A parametric study of effect of experimental tibialis posterior muscle pain on joint loading and muscle forces-Implications for patients with rheumatoid arthritis?

Simonsen, Morten Bilde; Yurtsever, Aysun; Næsborg-Andersen, Ketill; Leutscher, Peter Derek Christian; Hørslev-Petersen, Kim; Hirata, Rogerio Pessoto; Andersen, Michael Skipper

Published in:
Gait & Posture

DOI (link to publication from Publisher):
10.1016/j.gaitpost.2019.06.001

Publication date:
2019

Document Version
Accepted author manuscript, peer reviewed version

Title: A parametric study of effect of experimental tibialis posterior muscle pain on joint loading and muscle forces—Implications for patients with rheumatoid arthritis?

Authors: Morten Bilde Simonsen, Aysun Yurtsever, Ketill Næsborg-Andersen, Peter Derek Christian Leutscher, Kim Hørslev-Petersen, Rogerio Pessoto Hirata, Michael Skipper Andersen

PII: S0966-6362(18)31951-9
DOI: https://doi.org/10.1016/j.gaitpost.2019.06.001
Reference: GAIPOS 6819

To appear in: *Gait & Posture*

Received date: 17 December 2018
Revised date: 22 May 2019
Accepted date: 4 June 2019

Please cite this article as: { https://doi.org/

Title: **A parametric study of effect of experimental tibialis posterior muscle pain on joint loading and muscle forces – implications for patients with rheumatoid arthritis?**

Morten Bilde Simonsen[1,2], AysunYurtsever[2,3], Ketill Næsborg-Andersen, Peter Derek Christian Leutscher[2], Kim Hørslev-Petersen[4], Rogerio Pessoto Hirata[1], Michael Skipper Andersen[5].

Journal: Gait and Posture

Word count: 2993

Article Type: Full paper

[1]SMI®, Department of Health Science and Technology, Aalborg University, Fredrik Bajers Vej 7, DK-9220 Aalborg East, Denmark
[2]Centre for Clinical Research, North Denmark Regional Hospital, Bispensgade 37, DK-9800 Hjoerring, Denmark
[3]Department of Rheumatology, Hjørring Hospital, Bispensgade 37, DK-9800 Hjørrring, Denmark
[4]King Christian 10th Hospital for Rheumatic Diseases, University of Southern Denmark, Toldbodgade 3, DK-6300 Gråsten, Denmark
[5]Department of Materials and Production, Aalborg University, Fibigerstraede 16, DK-9220 Aalborg East, Denmark

Corresponding author:

Associate Professor Michael Skipper Andersen, M.Sc.EE., Ph.D.
Department of Materials and Production
Aalborg University, Denmark
Fibigerstraede 16, DK-9220 Aalborg East
Email: msa@mp.aau.dk
Phone: +45 30354170

Highlights

- TP pain and simulated reduced TP muscle strength caused altered muscle recruitment
- Flexor digitorum longus and flexor hallucis longus compensates for the TP muscle.
- The found compensating strategy puts higher forces on the ankle joint.
- This could be a contributing factor to development of forefoot pain in RA.

Abstract word count: 297

Background

Foot pain and deformities are commonly encountered in patients with rheumatoid arthritis (RA). Likewise, Posterior tibial tendon dysfunction (PTTD) is commonly involved in development of foot and ankle abnormalities and has been reported with a prevalence in two-thirds of the RA patients.

Research question

Redundancy in the physiological function between different muscles provides the central nervous system multiple options to perform the same movement but which muscles compensate for the impairment of the tibialis posterior (TP) muscle? And how does these changes affect ankle joint loading?

Methods

Experimental and computational disciplines were applied to investigate changes in muscle forces as result of induced pain in the right TP muscle. Twelve healthy subjects were enrolled in the study. Experimental pain was induced in the TP by a single ultrasound graphically guided injection of 1 mL hypertonic saline (5.0% Sodium Chloride). The participants' gait was assessed by skin marker-based motion capture and force plates. Musculoskeletal models were used to investigate compensation mechanisms systematically in the lower under extremity when TP muscle was recruited less as a consequence of the induced pain.

Results

Experimental TP muscle pain and simulated reduced strength caused altered muscle recruitment and made the flexor digitorum longus and flexor hallucis longus muscles compensated for the impairment of the TP muscle. Further, the resultant ankle joint force was increased as the strength of the TP muscle was reduced.

Significance

The compensation mechanism observed in the present study indicate that alterations in muscle recruitment and muscle force distribution as a result of the underlying disease inflammation itself may contribute to development of chronic foot pain and deformities in patients with RA . Further studies are required to understand the role of PTTD in occurrence of those late adverse musculoskeletal manifestations aiming at search for early preventive strategies.

Keywords: max 5

Tibialis posterior, dysfunction, musculoskeletal modelling, muscle compensation, parametric study

Introduction

Posterior tibialis tendon dysfunction (PTTD) , which affects different patient groups, is one of the most common causes in development of foot and ankle abnormalities [1]. The TP muscle is the strongest inverter of the foot and also serves as a plantar flexor. Moreover, the muscle also functions as a dynamic foot arch stabilizer [1]. During stance of normal gait, the TP muscle generates a semi-rigid midfoot by locking the bones in the midfoot [1]. In patients with rheumatoid arthritis (RA), PTTD has been reported with a high prevalence of 64% [2]. RA is a chronic polyarticular autoimmune disease that frequently affects the joints and soft tissues of the feet, with foot pain being the most common reason for incapacity in RA patients [3]. Besides pain, foot deformities, such as hallux valgus, claw toes, metatarsal instability, cross over toes and pes planus, are common consequences of the disease [4].

