper by Hicks [5] refers to this as the reverse windlass. The resulting rotation of the MTPj depends on the moment balance with the external moment of the ground reaction force.

Plantar aponeurosis length data alone does not explain the foot mechanics observed in Experiment 1. Plantar aponeurosis length at the onset of MTPj extension was not different between loading conditions (Fig. 5A), despite the greater compression of the MLA with increased actuator loading. This was because the additional MLA compression with increased actuator loading was offset by slight MTPj flexion. Interestingly, this unintended scenario meant that at the start of MTPj extension, the plantar aponeurosis was at a similar length in all three loading conditions (Fig. 5A). Given this, if the windlass mechanism were purely passive, one would expect that when the matched toe extension torque was applied, the MTPj would extend by a similar amount in all conditions. This was not the case, with toe extension being significantly less in the mid and high loading conditions (Figure 4A) even though the same torque was applied. This resulted in a greater quasi-stiffness of the MTPj, as actuator load (and SO activation) increased, but no increase in plantar aponeurosis stretch was observed with increasing load (Fig. 5). This is a different result from similar mechanical tests on cadaver feet [13], demonstrating that in-vivo there are active mechanisms contributing. The explanation lies in the finding that quasi-stiffness of the MTPj was significantly influenced by activation of the plantar intrinsic foot muscles. Therefore, not only is the windlass mechanism dependent on ankle plantar flexor contraction, it is mediated by activation of the plantar intrinsic foot muscles. Interestingly this shows that the plantar intrinsic foot muscles can limit the strain experienced by the plantar aponeurosis by resisting MTPj extension.

Experiment 2 sought to extrapolate some findings of Experiment 1 to walking gait. It showed that during walking, greater MTPj flexion moments were produced during a more forceful push-off without the need for increased MTPj extension (Fig. 6C), and without increased strain of the plantar aponeurosis (Fig. 6D). In fact, our results showed that only minimal length change of the plantar aponeurosis occurred during MTPj extension (Fig. 6D). This agrees with more detailed modelling of plantar aponeurosis mechanics [24] that showed minimal changes in plantar aponeurosis tension during this phase. This suggests that increasing MTPj moments during MTPj extension (Fig. 6C) are not linked to rising plantar aponeurosis strain. Instead, the greater MTPj flexion moments observed with a more forceful push-off occurred concurrently with large increases in activation of toe flexor muscles FDB and AH (despite its name, AH contributes to great toe flexion). Thus, it seems that plantar intrinsic foot muscles are responsible for increasing tension about, or 'stiffening', the MTPj, when it is necessary to alter gait mechanics. This supports ideas proposed in our prior work, where using a nerve block to prevent active contraction of plantar intrinsic foot muscles reduced MTPj quasi-stiffness, and that this affected push-off power generated by foot and ankle structures [20]. The present findings also support Caravaggi et al.'s [9] conclusion that muscular contributions modulate foot function with changes in walking speed, as plantar aponeurosis lengths alone could not explain the observed mechanics. Of course, we have only described one case of changing gait mechanics, and it would be beneficial to observe the same for other scenarios such as those from other studies linking plantar intrinsic muscle activation to changing foot

power profiles for various locomotor tasks [15, 18, 25, 26]. Regardless, we believe the two experiments in this paper should provoke further discussion of the role of the windlass mechanism statically, and in gait.

We are not refuting the existence of the windlass mechanism as described by Hicks [5]. Clearly the plantar aponeurosis connects the toes to the calcaneus, and in the absence of other factors, extending the MTPj will 'tie the arch together'. It is also seems that, during late stance of walking and running, extension of the MTPj allows the plantar aponeurosis to shorten more slowly. In this sense, the windlass mechanism can be considered to help prevent an earlier drop in plantar aponeurosis tension than would be observed if the MTPj were locked. As such, the toes and MTPj might be considered to provide a catch -like effect, delaying the release of energy from the plantar aponeurosis until very late in the stance phase. However, our present study and prior data [20] suggest that the required tension across the MTPj during gait is achieved via active muscular contraction, and that any adjustments to this tension rely on active muscular control. Ultimately this seems sensible, as to achieve greater tension passively would require the plantar flexors to maintain or increase force output in late stance [12], and the MTPj to extend further to strain the plantar aponeurosis [13].

