Title Page

Increased trunk flexion may underlie elevated knee flexor activity in people with knee osteoarthritis

Stephen J. Preece[1] and Wael Alghamdi[1,2]

[1]Centre for Health Sciences Research, University of Salford, Salford, Manchester, M6 6PU, UK

[2]Al Baha University, Al Baha, Saudi Arabia

Stephen Preece: s.precce@salford.ac.uk ***corresponding author***

Wael Alghamdi: waelalghamdi@bu.edu.sa

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Abstract

Background: Previous research has demonstrated elevated activation of the knee flexor muscles in people with knee osteoarthritis. People with this condition have also been observed to walk with increased trunk flexion; this may alter biomechanical loading patterns and change muscle activation profiles. Therefore, the aim of this study was to understand the biomechanical effect of increasing trunk flexion during walking.

Methods: Kinetic and EMG data were collected from a sample of 20 people with knee osteoarthritis and a sample of 20 healthy matched controls during normal walking. Using a biofeedback protocol, participants were subsequently instructed to walk with a 5° increase in trunk flexion. Sagittal moments, muscle activations and co-contractions were then compared across a window in early stance with a two-way ANOVA test.

Results: When trunk flexion was increased, there was a corresponding increase in activity of the medial and lateral hamstrings and gastrocnemius muscles as well as a rise in medial co-contraction. This effect was consistent across the two groups. The most pronounced effect was observed for semitendinosus, which showed a dramatic change in activation profile in the healthy group and a 127% increase in activation during early stance.

Conclusions: This is the first study to demonstrate that increased trunk flexion in people with knee osteoarthritis may explain, to some degree, the elevated knee flexor activity and medial co-contraction which is associated with this disease. These findings motivate further work to understand the therapeutic potential of interventions designed to improve postural alignment.

Key words

Knee osteoarthritis; co-contraction; trunk flexion; biofeedback; walking; biomechanics

1 Introduction

Knee osteoarthritis (OA) is a chronic long-term condition which is associated with pain and impaired mobility [1] and affects a large proportion of the global population [2]. This disease is characterised by changes in the articular cartilage and surrounding tissues [3]. While the factors underlying cartilage degeneration are complex, involving biological, mechanical and structural pathways [4], elevated mechanical loading is known to play a role in disease progression [5]. Given this link, there has been a large body of research which has focused on surrogate markers of mechanical loading in knee OA, such as the knee adduction moment [6] and measures of muscular co-contraction [7].

Research has consistently shown that people with knee OA exhibit elevated activity in the hamstrings [8, 9], quadriceps [10, 11] and gastrocnemius [12, 13] muscles during walking. These altered patterns of muscle coordination are characterised by both prolonged [12] and increased muscle activation [8], resulting in elevated co-contraction [14]. Experimental and modelling studies have demonstrated the potentially damaging effects of these muscle patterns, showing that increased co-contraction will increase compressive load on the joint [15] and accelerate cartilage loss [16] . Importantly, longer-term follow up studies have identified that patients who exhibit elevated co-contraction at baseline, are more likely to opt for a knee replacement at five-year follow up [17]. These findings indicate that co-contraction could be an intervention target. However, if therapeutic interventions are to be developed to reduce co-contraction, it is important to understand the biomechanical mechanisms which may underlie increased muscle activity in people with knee OA.

Two potential biomechanical mechanisms for co-contraction have been suggested. The first is that higher muscle activity on the lateral side of the knee unloads the medial compartment [18], which is most commonly affected by OA. However, modelling studies have disputed this theory, showing that a lateral

activation strategy may actually increase, not decrease, the medial contact load [15]. The second mechanism proposed to explain co-contraction is that it functions as a mechanism to increase knee stability in order to compensate for ligament laxity [19]. Evidence to support this idea comes from research which has demonstrated that people with knee OA exhibit elevated muscular responses to a sudden perturbation [20]. However, it is unclear whether findings from perturbation studies can be used to infer that increased knee flexor activity, observed during walking, functions primarily to stabilise the joint. Instead, other mechanisms may underlie increased knee muscle activity which result from biomechanical changes proximal to the knee.

A large proportion of the body's mass is contained within the head, arms and trunk segment [21]. Therefore, small changes in trunk flexion have the potential to change biomechanical loads at lower extremity joints [22] and impact on lower limb muscle patterns [23]. In line with this idea, we have observed increased activity of the lateral knee flexor muscles in healthy young people who habitually walk with elevated trunk flexion [23]. In another study, we demonstrated that when healthy young people are instructed to increase trunk flexion by 5° [24], there are pronounced increases in both medial and lateral gastrocnemius and hamstring muscles. Given that people with knee OA have been shown to walk with an increased flexion of the trunk [25], it is possible that the elevated knee flexor activity, observed in people with knee OA, is related to an alteration in sagittal trunk inclination. Therefore, the aim of this study was to quantify the effect of increasing trunk flexion in older healthy people and individuals with knee OA.

2 Materials and methods

2.1 Study participants

We recruited 20 participants with knee osteoarthritis (OA) along with 20 matched healthy control participants. Participants in the group with knee OA were included if they were over the age of 40, satisfied ACR criteria [26], had a radiological diagnosis of knee OA and had experienced knee pain for at least 6 months prior to testing. Healthy participants were included if they had not experienced lower limb pain or

back pain within the last six months. Exclusions for both groups included any neurological disease, cardiovascular disorder, previous surgery to the lower limb (excluding exploratory arthroscopy) or an inability to walk 100m unaided. This latter criterion was included to ensure that all participants had a reasonable level of physical function. Patients were recruited through a range of avenues, including community advert, GP invitation letter, physiotherapy outpatient clinics and through a local citizen scientist website for the recruitment of research volunteers. Ethical approval was obtained from a UK NHS ethics committee (REF 18/NW/0030); all subjects gave informed consent to participate, and all procedures were performed in accordance with the declaration of Helsinki.