The muscles and joints of the foot and ankle are complex in their geometry and function, and have a high level of redundancy. It is well-known that redundancy in the muscular system gives the central nervous system multiple solutions in order to perform the same movement [5]. Dysfunction, pain or even loss of a single muscle, will force other muscles to compensate for the weakened muscle [6]. A previous study, investigating the effect of experimental knee pain on quadriceps muscle strength, found a reduction of up to 15% in the maximal joint torque, measured with an isokinetic dynamometer, indicating that pain is a significant cause of muscle weakness [7].

Patients with musculoskeletal pain present a number of confounding factors such as inflammation, joint laxity, effusion, muscle atrophy, receptor damage, and other musculoskeletal changes [8,9]. As a result, different types of experimental pain models have been developed within pain research to evaluate the effect of pain in an otherwise healthy system [9]. These experimental pain models have shown good replication in patients with musculoskeletal pain [9,10]. Various theories exist on how

pain affects muscle recruitment. One of the most well-documented of these is the pain adaptation theory that proposes that a painful muscle is less likely to be recruited and generate force [11]. To this end, musculoskeletal modelling can be used as a tool to investigate muscle compensation mechanisms systematically when specific muscles becomes dysfunctional.

The aims of this combined experimental and computational study were two-fold: 1) to investigate which muscles compensate for impairment of the tibialis posterior (TP) muscle caused by inducement of pain, and 2) to examine how the muscle changes affect the ankle joint loading.

Methods

Subjects

Twelve healthy subjects with no preexisting musculoskeletal abnormalities or pain were recruited for this study. Participant characteristics (mean ± SD) were as follows: age 28.3 ± 1.8 years, height 180.3 ± 9.8 cm and body mass 83.7 ± 12.0 kg.

Ethical considerations

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval for this study was granted by the North Denmark Region Committee on Health Research Ethics (N20170066). Written informed consent was obtained from all participants.

Experimental protocol

The experimental design was consisting of one session. Three self-paced baseline gait trials (1.06 ± 0.09 m/s) were performed before experimental pain was induced by injection of hypertonic saline via a single injection into the TP muscle. Prior to injections, an ultrasound examination was performed with subjects placed in a supine position with their legs slightly internally rotated. A safety window was identified to avoid artery, veins and neurovascular bundles [12]. After prepping

the skin with an alcohol wipe and iodine, 1 mL of hypertonic saline (5.0% NaCL) was injected at the upper third point of the tibia via an anterior approach by use of a ultrasound-guided techniques described by Rha et al. (2010) [12]. Immediately after the injection, three self-paced (1.05 ± 0.09 m/s) gait trials were performed again. The experimental pain intensity was assessed on an eleven-point numerical verbal rating scale (NRS) where "0" indicated 'no pain' and "10" 'maximum pain'.

Gait analysis

Motion capture of gait was collected at 100 Hz by an eight-camera infrared Qualisys system (Oqus 300 series, Qualisys, Sweden) for the baseline and after an injection of hypertonic saline in the right TP muscle. Ground reaction forces and moments were collected at 1000 Hz from force plates installed in the floor (AMTI, USA). A protocol of 32 passive reflective markers were used to track the lower body (Figure 1). Twelve markers were placed on palpable anatomical landmarks and four markers on each shoe. Four clusters with three markers in each were placed on the lower and upper leg. Data were processed in Qualisys Track Manager Version 2.16 (Qualisys, Gothenburg, Sweden) and exported in to a C3D format.

Estimation of joint loading and muscle forces

To estimate muscle and joint loading, musculoskeletal models were created in the AnyBody Modeling System v. 7.0.1 (AnyBody technology, Aalborg, Denmark). Muscle forces and joint loading during stance of gait were estimated by the "Anatomically Scaled Model" from Lund et al. (2015) [13]. The model was previously validated against in-vivo measured knee forces [14]. The model is scaled based on a standing reference without any need for additional input from the modeler. A nine-segment stick-figure of the lower extremity was generated from a standing reference trial, performed in a natural position with arms along the side. The standing reference

included four additional markers, compared to the gait trials, representing the medial malleolus and medial femoral epicondyles these was used to compute the joint axis of the knee and ankle joints [13]. The hip joint was modeled as a spherical joint and its position was calculated via a regression formula with leg length, pelvic depth and width as predictors [15]. The knee and talocrural joints were modeled as revolute joints. The subtalar joint was positioned 10 mm inferior to the ankle joint and oriented with a joint inclination of 42° and deviation of 23° with respect to the line joining the heel and front toe marker [16]. The Twente Lower Extremity model available in the AnyBody Managed Model Repository based on the cadaver dataset of Horsman et al. (2007) was nonlinearly morphed to match the joint morphology of the stick-figure model based on a non-linear radial basis function interpolation scheme, making the two models identical with respect to segment sizes and joint axis [13,17]. The stick-figure was used to track marker data and compute joint angles during the entire stance phase using inverse kinematics [18]. Estimates of muscle and joint reaction forces were obtained through inverse dynamic analysis on the morphed musculoskeletal model with kinematic inputs estimated with the stick-figure model and the measured ground reaction forces (Figure 2). Joint coordinate systems were defined in accordance with ISB recommendations [19].