The necessity for active muscular control to produce normal foot mechanics should be of interest to designers of prosthetic feet, or of footwear designed to assist foot mechanics. At present, the majority of foot prostheses are passive elastic structures, and these will never be able to provide the adaptability of function that the human foot achieves through active muscular contributions. Similar issues face shoe manufacturers who have developed passive-elastic midsole plates to optimise foot mechanics during long distance running. These shoes will not have tuneable stiffness properties that might be needed for changes of speed, surface grade, or terrain.

From a static perspective, one must be careful not to assume any manual raising of the toe to be passive. A common clinical test for plantar fasciitis is the windlass test, which uses theoretically passive manual extension of the MTPj by the clinician to load the plantar fascia and potentially elicit a pain response [27]. Our results suggest that a muscular response might be evoked by this test (particularly when weight bearing) that would confound the assumption of it being passive. Also, that the plantar intrinsic muscles can offload plantar aponeurosis strain might be of interest to clinicians and athletic trainers who seek to rehabilitate or prevent plantar fasciitis. This is a common syndrome resulting from repeated loading of the plantar structures in the foot [23]. Our findings suggest that strengthening of the plantar intrinsic muscles has the potential to shield passive structures from larger strains that could be contributing to this condition.

A limitation of this study is that we did not measure muscle activation from the extrinsic muscles that flex the MTPj, flexor hallucis longus and flexor digitorum longus. These muscles are larger than the intrinsic muscles analysed here, and could also be making a contribution to MTPj mechanics. However, if these muscles are also important, it would only serve to strengthen the argument that active muscular contributions are key to mediating the windlass mechanism. It would be valuable to know if these muscles contribute, to help inform specificity of training relevant muscles in a clinical setting. We also used a relatively simple conceptual model of the plantar aponeurosis to capture significant changes in length. More detailed models informed by the elastic properties of the plantar aponeurosis might allow future studies to assess its force contribution and true strain. For the present study, we believe our model is sufficient to capture that there must be muscular contributions to MTPj mechanics. Unfortunately, due to poor motion capture data for the calcaneus markers during early-mid stance, we were unable to model plantar aponeurosis length until the second half of stance in Experiment 2. Finally, we only measured EMG from one plantar flexor (SO) and not from either head of gastrocnemius, whose force production might also be relevant. In Experiment 1, where this was of interest, we placed the participants in a flexed knee position that would minimise the contribution of the gastrocnemius to ankle joint moments.

Conclusions

In summary, our results show that maintaining tension across the plantar aspect of the foot involves active muscular contraction of ankle plantar flexors and plantar intrinsic muscles. Therefore, the passive windlass mechanism is not the foot's source of rigidity for push off against the ground during gait. In fact, within the foot, increasing push-off for changes in gait mechanics relies primarily on foot muscles and not winding of the plantar aponeurosis via the windlass mechanism.

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Data, code and materials

Data underlying the analyses presented in this paper have been uploaded as supplementary material

Author's contributions

DF conceived and designed the experiments, collected and analysed the data for both experiments, drafted and approved the manuscript. LK conceived and designed the experiments, collected the data for experiment 1, and revised drafts of the manuscript. JB collected and analysed data for experiment 2, and contributed to drafting of the manuscript.

