Title: Individuals with knee osteoarthritis demonstrate increased passive stiffness of the hip flexor muscles

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Structured abstract

Background: People with knee osteoarthritis stand and walk with increased trunk flexion. This altered postural alignment will increase hamstring activation, elevating mechanical knee loads during walking. Increased hip flexor stiffness may lead to increased trunk flexion. Therefore, this study compared hip flexor stiffness between healthy individuals and individuals with knee osteoarthritis. This study also sought to understand the biomechanical effect of a simple instruction to reduce trunk flexion by 5° during walking.

Methods: Twenty individuals with confirmed knee osteoarthritis and twenty healthy individuals participated. The Thomas test was used to quantity passive stiffness of the hip flexor muscles and 3D motion analysis used to quantify trunk flexion during normal walking. Using a controlled biofeedback protocol, each participant was then instructed to decrease trunk flexion by 5°.

Results: Passive stiffness was larger in the group with knee osteoarthritis (effect size = 1.04). For both groups, there was relatively strong correlation between passive stiffness and trunk flexion in walking (r=0-61-0.72). The instruction to decrease trunk flexion produced only small, non-significant, reductions in hamstring activation during early stance.

Conclusions: This is the first study to demonstrate that individuals with knee osteoarthritis exhibit increased passive stiffness of the hip muscles. This increased stiffness appears to be linked to increased trunk flexion and may therefore underlie the increased hamstring activation which is associated with this disease. As simple postural instruction does not appear to reduce hamstring activity, interventions may be required which can improve postural alignment by reducing passive stiffness of the hip muscles.

Keywords

Knee osteoarthritis, gait, hip flexor, Thomas test, trunk inclination, trunk flexion

Introduction

There is a substantial body of literature demonstrating that individuals with knee osteoarthritis (OA) walk with increased co-contraction of the knee flexor and extensor muscles [1]. This increased co-contraction has been linked with elevated joint loading [2], increased loss of articular cartilage [3] and has been shown to increase the likelihood that patients will opt for a knee replacement at five-year follow up [4]. Given the potentially damaging effect of co-contraction, it is important to understand the underlying biomechanical mechanisms. During functional tasks, such as walking, muscles work to maintain postural support and generate limb motions. It is therefore possible that the increased activity of the knee muscles, previously observed in people with knee OA, is the result of an alteration in postural control.

We have performed a series of studies to understand the potential link between alterations in sagittal plane inclination of the trunk and activity of the knee muscles during walking. These studies demonstrated that individuals with knee OA walk with a subtle increase in trunk flexion [5]. We have also shown that healthy individuals, who habitually walk with increased trunk flexion, exhibit increased activity of the lateral knee flexors [6]. Through two subsequent studies, we demonstrated that instructing healthy individuals to increase their trunk flexion by 5° led to large increases in the activity of both the medial and lateral hamstrings [7, 8]. Critically, when healthy individuals adopted this slight trunk flexion, the profile of the medial hamstrings changed to become similar to the profile observed in individuals with knee OA during normal walking [8]. Given the clear links between postural alignment and muscle activity, further research is required to understand whether training to reduce this increased trunk flexion could lead to a reduction in hamstring activity during walking.

Postural alignment during functional tasks is maintained through a combination of active muscular control and passive stiffness in connective tissue and other musculotendinous structures [9]. While it is possible that proprioceptive training alone may be sufficient to reduce trunk flexion in individuals with knee OA, increased passive stiffness in structures proximal to the knee may prevent individuals from achieving an optimal postural alignment. Interestingly, several studies have demonstrated altered postural alignment during standing in individuals who suffer with this disease [10-12]. This finding is consistent with our observation of increased trunk flexion in walking [5] and may indicate increased levels of muscle stiffness in people with knee OA.

The idea of muscle imbalance is widely accepted with the physiotherapy profession [9] and used in the management of low back pain [13]. First identified by Janda [13], pelvic crossed syndrome is a common clinical presentation in which patients exhibit increased passive stiffness in the hip flexor muscles and increased activity of the erector spinae muscles. This leads to an anterior rotation of the pelvis on the hip and an increase in the lumbar lordosis. An increase in anterior pelvic rotation is likely to affect sagittal plane trunk inclination, increasing trunk flexion. Interestingly, the clinical presentation of pelvic crossed syndrome fits with the findings of research which has examined postural alignment in individuals with knee OA. Specifically, individuals with knee OA have been shown to demonstrate a flexed posture [10], poor lumbo-pelvic alignment [11] and an increase in forward spinal inclination [12, 14].

In a previous study, we showed that acute stretching of the hip flexor muscles led to a within-session reduction in pelvic tilt in healthy individuals [15]. This finding is consistent with the muscle imbalance theory which proposes that the sagittal plane alignment of the pelvis will be determined, to a large degree, by stiffness in the hip flexor muscles [9]. If this is the case, then a simple instruction to decrease trunk flexion during walking may not be sufficient to bring about a reduction in hamstring activity.