Assessment of muscle recruitment

The muscle recruitment was solved by minimizing a polynomial cost function, *G*, subject to the dynamic equilibrium equations and to the constraint that muscles can only pull and cannot generate a force larger than their instantaneous strength [20]. To account for sub-divided muscles, a normalization factor,n_{i}, based on muscle physiological cross sectional area was implemented

$$\begin{aligned} \min_{\mathbf{f}} & \; G\left(\mathbf{f}^{(\mathrm{m})}\right) = \sum_{i=1}^{n^{(\mathrm{m})}} n_{\mathrm{i}} \left(\frac{f_{\mathrm{i}}^{(\mathrm{m})}}{N_{\mathrm{i}}}\right)^3 \\ \text{s.t.} & \; \mathbf{Cf} = \mathbf{d} \\ & \; 0 \le f_i^{(M)} \le N_{i,} \end{aligned} \tag{1}$$

(m) indicates muscles elements and $\mathbf{f}^{(m)}$ is a vector of muscle forces, $n^{(m)}$ denotes the number of muscles in the model, $f_i^{(m)}$, denotes the muscle force of the i^{th} muscle, and N_i is the muscle strength of the i^{th} muscle element. $\mathbf{C}$ is a coefficient matrix for all the unknown forces in the problem and $\mathbf{f}$ denotes joint reaction and muscle forces. $\mathbf{d}$ contains the external loads and inertia forces.

Parametric study

To investigate the compensatory mechanism, the isometric strength $N_{i,}$ of the TP muscle was systematically reduced to 40, 50, 60, 70, 80, and 90% of the default strength. Multiple different reductions factors of the TP muscle strength were chosen due to the uncertainty of how significantly the induced pain affects the recruitment of the TP muscle. The stance phase was defined as the first frame before the foot hit the force plate and the first frame after. The stance phase of three trials for each subject were used for further analysis. Joint and muscle forces were normalized to bodyweight.

Results

Pain

Subjects reported by use of NRS an experimental mean pain level of 5.8 ± 1.7 after the gait trials.

Muscle forces of the shank

The mean TP muscle force decreased after simulating the experimental pain by lowering the maximal isometric force of the TP muscle (Figure 3), and, at the same time, the force of the flexor digitorum longus and flexor hallucis longus increased. The muscle force of soleus and gastrocnemius increased, but only for the lateral side of the muscle. The forces of extensor hallucis longus and extensor digitorum longus decreased. The general dose response relationship observed is

that, the lower the isometric strength of the TP muscle, the greater compensation from the surrounding muscles in the lower leg.

Peak muscle forces for all conditions

Almost no change in peak muscle forces for the TP muscle was observed between the pain-free and pain walk with 100 % isometric strength of the TP muscle. The peak muscle forces for both parts of the TP muscle was gradually reduced for all subjects during the pain walk (Figure 4). Meanwhile, the muscle forces of soleus lateralis, flexor digitorum longus, flexor hallucis longus, and gastrocnemius lateralis were decreased. Minor changes were observed for soleus medialis, gastrocnemius medialis, tibialis anterior, extensor digitorum longus, extensor halluces longus and plantaris.

Joint ankle force

Reduced isometric strength in the TP muscle increased the total resultant ankle joint force increased with altered muscle recruitment. Additionally, the anterior/posterior force is making a substantial shift from being only a posterior force for the pain walk with 100% TP strength to shift the force to the anterior direction already at 90% of default TP isometric strength. Furthermore, for the lateral/medial force, the most conspicuous feature is the temporal pattern for the 100% walk compared to the reduced conditions, especially around 60-70 % of the stance phase. Finally, the ankle loading in the ML direction shows a substantial increase in the medially-directed peak in late stance between 100% and 90% of TP strength, with subsequent reductions in TP strength leading to lower peak medially-directed force in late stance. In fact, the late-stance peak force at 100% of TP strength is comparable to that at 50% of TP strength. (Figure 5).

Discussion

Deep muscles of the posterior compartment

The present study has shown that other plantar flexors and inverter muscles compensate for the impairment of the TP as simulated by a reduced isometric strength. Particularly interesting is it that the flexor digitorum longus and flexor hallucis longus becomes more active when the TP is impaired. From an anatomical point-of-view, this would make good sense, since all three muscles act as inverters and plantar flexors of the foot. However, flexor digitorum longus and flexor hallucis longus muscles attach on the four lateral distal phalanges and the hallux toe, respectively. This creates a situation where the plantarflexion and inversion of the foot would to a greater extent be driven by the toes. These alterations in the muscle recruitment could be a possible explanation for the development of the severe forefoot pain observed in patients with RA and perhaps also the foot deformities seen in patients with RA. Previous studies have proposed this mechanism [21,22], but it remains challenging to investigate. A magnetic resonance imaging morphometry study by Wacker and colleagues (2003) have shown that patients with adult-acquired flat foot, due to PTTD, are observed increased the cross-sectional area of the flexor digitorium longus. However, the study did not report findings related to the flexor hallucis longus muscle [23].

In study applying intramuscular electromyography (EMG), by Barn et al. (2013), it was shown that patients with co-existence of RA and PTTD were found with increased TP muscle activity compared to healthy subjects [24]. However, since the TP is dysfunctional in these patients, it is questionable whether the muscle is capable of producing a higher force than a healthy TP. For comparison between subjects, normalization of the EMG to maximum voluntary contractions is required. However, with the presence of muscle and joint pain, obtaining an accurate measurement may be compromised, which will make the EMG signals during the dynamic trials to appear higher than they truly are [25].

Superficial muscles of the posterior compartment

The results of the present simulation study also revealed alterations of the lateral part of the soleus and gastrocnemius muscles, whereas changes in the medial part of the muscles were limited compared to the lateral part. This observation could be because the lateral part of the muscles have similar prospect to generate moments like TP. This might partly be explained by the fact that the anatomical location of the origin of the lateral part of soleus and gastrocnemius are close to the origin of the impaired TP muscle, making them more mechanical suitable to compensate for the impaired TP. A small increase of the muscle force of the plantaris was also observed, but it is questionable if the small increase would have any clinical relevance. Regardless, the study shows that all the plantar flexors muscles of the superficial posterior compartment assist in compensating for the impaired TP.