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Figures

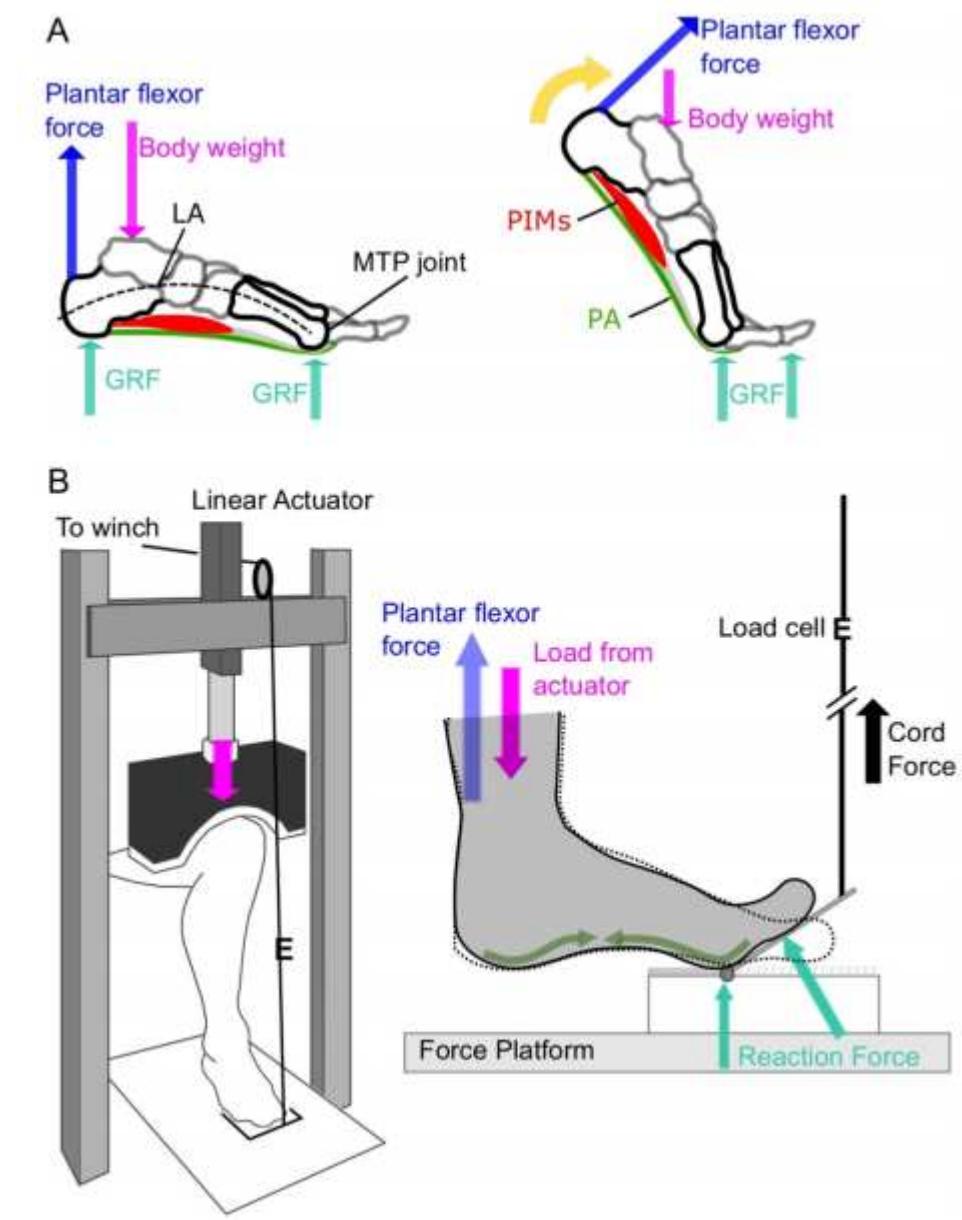

Figure 1 (A) An illustration of relevant foot structures and their interaction with foot posture and loading during gait. The plantar aponeurosis (PA) connects the calcaneus and the proximal phalanges along the plantar aspect of the foot by wrapping around the metatarsal-phalangeal joint (MTP_j). Body weight and plantar flexor forces flatten the

longitudinal arch (LA) and tension the plantar aponeurosis in early-mid stance (left). In late stance the foot rotates, extending the MTP_j, and supposedly engaging the windlass mechanism to tension the plantar aponeurosis (right). The plantar intrinsic foot muscles (PIMs) are highly active at this time, resisting MTP_j extension. (B) the setup from experiment 1, implemented to emulate loads experienced during stance in a controlled manner. The left image shows the full setup of the participant's leg in the loading apparatus. The right image is a close-up of the setup around the foot and the forces acting on the foot. Force was initially applied by the actuator to represent different vertical loads (0.5, 1.0, and 1.5 body weights). Winching of a cord then extended the MTP_j via a hinged metal plate, until a predefined maximum target force of 250 N was reached in the cord.