2.2 Experimental data collection

Kinematic, kinetic and EMG data were collected during a normal walking (baseline) condition and under a condition in which participants were instructed to increase trunk flexion by 5°. For this increased trunk flexion condition, we used a biofeedback protocol, explained below. Kinematic data were collected using an Oqus camera system (Qualisys, Sweden) (100Hz) with two AMTI force plates (1500Hz) embedded in the walkway. Reflective markers, attached to the skin, were used to track motions of the pelvis and trunk, along with the thigh, shank and foot of one lower limb. This limb corresponded to the symptomatic limb in the people with knee OA and was selected at random in the healthy individuals.

To track the thorax (trunk), we followed the protocol suggested by Armand *et al.* [27], defining this segment with markers placed on the greater trochanters and acromions. The trunk segment was tracked using markers on the jugular notch and on the second and eighth thoracic vertebrae. Preliminary testing, on five participants, showed a standard error of measurement of the trunk angle of 0.9° from test-retest data collected during two test sessions, separated by one week. The pelvic segment was defined using markers placed over the right and left anterior superior iliac spines and the right and left posterior superior iliac spines; these were tracked with a rigid cluster of 3 markers positioned over the sacrum. Two rigid clusters of 4 markers were also used to track the motions of the thigh and shank; a system of 4 markers, placed over anatomical landmarks, were used to track motion of the foot. Ankle and knee joint centres were calculated as midpoints between the malleoli and femoral epicondyles respectively. The hip joint

centre was calculated using the regression model of Bell et al. [28] based on the anterior and posterior superior iliac spine markers.

Surface electromyography (EMG) data were collected from the same limb selected for the kinematic/kinetic data. These data were collected using a Noraxon DTS system, sampling at 1500 Hz, from six muscles: vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), semitendinosus (ST), medial gastrocnemius (MG) and lateral gastrocnemius (LG). Electrodes were placed according to SENIAM guidelines [29] and skin preparation was performed using abrasive gel and an alcohol wipe.

All participants were tested under a baseline normal walking condition. For these trials, participants walked barefoot at a self-selected speed along a 6-metre walkway. A minimum of five successful walking trials were recorded for which walking speed (measured using optical timing gates) was consistent (within a 5% tolerance). Data from these normal walking trials were then processed to obtain a kinematic trajectory for trunk flexion angle relative to the laboratory. This processing involved low pass filtering of raw marker and force data at 12Hz and 25Hz respectively and the use of a six degree of freedom model, implemented using the Visual 3D software (C-Motion, Rockville, Maryland), to calculate the kinematic trajectory [23]. Gait events were calculated by applying a 20N threshold to the vertical ground reaction force data and used to time normalise the trunk flexion data to a full gait cycle. An ensemble average for trunk flexion was calculated for all walking trials and the mean (across the gait cycle) used as that participant's trunk flexion angle during normal walking (NW). This was taken as the baseline condition.

All participants were then instructed to increase their trunk flexion angle by 5° during walking (NW+5°). Previous research has shown a smaller difference of approximately 3° in trunk flexion between healthy participants and people with knee OA [25]. However, through pilot testing, a change of 5° was found to be smallest increase that could be consistently adopted by our participants. A two-stage process was used to instruct participants to increase trunk flexion, which focused first on standing and then on walking. Throughout this process, participants were instructed to move their hip backwards in order to change upper body inclination. Pilot testing showed this instruction to be the most effective method of changing trunk flexion, whilst minimising associated changes in knee angle or spinal alignment in standing.

The initial phase, which focused on standing, was implemented using a real-time biofeedback programme, deployed in MATLAB (The MathWorks), which visualised trunk flexion on a screen, indicating the target angles.

Once participants could repeatably reproduce the target angle in standing without the need for biofeedback, walking trials at the increased trunk flexion condition were carried out. To achieve the target trunk angle, participants were provided with verbal feedback after each trial so that they could make an appropriate adjustment to trunk angle during the subsequent trial. To facilitate this approach, trunk angle was calculated using the Visual 3D software after each trial and compared to the target trunk angle. A trial was considered successful if it was within 5% of the baseline walking speed and if the mean trunk flexion angle (across the gait cycle) was within 2° of the target trunk angle.

Reference data from a maximum voluntary isometric contraction (MVIC) were collected for each muscle group following the walking trials. These data were collected using the protocol suggested by Rutherford *et al.* [30], described in an earlier publication [23]. Three separate tests, of five seconds duration, were recorded for each of the three muscle groups, with a 60 second rest between contractions. To process the MVIC data, a high pass filter (20Hz) was applied, the signal rectified and a linear envelop (6Hz) created [10]. A 0.1s moving window algorithm [10] was applied to the linear envelope and a maximum value was identified for each trial. The dynamic EMG was processed in a similar way, with high pass filtering (20Hz), followed by rectification and creation of a linear envelope (6Hz) [10]. Dynamic EMG data were time normalised to stance phase and an ensemble average created for each muscle for both the baseline condition and for the trunk flexed condition. These data were then normalised by the MVIC reference value, which was selected as the maximum from the three MVIC tests.

To understand the effect of trunk flexion on co-contraction, we derived four co-contraction activation profiles. The first two profiles were obtained by summing medial/lateral hamstring and quadriceps activity (ST-VM & BF-VL) and the second two profiles obtained by summing medial/lateral gastrocnemius and quadriceps activity (MG-VM & LG-VL). Although previous researchers have used a specific co-contraction ratio [14], we chose to sum knee flexor and knee extensor activity. This is because

165 modelling studies [7] have shown that this method of quantifying co-activation is more closely related to
166 joint contact forces than the co-contraction ratio. Following EMG processing, kinematic trajectories for the
167 hip, knee and ankle, along with lower limb moments, were derived with the Visual 3D software using the
168 modelling approached reported in a previous paper [23]. All moment data were normalised by the
169 participant's body mass.