Anterior compartment of the leg

Almost no change in muscle forces of the muscles located in the anterior compartment was observed. Although previous literature has speculated whether the tibialis anterior compensates for the dysfunction of the TP muscle [26], this study could not demonstrate any increase in the muscle forces of the tibialis anterior, extensor hallusic or extensor digitorum longus muscles when the TP muscle was less recruited. This observation indicates that the muscles located in the posterior compartment are better suited mechanically for this purpose of muscle compensation.

Ankle joint load

The study also found that the total resultant ankle joint forces increase when the TP muscles is less recruited, which indicates that the applied compensating muscle recruitment solution puts a higher stress load on the ankle joint, especially at the push off (at 80% of the stance phase). Particularly, the compression force of the ankle joint is increased when TP becomes recruited less. A cause for

modifications in ankle loading could be that the increased forces in the lower leg muscles are intensifying the ankle joint loading. All of the muscle forces of the posterior compartment muscles are increased, except for the TP, showing the importance of normal TP function in order to have normal ankle loading. The increased ankle joint loading might be a contributing factor to ankle pain among patients with RA [3]. It is plausible that loading of some of the intrinsic foot joints would also have been affected by the reduced recruitment of TP. Especially, since the muscles attaching on the distal phalanges (flexor digitorium longus and flexor hallucis longus), to a greater extent compensate for the impaird TP muscle. However, this mechanical element requires a multiple segment foot model and could therefore not be investigated with the one segment foot model applied in this study.

Limitations

Besides the literature supporting the decrease in muscle force during pain [7,27], different reduction of the TP muscle strength were chosen due to the uncertainty of how significantly the induced pain affects the recruitment of the TP muscle. Despite different reduction levels, the trend was similar for all simulated conditions, whereas only the extent of compensation by other muscles varied.

The short pain habituation period (2-5 minutes) could have been considered as limitation in the study. It is possible that the kinematic data may have given different effects with a longer adaptation period, as studies of chronic pain patients have shown [1,22,24]. However, it is not expected that this would have affected the observed compensation mechanism, since kinematic input has only minor effect on muscle force distribution compared to the changes in the polynomial cost function. Comparison of the pain-free and pain induced walk with 100% isometric strength supports this assumption (Figure 4), indicating that altered muscle capacity is more significant than pure kinematics for the present study for the TP, flexor hallucis longus and flexor digitorum longus

muscles. However, a recent study based on the same gait data showed altered knee and hip joint angles during walking with experimental pain in the TP muscle [28], indicating that even temporary pain changes gait mechanics.

Another important limitation is the assumption that the underlying distribution of the muscle forces by the central nervous system is governed by the recruitment criterion in equation (1). However, several studies obtained good agreement with the measured joint forces as well as qualitative measured EMG activities with this criterion [29,30]. However, these agreements have been among subjects without pain and normal muscle function. Further, this study assumed that optimal compensation would occur when a muscle is in a state of pain. Therefore, the results from the simulation must also be interpreted with caution. The first initial reduction of TP muscle strength led to a considerable change in muscle recruitment and joint loading. This observation is probably due to the cubical muscle recruitment criteria used in the present study. In the recruitment criteria, muscle activities are raised to a power of three. Therefore, a nonlinear relationship between the muscle strength reduction and resulting muscle force is observed. For the same reason, we initially observe a relatively larger effect of the strength reduction than when the muscle strength is already reduced. Another limitation of the study is that EMG was not measured during the experiment. Future studies could try to measure intramuscular EMG of the involved muscles. This would also be a possible technique to validate the approach used in the present study.

In summary, this study has demonstrated that experimental TP muscle pain and simulated reduced TP muscle strength caused altered muscle recruitment and also that flexor digitorum longus and flexor hallucis longus muscles compensate for the impairment in the TP muscle. Both muscles shares the same function of TP. This could potentially be a contributing factor to development of forefoot pain and deformities in patients with RA. Even the initial 10% isolated reduction in TP

force caused the largest force incensement of the flexor digitorum longus and flexor hallucis longus muscles during gait. Suggesting the importance of early treatment.

ConflictOfInterest

The authors have no real or perceived financial and personal relationships with other people or organizations that could inappropriately influence (bias) our work. Examples of potential conflicts of interest include employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations.

Disclosure

The authors have no real or perceived financial and personal relationships with other people or organizations that could inappropriately influence (bias) our work. Examples of potential conflicts of interest include employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations.

Acknowledgement

This study was supported by Trygfonden (grant number 124714) and the Danish Rheumatism Association, (grant number R161-A5276). Trygfonden (www.trygfonden.dk/english). The Danish Rheumatism Association (www.gigtforeningen.dk/for-forskere/the-danish-rheumatism-association)

References

[1] R. Semple, G.S. Murley, J. Woodburn, D.E. Turner, Tibialis posterior in health and disease: a review of structure and function with specific reference to electromyographic studies, J. Foot Ankle Res. 2 (2009) 24. doi:10.1186/1757-1146-2-24.

[2] J. Michelson, M. Easley, F.M. Wigley, D. Hellmann, Posterior tibial tendon dysfunction in rheumatoid arthritis., Foot Ankle Int. 16 (1995) 156–161.

doi:10.1177/107110079501600309.

[3] L. Grondal, B. Tengstrand, B. Nordmark, P. Wretenberg, A. Stark, The foot: still the most important reason for walking incapacity in rheumatoid arthritis: distribution of symptomatic joints in 1,000 RA patients., Acta Orthop. 79 (2008) 257–61. doi:10.1080/17453670710015067.

[4] F. Brooks, K. Hariharan, The rheumatoid forefoot, Curr. Rev. Musculoskelet. Med. 6 (2013) 320–327. doi:10.1007/s12178-013-9178-7.

[5] J.J. Kutch, F.J. Valero-Cuevas, Muscle redundancy does not imply robustness to muscle dysfunction, J. Biomech. 44 (2011) 1264–1270. doi:10.1016/j.jbiomech.2011.02.014.