Given the potential link between passive stiffness of the hip flexor muscles and postural alignment, this study sought to compare hip flexor stiffness between healthy individuals and those with knee OA. The study also sought to investigate the link between passive stiffness of the hip flexors and trunk flexion during walking. The final objective was to investigate whether Instruction to decrease trunk flexion by 5° leads to a reduction in hamstring activity.

Methods

Individuals with knee OA and a matched healthy control group were recruited into the study. All participants were over the age of 40 and had to be able to walk for 100m unaided. The group with knee OA were required to have a radiological diagnosis, satisfy ACR criteria [16] and to have experienced knee pain for at least 6 months prior to testing. Healthy volunteers were accepted onto the study if they had not experienced lower limb pain or back pain within the last six months and had not been diagnosed with any neurological disease. Participants were recruited through different avenues, including community advert, GP invitation letter and through physiotherapy outpatient clinics. Ethical approval was obtained from a UK NHS ethics committee (REF 18/NW/0030) and all subjects gave informed consent to participate.

In order to quantify passive stiffness of the hip flexor muscles, we used the Thomas test, which has accepted face-validity for use as a measurement tool in research [17]. During this testing, the participant lay in a supine position with the lower gluteal folds maintained over the edge of the examination table. In this position, a pressure biofeedback cuff, positioned under the back, was inflated to 100 mmHg. The participant was then instructed to hold their knees to their chest and then to slowly lower their tested leg over the edge of the examination table until hip extension was prevented by passive tissue stiffness. At the same time, the assessor ensured that the pressure biofeedback indicator did not drop below 60 mmHg. To measure the degree of hip flexion, a digital goniometer was aligned between the greater trochanter and the lateral epicondyle of the knee. More details on this measurement, including repeatability, are reported in an earlier paper [18]. A measurement of passive hip flexion was taken separately on each side and an average calculated for analysis. For this study, a positive Thomas test angle indicated that the hip was flexed in the measurement position.

Following the hip flexor testing, biomechanical data were collected during normal, barefoot walking at a self-selected speed. Kinematic data were collected using an Oqus camera system (Qualisys, Sweden)

(100Hz) with two AMTI force plate (1500Hz) embedded in the walkway. Reflective markers, attached the skin, were used to track motions of the pelvis, trunk and both thighs, as detailed in an earlier publication [6]. To track the thorax (trunk), we followed the protocol suggested by Armand *et al.* [19], 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 0.9° from test-retest data collected during two test sessions, separated by one week. Surface electromyography (EMG) data were collected from the most painful limb in the participants with knee OA and a matched limb in the healthy group. These data were collected using a Noraxon DTS system, sampling at 1500 Hz, from two muscles: biceps femoris and semitendinosus. Electrodes were placed according to SENIAM guidelines [20] and skin preparation was performed using abrasive gel and an alcohol wipe.

Data from the normal walking trials were processed, immediately after measurement, to obtain a kinematic trajectory for trunk flexion angle relative to the laboratory frame. 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. 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 then 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.

Participants were then instructed walk under two other conditions in a random order: an increased trunk flexion condition (NW+5°) and a decreased trunk flexion condition (NW-5°). A two-stage biofeedback approach was used to instruct participants to change trunk flexion by 5°, which focused first on standing and then on walking. For each condition, participants were first instructed to move their hip backwards/forwards without flexing/extending the spine. We selected this instruction to encourage participants to increase trunk flexion by increasing anterior pelvic tilt and to decrease trunk flexion by

decreasing anterior pelvic tilt, without altering spinal alignment. This initial phase, which focused on standing, was implemented using a real-time feedback 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 feedback, walking trials at the increased/decreased trunk flexion condition were carried out. Trunk angle during each walking trial was monitored using the real-time Visual 3D software plugin to calculate trunk angle and verbal feedback provided to enable participants to adjust trunk angle as appropriate. A trial was considered successful if it was within 5% of the baseline walking speed (measured using optical timing gates) 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 then collected for each of the hamstring muscles, using a protocol described earlier [6, 21]. To process the MVIC data, a high pass filter (20Hz) was applied after which each signal was rectified and a linear envelop (6Hz) created. A 0.1s moving window algorithm [22] was then applied to the linear envelope after which 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). Dynamic EMG data were time normalised to stance phase and an ensemble average created for both muscles for the three walking conditions. These data were then normalised by the MVIC reference value which was selected as the maximum from the MVIC testing. Following EMG processing, hip angles and hip moments were derived using the Visual 3D software using the modelling approached reported in a previous paper [6]. Hip moment data were normalised by participant's body mass.

In order to define specific outcome measures for the kinematic, kinetic and muscle activation signals, each signal was averaged across a specific window of the gait cycle. Modelling studies of knee contact loads [2] have identified a point of peak load during initial stance at approximately 13% of the gait cycle, equivalent to 20% of stance phase. We therefore chose to focus on a window of 15-25% stance phase for kinematic/kinetic data. This was adjusted backwards by 5% of stance (approximately 30ms) for EMG

signals, to account for electromechanical delay. Derivation of the specific outcomes was performed in MATLAB.