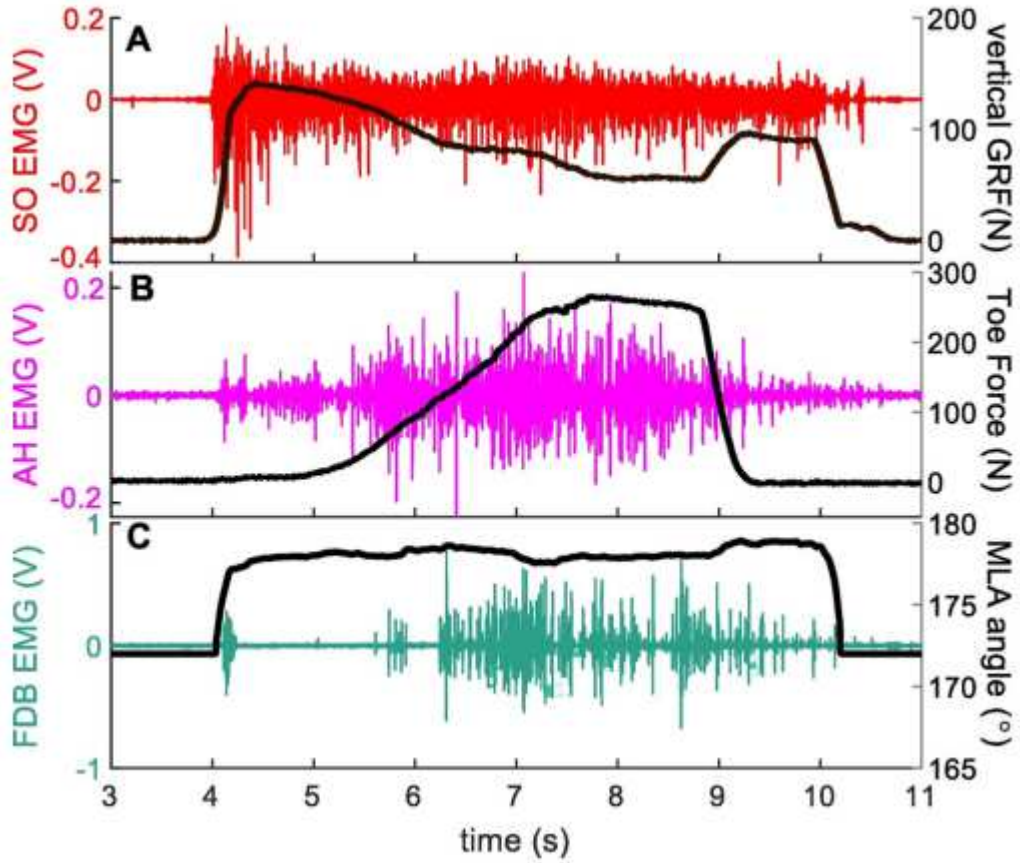

Figure 2 Representative data from a 1.5 BW loading trial from one participant. (A) Soleus (SO) EMG (red line) typically showed a large burst from the onset of actuator loading (indicated by rapid rise in vertical ground reaction force (GRF) – black line) until release of the actuator load. EMG signals for abductor hallucis (AH – panel B, magenta line) and flexor digitorum brevis (FDB – panel C, teal line), tended to increase as the extending force was applied to the toes (panel B- black line) was applied. The medial longitudinal arch (MLA) was compressed (panel C – black line) upon initial actuator loading. For the 1.5 BW loading, the MLA did not notably rise when the MTP_j was extended, indicating that the windlass mechanism could not be implemented under those conditions.