[6] C.S. Simpson, M.H. Sohn, J.L. Allen, L.H. Ting, Feasible muscle activation ranges based on inverse dynamics analyses of human walking, J. Biomech. 48 (2015) 2990–2997. doi:10.1016/j.jbiomech.2015.07.037.

[7] M. Henriksen, S. Rosager, J. Aaboe, T. Graven-Nielsen, H. Bliddal, Experimental knee pain reduces muscle strength, J. Pain. 12 (2011) 460–467. doi:10.1016/j.jpain.2010.10.004.

[8] L. Arendt-Nielsen, T. Graven-Nielsen, Muscle pain: Sensory implications and interaction with motor control, Clin. J. Pain. 24 (2008) 291–298. doi:10.1097/AJP.0b013e31815b608f.

[9] L. Arendt-Nielsen, T. Graven-Nielsen, H. Svarrer, P. Svensson, The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study, Pain. 64 (1996) 231–240.

[10] M. Henriksen, T. Graven-Nielsen, J. Aaboe, T.P. Andriacchi, H. Bliddal, Gait changes in patients with knee osteoarthritis are replicated by experimental knee pain, Arthritis Care Res. 62 (2010) 501–509. doi:10.1002/acr.20033.

[11] J.P. Lund, R. Donga, C.G. Widmer, C.S. Stohler, The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity, Can. J. Physiol. Pharmacol. 69 (1991) 683–694. doi:10.1139/y91-102.

[12] D.-W. Rha, S.H. Im, S.C. Lee, S.-K. Kim, Needle insertion into the tibialis posterior: ultrasonographic evaluation of an anterior approach., Arch. Phys. Med. Rehabil. 91 (2010) 283–287. doi:10.1016/j.apmr.2009.09.024.

[13] M.E. Lund, M.S. Andersen, M. de Zee, J. Rasmussen, Scaling of musculoskeletal models from static and dynamic trials, Int. Biomech. 2 (2015) 1–11. doi:10.1080/23335432.2014.993706.

[14] R.E. Richards, M.S. Andersen, J. Harlaar, J.C. Van Den Noort, Relationship between knee joint contact forces and external knee joint moments in patients with medial knee osteoarthritis : effects of gait modi fi cations, Osteoarthr. Cartil. 26 (2018) 1203–1214. doi:10.1016/j.joca.2018.04.011.

[15] M.E. Harrington, A.B. Zavatsky, S.E.M. Lawson, Z. Yuan, T.N. Theologis, Prediction of the hip joint centre in adults, children, and patients with cerebral palsy based on magnetic resonance imaging, J. Biomech. 40 (2007) 595–602. doi:10.1016/j.jbiomech.2006.02.003.

[16] V.T. Inman, The joints of the ankle, Williams & Wilkins, 1976. https://books.google.dk/books/about/The_Joints_of_the_Ankle.html?id=jplqAAAAMAAJ&redir_esc=y (accessed September 25, 2018).

[17] M.D. Klein Horsman, H.F.J.M. Koopman, F.C.T. van der Helm, L.P. Prosé, H.E.J. Veeger, Morphological muscle and joint parameters for musculoskeletal modelling of the lower extremity, Clin. Biomech. 22 (2007) 239–247. doi:10.1016/j.clinbiomech.2006.10.003.

[18] M.S. Andersen, M. Damsgaard, J. Rasmussen, Kinematic analysis of over-determinate biomechanical systems., Comput. Methods Biomech. Biomed. Engin. 12 (2009) 371–384. doi:10.1080/10255840802459412.

[19] G. Wu, S. Siegler, P. Allard, C. Kirtley, A. Leardini, D. Rosenbaum, M. Whittle, D.D. D'Lima, L. Cristofolini, H. Witte, O. Schmid, I. Stokes, ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion—part I: ankle, hip, and spine, J. Biomech. 35 (2002) 543–548. doi:10.1016/S0021-9290(01)00222-6.

[20] J. Rasmussen, M. Damsgaard, M. Voigt, Muscle recruitment by the min/max criterion - A comparative numerical study, J. Biomech. 34 (2001) 409–415. doi:10.1016/S0021-9290(00)00191-3.

[21] J. Tome, D.A. Nawoczenski, A. Flemister, J. Houck, Comparison of Foot Kinematics Between Subjects With Posterior Tibialis Tendon Dysfunction and Healthy Controls, J Orthop Sport. Phys Ther. 36 (2006) 635–644. doi:10.2519/jospt.2006.2293.

[22] J.W. Brodsky, J.J. Zubak, F.E. Pollo, B.S. Baum, Preliminary Gait Analysis Results After Posterior Tibial Tendon Reconstruction: A Prospective Study, Foot Ankle Int. 25 (2004) 96–100. doi:10.1177/107110070402500210.

[23] J. Wacker, J.D.F. Calder, C.M. Engstrom, T.S. Saxby, MR Morphometry of Posterior Tibialis Muscle in Adult Acquired Flat Foot, Foot Ankle Int. 24 (2003) 354–357. doi:10.1177/107110070302400409.

[24] R. Barn, D.E. Turner, D. Rafferty, R.D. Sturrock, J. Woodburn, Tibialis posterior tenosynovitis and associated pes plano valgus in rheumatoid arthritis: Electromyography, multisegment foot kinematics, and ultrasound features, Arthritis Care Res. (2013). doi:10.1002/acr.21859.

[25] W. Dankaerts, P.B. O'sullivan, A.F. Burnett, L.M. Straker, L.A. Danneels, Reliability of EMG measurements for trunk muscles during maximal and sub-maximal voluntary isometric contractions in healthy controls and CLBP patients, J. Electromyogr. Kinesiol. 14 (2004) 333–342. doi:10.1016/j.jelekin.2003.07.001.