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171 **2.3 Outcome measures and statistical analysis**

172 In order to define specific outcome measures for each of the kinetic, muscle activation and co-
173 contraction signals, we calculated an average across a specific window of the gait cycle. Modelling studies
174 of knee contact loads [15] have identified a point of peak load at approximately 13% of the gait cycle,
175 equivalent to 20% of stance phase. We therefore chose to focus on a window of 15-25% stance phase for
176 kinematic/kinetic data. This was adjusted backwards by 5% of stance (approximately 30ms) for EMG
177 signals, in order to account for electromechanical delay. Derivation of the specific outcomes was performed
178 in Matlab. A two-way ANOVA test (group x trunk flexion) was applied to explore the effect of increasing
179 trunk flexion on muscle activation parameters, sagittal moments and to identify any possible interactions,
180 i.e. whether the effect of increasing trunk flexion differed between the two groups. All data were found to
181 be normally distributed using the Kolmogorov-Smirnov test; therefore, it was not necessary to use any non-
182 parametric tests. All statistical analyses were performed in MATLAB. To guard against type 1 error, a critical
183 $\alpha = 0.01$ was selected.

184 # 3 Results

185 All 20 people with knee OA (7 male) completed the testing protocol. Of this group, two had a KL
186 grade 1, six had a grade 2, nine had a grade 3 and three had a grade 4. This group had a mean (SD) age of
187 56 (9) years old, mass 81(14) kg, height 1.70 (0.07) m and BMI 28.7 (4.9) kg/m^2. The 20 healthy people (7
188 male) also completed the full testing protocol. The mean (SD) age of this group was 57 (9) years, mass 80

(11) kg, height 1.70 (0.06) m and BMI 27.4 (3.9) kg/m^2. Comparison of demographic characteristics showed minimal differences between the healthy group and the group with knee OA.

The individuals with knee OA walked with 2.8° more trunk flexion in the baseline condition than the healthy participants (Table 1). Following the biofeedback protocol, trunk inclination increased by an average of 5.9° and 5.8° in the healthy and OA groups respectively (Table 1). This change was associated with a significant increase in both the hip moment and the ankle moment over the window of interest, but a non-significant decrease in the knee moment (Table 1). Specifically, when trunk flexion was increased in the healthy group, the hip moment increased by 86% and the ankle moment increased by 250% (Table 1 & Figure 1). Interestingly, the ankle moment profile was very similar between the baseline OA condition and the healthy NW+5° condition during midstance (Figure 1c). No group x trunk flexion interactions were observed for any of the moment parameters.

The activation profiles of the two quadriceps muscles were relatively unaffected by increasing trunk flexion (Figure 2 & Table 1). However, both hamstring and gastrocnemius muscles increased significantly with no group x trunk flexion interactions (Table 1). The largest increases, over the window of interest, were observed in the two hamstring muscles with a 127% increase in semitendinosus and a 56% increase in biceps femoris for the healthy group (Table 1, Figure 3). Interestingly, when trunk flexion was increased in the healthy group, the activation profile of semitendinosus became similar in shape to the baseline OA profile (Figure 3a), with a peak which occurred at approximately 10% of stance. Although there were less pronounced changes in the activation profile of biceps femoris, the healthy profile did change to resemble the baseline OA profile more closely as trunk flexion was increased (Figure 3b). Increases in the medial and lateral gastrocnemius were smaller than those observed for the hamstrings, with changes of 30% and 35% respectively for the healthy group over the window of interest (Table 1 & Figure 3c,d).

Co-contraction between the semitendinosus and vastus medialis muscles increased significantly as trunk flexion was increased (Table 1). Interestingly, when the healthy group increased trunk flexion, this co-contraction profile became almost identical to the baseline OA profile (Figure 4a), with a 31% increase across the window of interest. There were minimal changes in the other co-contraction profiles for the

healthy group, with only small increases in the group with OA (Figure 4). However, despite these subtle between-groups differences, there were no group x trunk flexion interactions (Table 1) for any of the co-contraction signals.

4 Discussion

This study was performed to understand the biomechanical effect of increasing trunk flexion and therefore gain insight into whether alterations in muscle activation, associated with knee OA, might be related to trunk flexion. When trunk flexion was increased, there were pronounced increases in hip and ankle moments, knee flexor muscle activation and ST-VM co-contraction. Importantly, there were no group x trunk flexion interactions, demonstrating that the effect of increasing trunk flexion was consistent across the two groups. Visual inspection of the muscle activation and co-contraction profiles demonstrated that, when trunk flexion was increased in the healthy group, the profiles changed to become similar to the baseline OA condition. These findings indicate that trunk flexion is likely to underlie, to some degree, previous observations of elevated knee flexor activation [8, 9] and medial co-contraction [31] in people with knee OA.

In a previous study we demonstrated that people with knee OA walk with 2.6° more trunk flexion that matched healthy controls [25]. While the findings of this study are consistent with our previous observation, other research has not identified clear differences in sagittal trunk inclination between healthy and OA groups [32]. However, it is possible that these inconsistent findings are the result of the kinematic approach used to model the trunk. Cadaver studies demonstrate that approximately 65% of the body's mass is concentrated in the head, arms and trunk segment [21]. Therefore, very small changes in upper body inclination could have a large effect on mechanical loads at the lower limb, altering muscle activation profiles. It is therefore critical that future research employs advanced kinematic modelling techniques to accurately quantify centre of mass trajectory in order to understand how this differs between healthy people and those with OA and how it may impact on muscle activation patterns. Research should also investigate whether alterations in postural alignment in people with knee OA, which have been observed in standing [33-36], are maintained during functional tasks, such as walking.