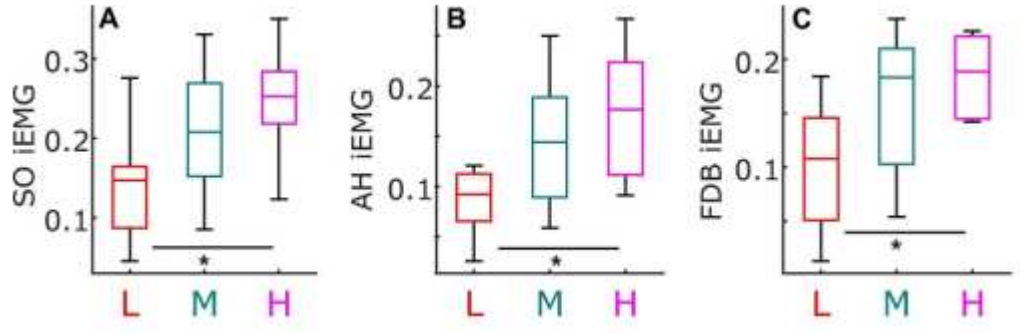

Figure 3 . Integrated normalised RMS EMG from the onset of actuator loading until peak toe extension increased for all three measured muscles across increasing loading conditions (Low - L, Mid – M, High – H). Linear mixed-effects modelling showed a significant fixed effect of condition on iEMG (n =10; MLRT p values - SO: p = 0.0005, AH: p =

0.0001, FDB: p = 0.004), while accounting for a random effect of participant. Therefore increased actuator load was considered synonymous with increased levels of muscle contraction.

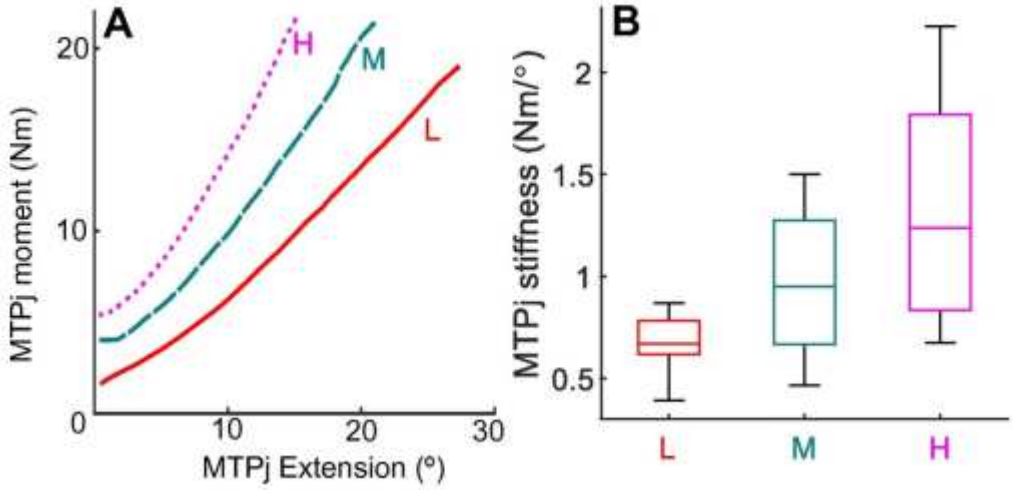

Figure 4. Quasi-stiffness of the MTP_j determined during forcible extension of the MTPj, increased with increasing actuator loads (and muscle activations). Group mean (n=10) plots of MTPj extension vs. MTPj moment highlight relatively less extension of the toes for a given magnitude of moment, as actuator load increased from low (L), to mid (M), to high (H). The slope of a linear to fit to the MTPj extension-moment relationship was calculated for each participant in each condition, and a box-and-whisker of this data is shown in panel B.