[26] M.B. Pohl, M. Rabbito, R. Ferber, The role of tibialis posterior fatigue on foot kinematics during walking, J. Foot Ankle Res. 3 (2010) 1–8. doi:10.1186/1757-1146-3-6.

[27] T. Graven-Nielsen, H. Lund, L. Arendt-Nielsen, B. Danneskiold-Samsøe, H. Bliddal, Inhibition of maximal voluntary contraction force by experimental muscle pain: A centrally mediated mechanism, Muscle and Nerve. 26 (2002) 708–712. doi:10.1002/mus.10225.

[28] M.B. Simonsen, A. Yurtsever, K. Næsborg-Andersen, P.D.C. Leutscher, K. Hørslev-Petersen, M.S. Andersen, R.P. Hirata, Tibialis posterior muscle pain effects on hip, knee and ankle gait mechanics, Hum. Mov. Sci. 66 (2019) 98–108. doi:10.1016/j.humov.2019.04.002.

[29] M.A. Marra, V. Vanheule, J. Rasmussen, N.J.J. Verdonschot, M.S. Andersen, R. Fluit, B.H.F.J.M. Koopman, J. Rasmussen, N.J.J. Verdonschot, M.S. Andersen, A Subject-Specific Musculoskeletal Modeling Framework to Predict in Vivo Mechanics of Total Knee Arthroplasty., J. Biomech. Eng. 137 (2015) 020904. doi:10.1115/1.4029258.

[30] R. a. Bogey, J. Perry, a. J. Gitter, An EMG-to-force processing approach for determining ankle muscle forces during normal human gait, IEEE Trans. Neural Syst. Rehabil. Eng. 13 (2005) 302–310. doi:10.1109/TNSRE.2005.851768.

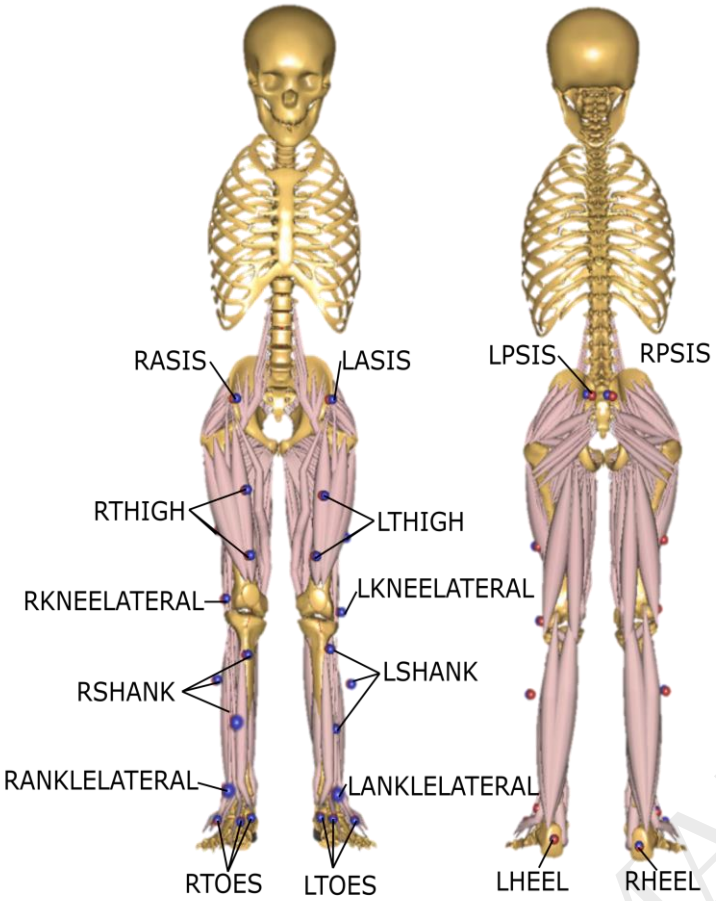

Figure 1: Illustration of the 32 passive reflective markers used to track the lower extremity.

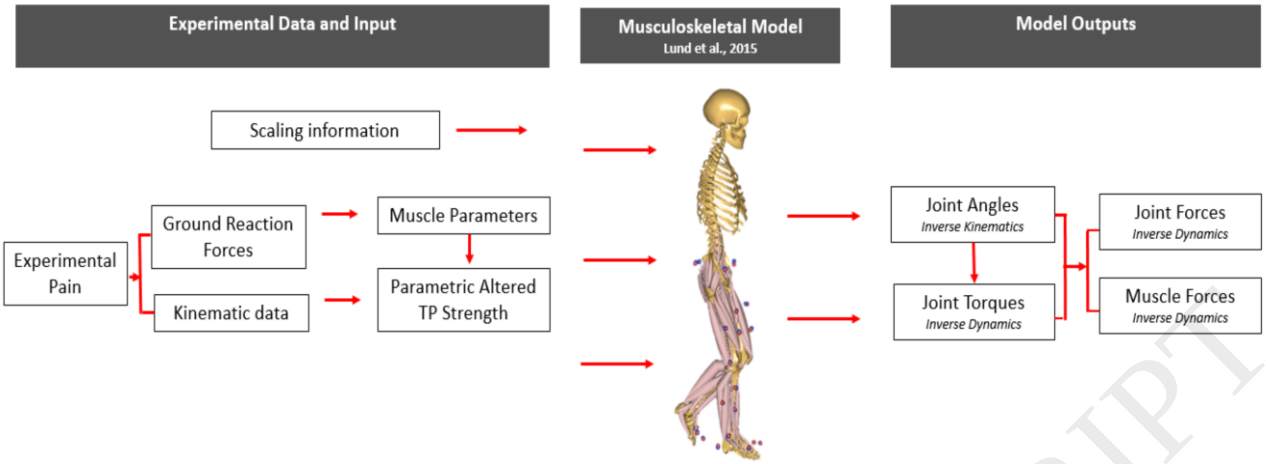

Figure 2: Overview of the study design and analysis workflow. An AnyBody musculoskeletal model was used to perform kinematic and inverse dynamic analysis of experimental data with TP pain and reduced TP muscle strength.