With our laboratory protocol, all participants were provided with biofeedback to enable them to increase trunk flexion by 5°. However, despite this tightly controlled protocol, the mean increase in trunk flexion was 5.9° in the healthy group which was more than twice the baseline difference of 2.6° between the healthy group and the people with knee OA. The findings of this present study are consistent with our previous study on younger people, in which we demonstrated a linear increase in muscle activation as trunk flexion was increased [24]. Assuming the data we present here are also linear, the increase in muscle activation, which may be attributed to increased trunk flexion in the knee OA group, is likely to be approximately 50% of the change we report between the baseline and NW+5° condition for the healthy group. Interestingly, our data showed that when trunk flexion was increased in the healthy group, semitendinosus increased to a level above that of the knee OA group in normal walking (Figure 3a). However, the magnitude of change was lower in the biceps femoris (Figure 3b). Given these finding, increased trunk flexion is unlikely to underlie the full spectrum of differences in muscle activation between healthy people and those with knee OA. Nevertheless, it is likely to play an important role in medial co-contraction which has been linked to increased rate of cartilage loss in people with knee OA [16] .

If increased trunk flexion does underlie, to some degree, elevated muscle activation in people with knee OA, then it is important to develop clinical interventions which specifically target trunk flexion. While most exercise-based approaches for knee OA tend to focus on strengthening of the knee flexor and extensor muscles [37], we have observed improvements in pain following neuromuscular retraining programmes which incorporate a postural component [31, 38]. In one of these studies, we quantified changes in muscle activation, demonstrating a group-level reduction in medial hamstrings-quadriceps co-ccontraction [31]. Importantly, greater improvements in pain were observed in those who exhibited larger reductions in medial co-contraction. These previous results, along with the clear link between trunk flexion and hamstring activation identified in this paper, motivate further research into the potential therapeutic effect of postural training in people with knee OA.

There are a number of limitations to this study which need to be acknowledged. Firstly, we used a maximal voluntary contraction to normalise the EMG data, which can be influenced by inter-subject

variation in motivation during reference contractions. Secondly, we used a single rigid segment to represent the trunk (thorax) in order to define a single outcome measure capturing sagittal trunk inclination. Given the multiple articulations in the spine, a more complex model would be required to accurately represent subtle inter-subject differences in spinal alignment. However, this would have made it difficult to independently manipulate a single variable. Thirdly, we acknowledge that our design does not provide a definitive measure of the proportion of the variance in muscle activation which is explained by differences in trunk flexion. However, given the difficulty of precisely quantifying muscle activation and trunk inclination, we suggest that our approach of independently manipulating trunk flexion is appropriate. This is because it minimises the impact of inter-subject variability on biomechanical outcomes. We suggest that such variability could lead to uncertainty in a correlational design.

5 Conclusion

In summary, our data support the idea that increased trunk flexion may underlie, to some degree, the increased knee flexor activation and medial co-contraction observed in people with knee OA. While such inter-subject differences in trunk flexion are likely to interact with other factors, these findings support the need for future research to develop and evaluate interventions which can improve postural alignment in people with knee OA. It is likely that such interventions will reduce knee flexor activation, reducing compressive load on the joint and may lead to improved clinical outcomes.

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Conflict of interest

None

References

[1] Woolf AD, Pfleger B. Burden of major musculoskeletal conditions. Bulletin of the World Health Organization 2003;81(9):646-56. <Go to ISI>://WOS:000185876300006

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2572542/pdf/14710506.pdf.

[2] Cross M, Smith E, Hoy D, Nolte S, Ackerman I, Fransen M, et al. The global burden of hip and knee osteoarthritis: estimates from the global burden of disease 2010 study. Ann Rheum Dis 2014;73(7):1323-30. https://doi.org/10.1136/annrheumdis-2013-204763.

[3] Litwic A, Edwards MH, Dennison EM, Cooper C. Epidemiology and burden of osteoarthritis. British Medical Bulletin 2013;105(1):185-99. https://doi.org/10.1093/bmb/lds038.

[4] Andriacchi TP, Mündermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann Biomed Eng 2004;32(3):447-57. https://doi.org/10.1023/b:abme.0000017541.82498.37.

[5] Andriacchi TP, Mundermann A. The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. Curr Opin Rheumatol 2006;18(5):514-8. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=16896293

http://graphics.tx.ovid.com/ovftpdfs/FPDDNCIBGFHLAO00/fs046/ovft/live/gv023/00002281/00002281-200609000-00015.pdf.

[6] Amin S, Luepongsak N, McGibbon CA, LaValley MP, Krebs DE, Felson DT. Knee adduction moment and development of chronic knee pain in elders. Arthritis Rheum 2004;51(3):371-6. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=15188321

http://onlinelibrary.wiley.com/store/10.1002/art.20396/asset/20396_ftp.pdf?v=1&t=ij5h8tz9&s=5c3fc7d26f2c4762c5140513af1e5a68d2730d6a.

[7] Winby CR, Gerus P, Kirk TB, Lloyd DG. Correlation between EMG-based co-activation measures and medial and lateral compartment loads of the knee during gait. Clinical Biomechanics 2013;28(9):1014-9. https://doi.org/http://dx.doi.org/10.1016/j.clinbiomech.2013.09.006.

[8] Hortobagyi T, Westerkamp L, Beam S, Moody J, Garry J, Holbert D, et al. Altered hamstring-quadriceps muscle balance in patients with knee osteoarthritis. Clin Biomech (Bristol, Avon) 2005;20(1):97-104. https://doi.org/10.1016/j.clinbiomech.2004.08.004.