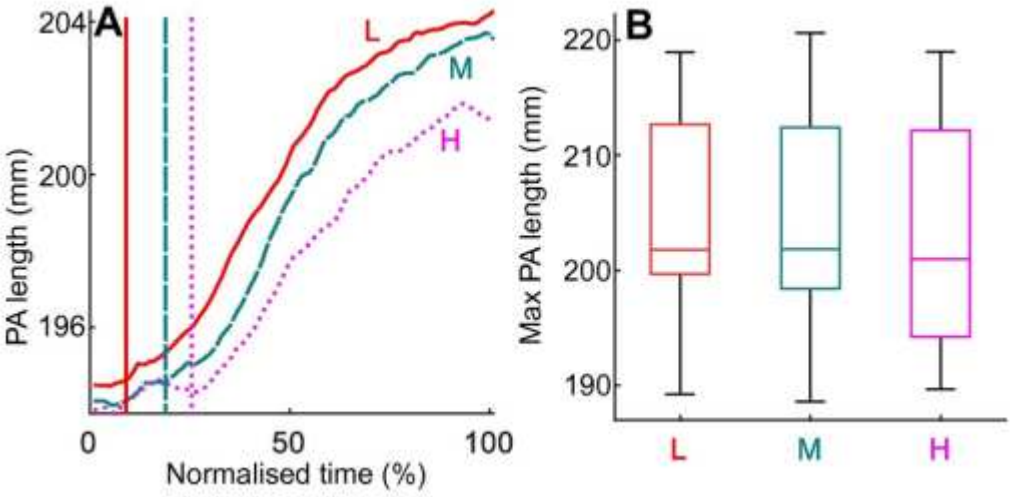

Figure 5. The plantar aponeurosis modelled length increased as the MTPj was extended in all actuator load conditions (A – onset of MTPj rotation indicated by vertical lines), but the final length reached was not significantly different between those conditions (B). Data in panel A are group means (n=10), and panel B shows a box and whisker plot of group data.

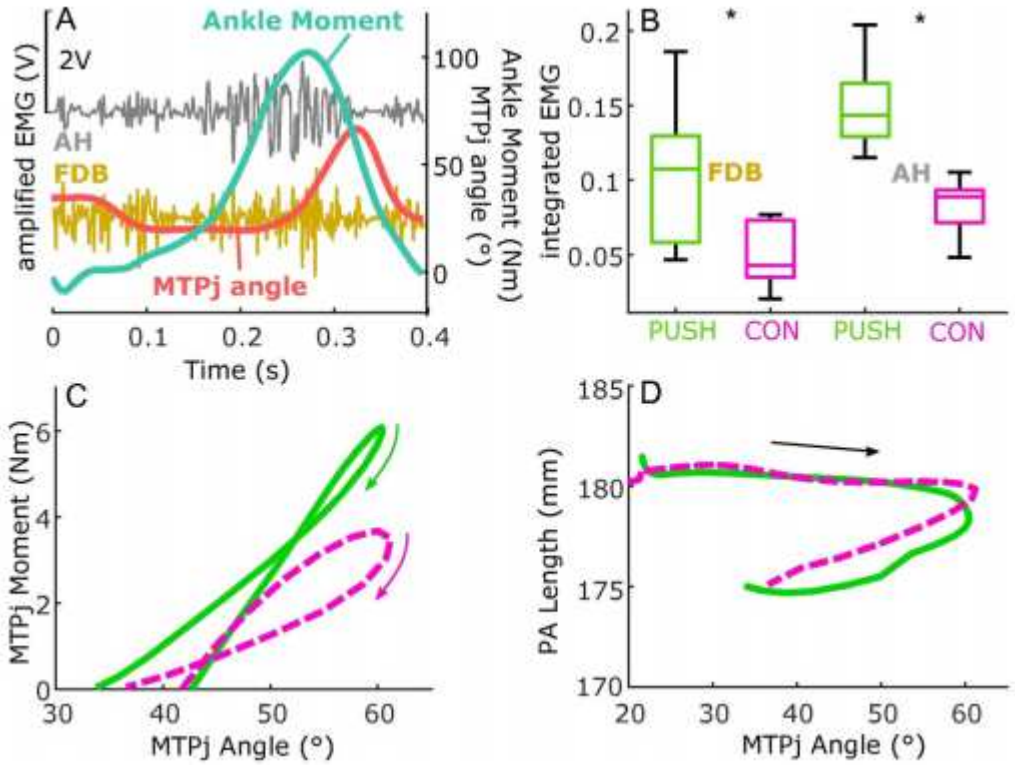

Figure 6. In Experiment 2, AH and FDB showed characteristic bursts of EMG activity during the stance phase of walking, but particularly in mid-late stance as the MTPj was extended (A – sample data from the stance phase one participant walking). MTPj extension occurred as the ankle plantar flexion moment lessened (A). The integrated RMS EMG for both muscles was significantly greater during the stance phase of the PUSH condition (B –box-and-whisker plot of group iEMG, n=9). As the MTPj extended then flexed in mid-late stance, the MTPj moment increased and decreased (C). As can be seen from the MTPj work loops in panel C, greater peak moments were reached in the PUSH condition (solid line) than the control walking (dashed line). Despite this, plotting plantar aponeurosis (PA) length vs MTPj angle during this phase of stance (D), showed no difference in PA length changes, inferring that joint level function is not dictated by purely passive elastic structures. Panels C and D show group (n=9) mean data.