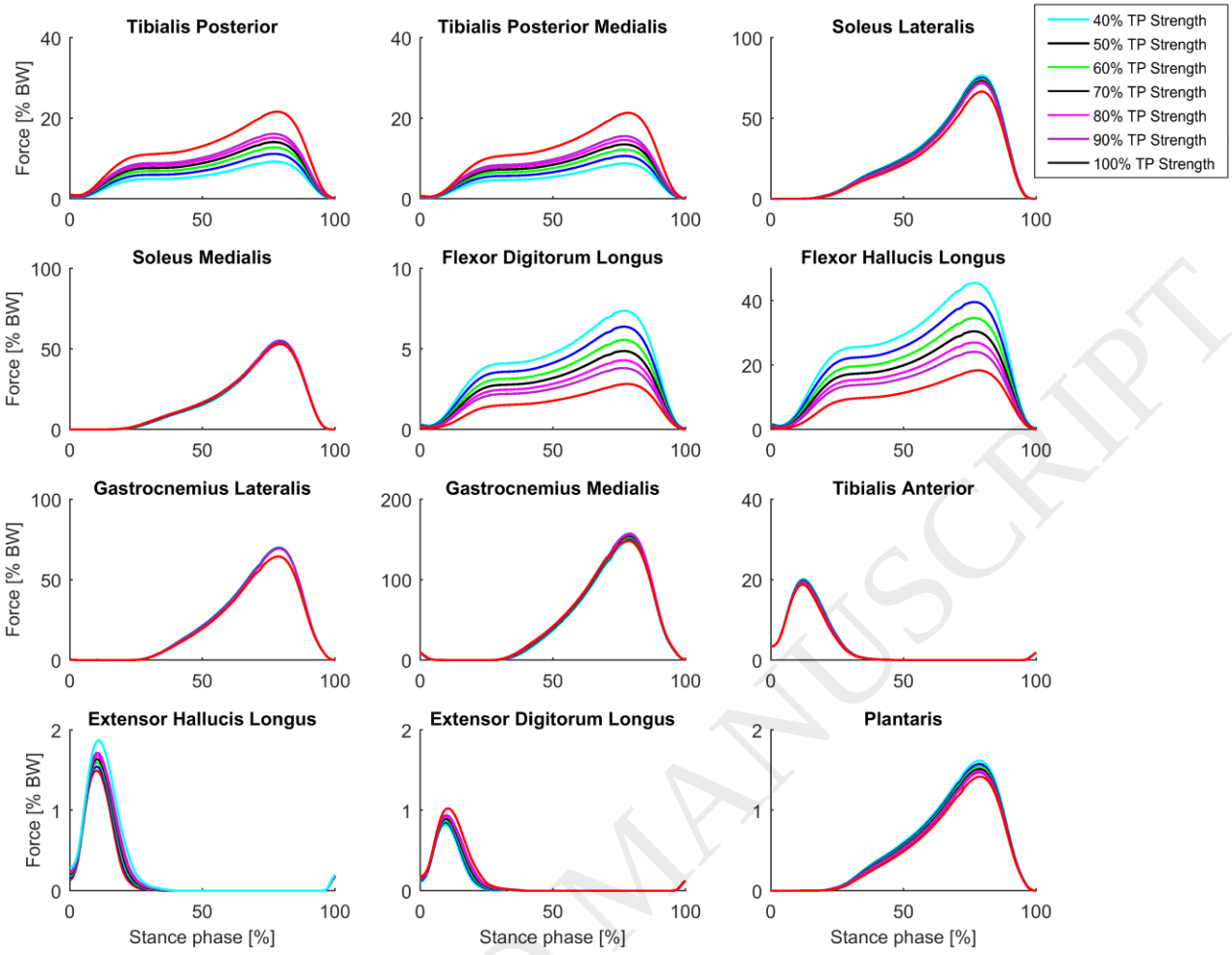

Figure 3: Mean muscle force of selected muscles of the lower leg for 100 to 40% of default TP muscle strength