[9] Rutherford, Hubley-Kozey CL, Stanish WD. Changes in knee joint muscle activation patterns during walking associated with increased structural severity in knee osteoarthritis. Journal of Electromyography and Kinesiology 2013;23(3):704-11. https://doi.org/10.1016/j.jelekin.2013.01.003.

[10] Hubley-Kozey CL, Deluzio KJ, Landry SC, McNutt JS, Stanish WD. Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. J Electromyogr Kinesiol 2006;16(4):365-78. https://doi.org/10.1016/j.jelekin.2005.07.014.

[11] Rutherford DJ, Hubley-Kozey CL, Stanish WD, Dunbar MJ. Neuromuscular alterations exist with knee osteoarthritis presence and severity despite walking velocity similarities. Clin Biomech (Bristol, Avon) 2011;26(4):377-83. https://doi.org/10.1016/j.clinbiomech.2010.11.018.

[12] Childs JD, Sparto PJ, Fitzgerald GK, Bizzini M, Irrgang JJ. Alterations in lower extremity movement and muscle activation patterns in individuals with knee osteoarthritis. Clinical Biomechanics 2004;19(1):44-9. https://doi.org/10.1016/j.clinbiomech.2003.08.007.

[13] Astephen JL, Deluzio KJ, Caldwell GE, Dunbar MJ, Hubley-Kozey CL. Gait and neuromuscular pattern changes are associated with differences in knee osteoarthritis severity levels. J Biomech 2008;41(4):868-76. https://doi.org/10.1016/j.jbiomech.2007.10.016.

[14] Heiden TL, Lloyd DG, Ackland TR. Knee joint kinematics, kinetics and muscle co-contraction in knee osteoarthritis patient gait. Clinical Biomechanics 2009;24(10):833-41. https://doi.org/10.1016/j.clinbiomech.2009.08.005.

[15] Brandon SCE, Miller RH, Thelen DG, Deluzio KJ. Selective lateral muscle activation in moderate medial knee osteoarthritis subjects does not unload medial knee condyle. Journal of Biomechanics 2014;47(6):1409-15. https://doi.org/10.1016/j.jbiomech.2014.01.038.

[16] Hodges PW, van den Hoorn W, Wrigley TV, Hinman RS, Bowles K-A, Cicuttini F, et al. Increased duration of co-contraction of medial knee muscles is associated with greater progression of knee osteoarthritis. Manual Therapy 2016;21:151-8. https://doi.org/http://dx.doi.org/10.1016/j.math.2015.07.004.

[17] Hatfield GL, Costello KE, Astephen Wilson JL, Stanish WD, Hubley-Kozey CL. Baseline gait muscle activation patterns differ for osteoarthritis patients who undergo total knee arthroplasty 5-8 years later from those who do not. Arthritis Care Res (doi:101002/acr24143) 2020. https://doi.org/10.1002/acr.24143.

[18] Schipplein OD, Andriacchi TP. Interaction between active and passive knee stabilizers during level walking. J Orthop Res 1991;9(1):113-9.

[19] Lewek MD, Rudolph KS, Snyder-Mackler L. Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis. Osteoarthritis and Cartilage 2004;12(9):745-51. https://doi.org/10.1016/j.joca.2004.05.005.

[20] Lewek MD, Ramsey DK, Snyder-Mackler L, Rudolph KS. Knee stabilization in patients with medial compartment knee osteoarthritis. Arthritis and Rheumatism 2005;52(9):2845-53. https://doi.org/10.1002/art.21237.

[21] Dempster WT. Space requirements of the seated operator, geometrical, kinematic, and mechanical aspects of the body with special reference to the limbs. Michigan State Univ East Lansing; 1955.

[22] Leteneur S, Gillet C, Sadeghi H, Allard P, Barbier F. Effect of trunk inclination on lower limb joint and lumbar moments in able men during the stance phase of gait. Clinical Biomechanics 2009;24(2):190-5. https://doi.org/10.1016/j.clinbiomech.2008.10.005.

[23] Alghamdi W, Preece SJ. How does normal variability in trunk flexion affect lower limb muscle activity during walking? Human Movement Science 2020;72:102630. https://doi.org/https://doi.org/10.1016/j.humov.2020.102630.

[24] Preece SJ, Alghamdi W. The effect of increasing trunk flexion during normal walking. Gait & Posture 2021;83:250-5. https://doi.org/https://doi.org/10.1016/j.gaitpost.2020.10.021.

[25] Preece SJ, Algarni AS, Jones RK. Trunk flexion during walking in people with knee osteoarthritis. Gait Posture 2019;72:202-5. https://doi.org/10.1016/j.gaitpost.2019.06.012.

[26] Altman R, Alarcon G, Appelroth D. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the knee. Arthritis Rheum 1986;29:1039-49. http://onlinelibrary.wiley.com/doi/10.1002/art.1780290816/abstract.

[27] Armand S, Sangeux M, Baker R. Optimal markers' placement on the thorax for clinical gait analysis. Gait & Posture 2014;39(1):147-53. http://usir.salford.ac.uk/31653/.

[28] Bell AL, Brand RA, Pedersen DR. Prediction of hip-joint center location from external landmarks. Human Movement Science 1989;8(1):3-16. https://doi.org/10.1016/0167-9457(89)90020-1.

[29] Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. Journal of Electromyography and Kinesiology 2000;10(5):361-74. https://doi.org/10.1016/s1050-6411(00)00027-4.

[30] Rutherford DJ, Hubley-Kozey CL, Stanish WD. Maximal voluntary isometric contraction exercises: a methodological investigation in moderate knee osteoarthritis. J Electromyogr Kinesiol 2011;21(1):154-60. https://doi.org/10.1016/j.jelekin.2010.09.004.

[31] Preece SJ, Jones RK, Brown CA, Cacciatore TW, Jones AK. Reductions in co-contraction following neuromuscular re-education in people with knee osteoarthritis. BMC Musculoskelet Disord 2016;17(1):372. https://doi.org/10.1186/s12891-016-1209-2.

[32] Iijima H, Shimoura K, Ono T, Aoyama T, Takahashi M. Proximal gait adaptations in individuals with knee osteoarthritis: A systematic review and meta-analysis. Journal of Biomechanics 2019;87:127-41. https://doi.org/https://doi.org/10.1016/j.jbiomech.2019.02.027.

[33] Turcot K, Sagawa Jr Y, Hoffmeyer P, Suvà D, Armand S. Multi-joint postural behavior in patients with knee osteoarthritis. The Knee 2015;22(6):517-21. https://doi.org/http://dx.doi.org/10.1016/j.knee.2014.09.001.

[34] Yasuda T, Togawa D, Hasegawa T, Yamato Y, Kobayashi S, Yoshida G, et al. Relationship between Knee Osteoarthritis and Spinopelvic Sagittal Alignment in Volunteers over 50 Years of Age. Asian Spine Journal 2020;14(4):495-501. https://doi.org/10.31616/asj.2018.0266.

[35] Wang WJ, Liu F, Zhu YW, Sun MH, Qiu Y, Weng WJ. Sagittal alignment of the spine-pelvis-lower extremity axis in patients with severe knee osteoarthritis: A radiographic study. Bone Joint Res 2016;5(5):198-205. https://doi.org/10.1302/2046-3758.55.2000538.

[36] Tauchi R, Imagama S, Muramoto A, Tsuboi M, Ishiguro N, Hasegawa Y. Influence of spinal imbalance on knee osteoarthritis in community-living elderly adults. Nagoya Journal of Medical Science 2015;77(3):329-37. <Go to ISI>://WOS:000365415100001

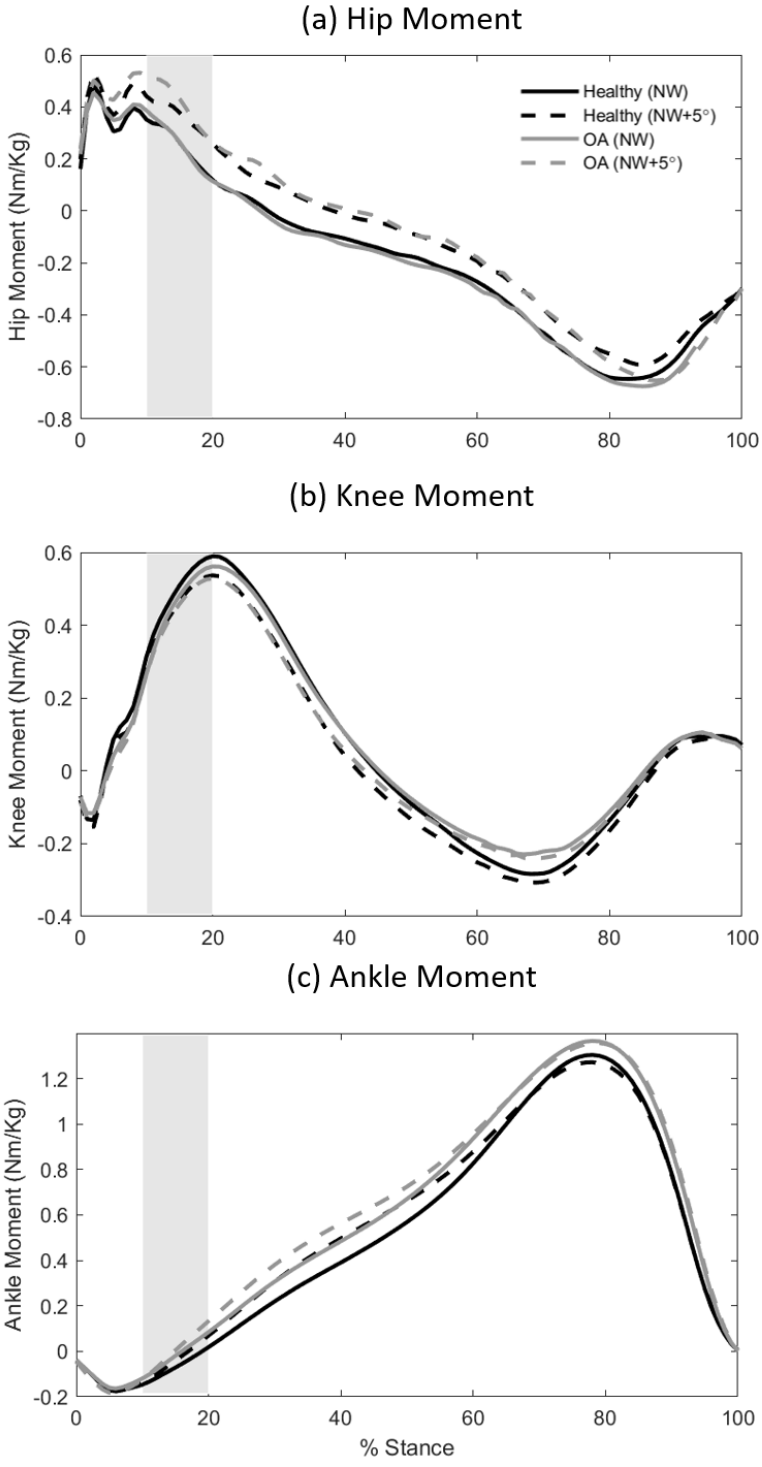
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4574319/pdf/2186-3326-77-3-0329.pdf.

[37] Fransen M, McConnell S, Harmer AR, Van der Esch M, Simic M, Bennell KL. Exercise for osteoarthritis of the knee. Cochrane Database of Systematic Reviews 2015(1). https://doi.org/10.1002/14651858.CD004376.pub3.