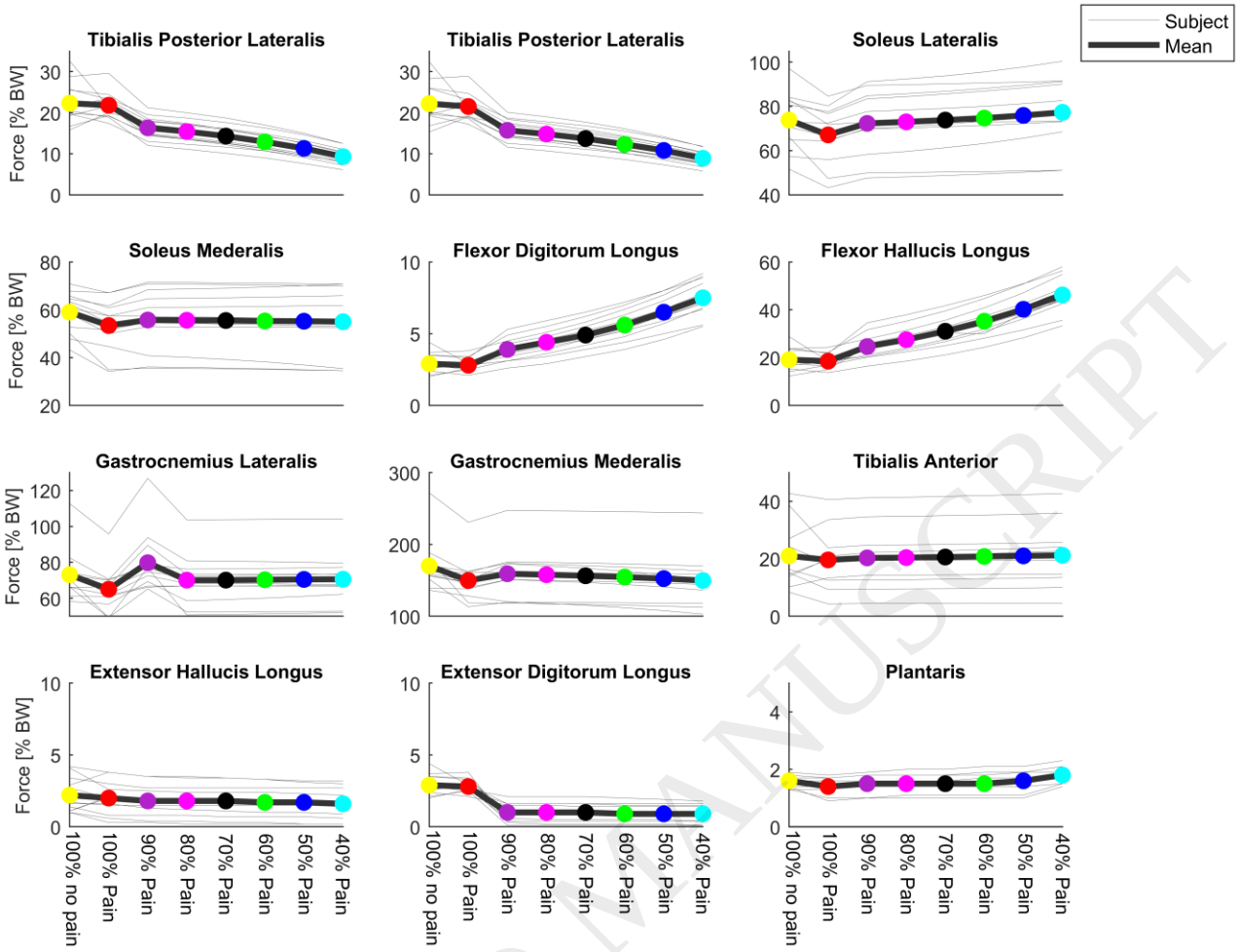

Figure 4 Peak muscle force for each individual subject (thin gray line) and mean muscle force peak for the no-pain walk with 100% default TP strength and the pain walk with 100, 90, 80, 70, 60, 50 and 40% of default TP muscle isometric strength.

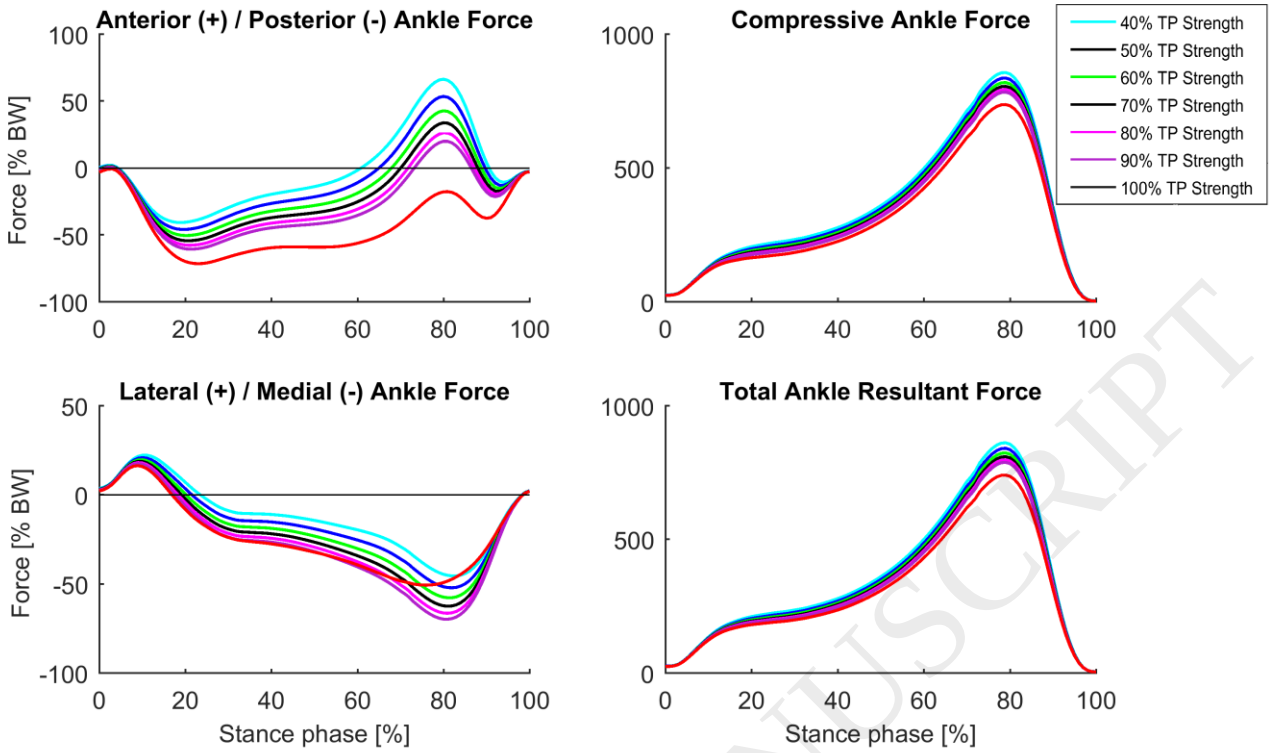

Figure 5: Mean ankle joint forces for 100 to 40% of default TP muscle strength.