[38] Preece SJ, Brookes N, Williams AE, Jones RK, Starbuck C, Jones A, et al. A new integrated behavioural intervention for knee osteoarthritis: development and pilot study. BMC Musculoskeletal Disorders 2021;22(1). https://doi.org/10.1186/s12891-021-04389-0.

Figures

Figure 1: Ensemble average profiles for the hip, knee and ankle moments for the OA and healthy groups in the normal walking (NW) condition and the increased trunk flexion (NW+5°) condition. The shaded regions indicate the sections of the waveforms which were averaged for statistical analysis.



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419 Figure 2: Ensemble average quadriceps activations for the OA and healthy groups in the normal walking
420 (NW) condition and the increased trunk flexion (NW+5°) condition. The shaded regions indicate the
421 sections of the waveforms which were averaged for statistical analysis.

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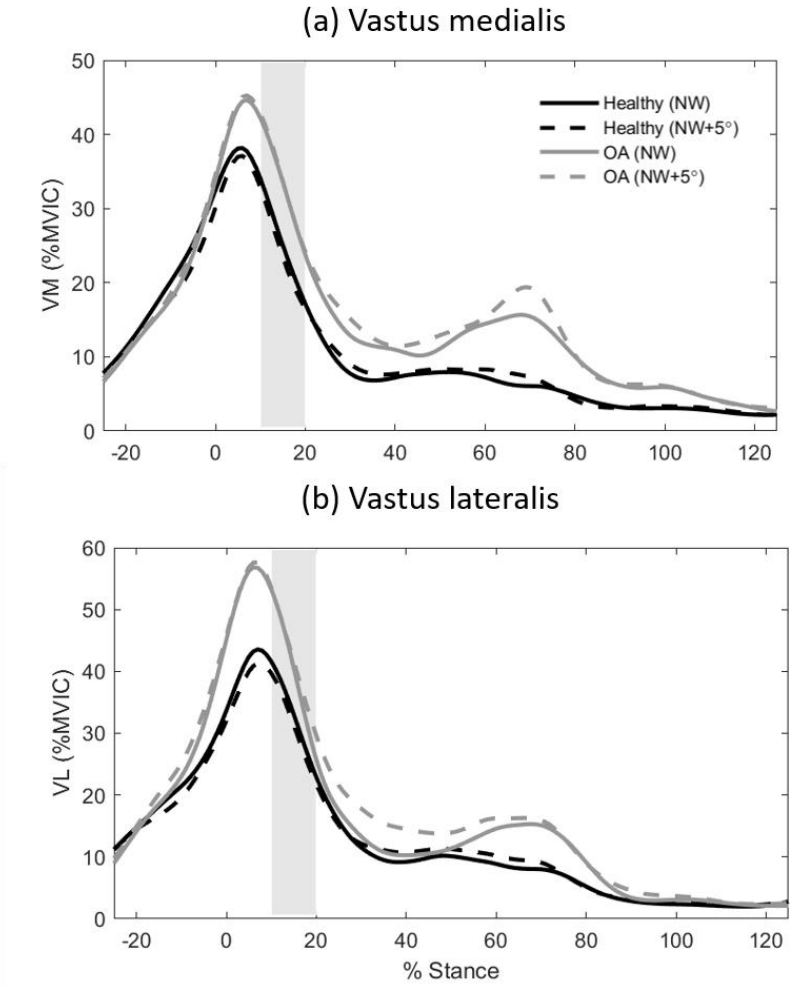

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Figure 3: Ensemble average hamstring and gastrocnemius activations for the OA and healthy groups in the normal walking (NW) condition and the increased trunk flexion (NW+5°) condition. The shaded regions indicate the sections of the waveforms which were averaged for statistical analysis.

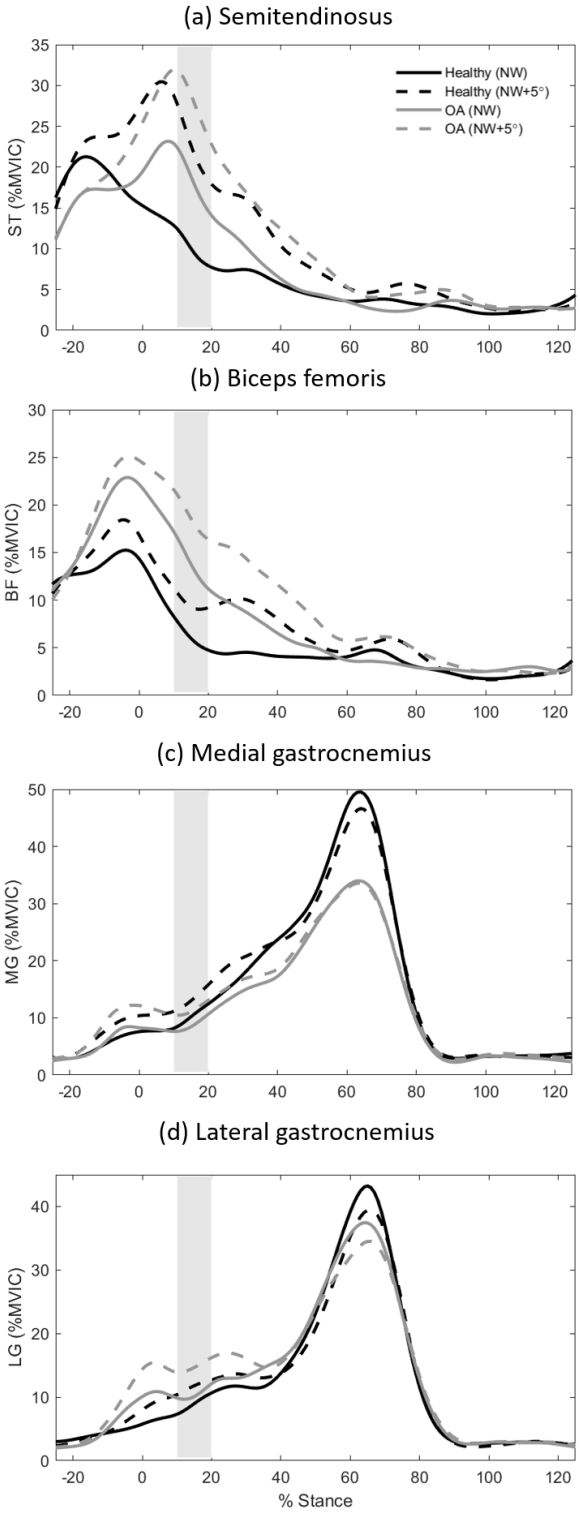
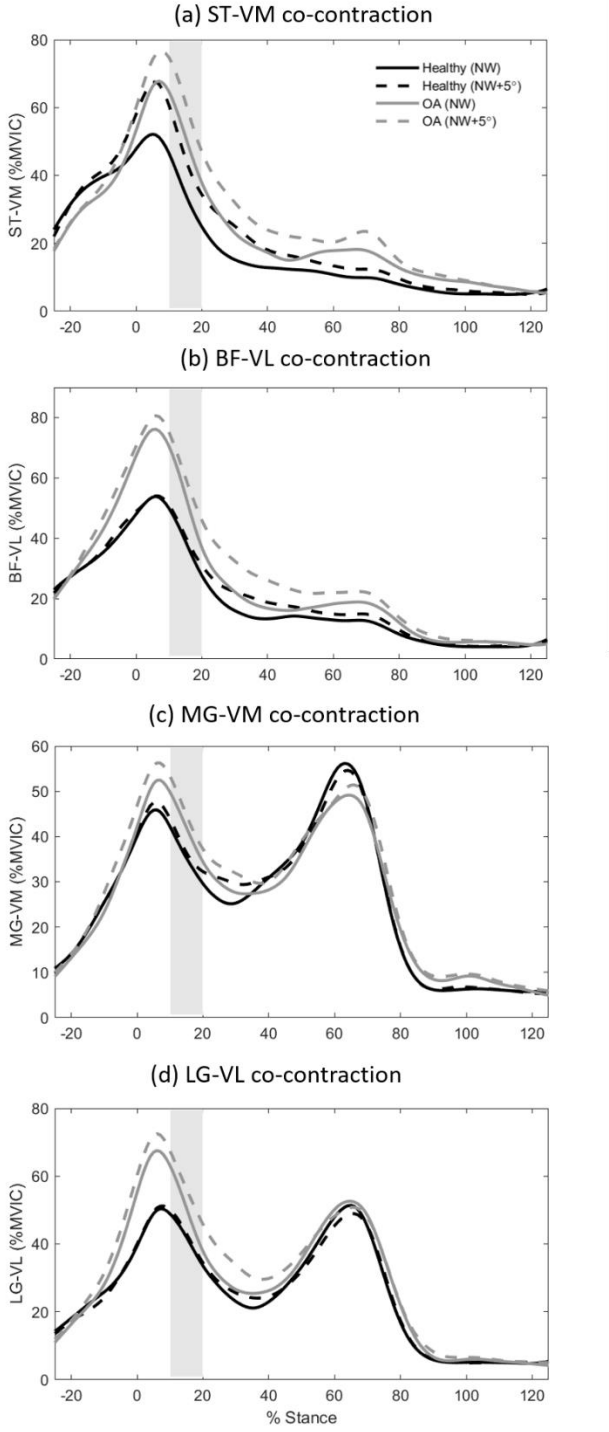


Figure 4: Ensemble average co-contraction signals for OA and healthy groups in the normal walking (NW) condition and the increased trunk flexion (NW+5°) condition. The shaded regions indicate the sections of the waveforms which were averaged for statistical analysis. VM – vastus medialis, MG – medial gastrocnemius, VL – vastus medialis, LG – lateral gastrocnemius, ST – semitendinosus, BF – biceps femoris.



Tables

Tables 1: Mean sagittal moment (Nm/Kg) and muscle activation (%MVIC) parameters for both groups in the normal walking and trunk flexed conditions. P-values have been presented to show the effect of trunk flexion and for the group x trunk flexion interaction. Trunk angle is averaged across the whole gait cycle and moments/muscle activations averaged across a window in early stance. VM – vastus medialis, MG – medial gastrocnemius, VL – vastus medialis, LG – lateral gastrocnemius, ST – semitendinosus, BF – biceps femoris.

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	Healthy		OA		p-Value	
	NW	NW+5°	NW	NW+5°	Effect of flexion	Interaction
Trunk flexion	1.6°	7.5°	4.2°	10.0°	-	-
Hip Moment	0.14	0.26	0.13	0.29	<0.001	0.395
Knee Moment	0.56	0.51	0.54	0.50	0.045	0.672
Ankle Moment	0.02	0.07	0.09	0.13	<0.001	0.852
VM	25.3	24.0	33.3	33.6	0.65	0.838
VL	32.7	31.1	40.1	41.6	0.971	0.391
ST	9.8	22.2	18.1	27.7	<0.001	0.476
BF	6.2	9.7	14.0	18.8	<0.001	0.372
MG	10.2	13.2	8.7	11.4	<0.001	0.838
LG	9.0	11.7	10.5	14.8	<0.001	0.391
ST-VM	35.1	46.1	51.4	61.3	<0.001	0.811
BF-VL	38.9	40.8	54.1	60.4	0.024	0.207
MG-VM	35.5	37.2	42.1	45.0	0.067	0.201
LG-VL	41.7	42.8	50.6	56.4	0.057	0.182

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