All data were found to be normally distributed using the Kolmogorov-Smirnov test; therefore, it was not necessary to use any non-parametric tests. Independent t-tests were used to compare hip flexor stiffness and trunk flexion between the healthy individuals and the individuals with knee osteoarthritis. The effect size was quantified using Cohen's D. A Pearson's correlation coefficient was used to quantify the strength of the relationship between trunk flexion during walking and hip flexor stiffness. This was done separately for the two groups. A two-way ANOVA test was then used to understand the effect of changing trunk flexion and to identify any group x trunk flexion interactions. When significant differences were found, post hoc tests with a Bonferroni correction were used to identify pairwise differences between normal walking and the two other trunk flexion conditions. All statistical analyses were performed in SPSS. To guard against type 1 error, a critical a = 0.01 was selected.

Results

A total of 20 healthy people (7 male) were recruited. 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. A group of 20 people with knee OA (7 male) were recruited. Of this group, two had a KL 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 56 (9) years old, mass 81(14) kg, height 1.70 (0.07) m and BMI 28.7 (4.9) kg/m^2. Comparison of demographic characteristics showed minimal differences between the healthy group and the group with knee OA.

Individuals with knee OA demonstrated greater passive stiffness of the hip flexors (Figure 1), with 4.6° more passive hip flexion than that healthy group. This difference was significant ($p=0.002$) with a large effect size of 1.04. Individuals with knee OA also demonstrated increased trunk flexion during walking (Figure 1), with 2.6° more trunk flexion ($p=0.002$) and an effect size of 1.06. The correlation analysis showed a clear link between trunk flexion in walking and passive hip flexion (Figure 2). For the knee OA group, the correlation was $r=0.67$ ($p<0.001$) and for the healthy group, the correlation was $r=0.61$ ($p<0.001$). This

196 indicated, that for each group, participants with elevated levels of passive stiffness tended to walk with
197 increased trunk flexion.

198 FIGURE 1 & 2 HERE

199 All participants were able to complete the walking biofeedback protocol, modifying their trunk
200 flexion angle across the gait cycle. Mean angles for each group/condition are illustrated in Figure 3. When
201 trunk flexion was increased, there was a corresponding increase in hip flexion (Figure 4, Table 1). However,
202 when trunk flexion was decreased, there was only a minimal change in hip angle (Figure 4). Post hoc tests
203 showed that only increasing trunk flexion produced a significant effect (Table 1). In contrast, both
204 increasing and decreasing trunk flexion led to significant changes in the hip extensor moment over the
205 region of interest. Nevertheless, changes were more pronounced when trunk flexion was increased (Table
206 1, Figure 4).

207 FIGURE 3 & 4 HERE

208 When trunk flexion was increased, there was a clear increase in the activity of both hamstring
209 muscles (Table 1, Figure 5). However, decreasing trunk flexion led to only small, non-significant changes in
210 hamstring activation (Table 1). Figure 5 clearly illustrates the similarity between the baseline condition and
211 the decreased trunk flexion condition for the two hamstring muscles. The ANOVA analysis showed no group
212 x trunk flexion interactions for hamstring activation (Table 1).

213 FIGURE 5 HERE

214 Discussion

215 Our data has identified that individuals with knee OA have increased passive stiffness of the hip
216 flexor muscles and that this stiffness is associated with an increase in trunk flexion during walking. These
217 findings support the idea of pelvic muscle imbalance in individuals with knee OA and may explain the
218 altered postural alignment previously observed in this group [10-12]. Despite being instructed to decrease
219 trunk flexion by moving the hip anteriorly, individuals did not demonstrate a meaningful decrease in hip
220 angle during walking (Figure 4). This indicates that the 5° decrease in trunk flexion (Figure 3) was most likely

achieved through changes in spinal alignment, rather than through a posterior rotation of the pelvis on the hip. This finding may suggest that pelvic alignment is determined by passive stiffness of the hip flexor muscles and may be difficult to modify with simple postural instruction.

When instructed to decrease trunk flexion, both healthy individuals and those with knee OA, demonstrated a decrease in the hip extensor moment (Figure 4). However, while significant, the magnitude of this change, over the period of interest, was approximately 50% of that which resulted from instruction to increase trunk flexion. This difference indicates that decreasing trunk flexion resulted in a smaller anterior-posterior shift in the centre of mass relative to the hip joint centre. This smaller change in relative centre of mass position is most likely the result of a relatively small posterior rotation of the pelvis on the hip, as explained above. These data suggest that simple postural instructions to modify trunk flexion may only lead to modest reductions in the hip extensor moment during early stance.

The relatively small increase of 5° in trunk flexion led to a dramatic change in the activation of the hamstring muscles, particularly semitendinosus (Figure 5, Table 1). This change reflects the altered postural demands placed on the hamstring when postural alignment is compromised. It is useful to consider these changes withing the framework of postural tone. This is defined as “tonic (sustained) activation of muscles in order to provide specific postural attitude and generate force against the ground to keep the limbs extended” [23]. While small increases in trunk flexion appear to increase postural tone in the hamstring muscles, decreasing trunk flexion did not appear to reduce postural tone to the same degree. We did not give participants instructions to posteriorly tilt the pelvis because this may have triggered increased hip extensor activity and the aim of the study was to provide instruction to reduce hamstring activity. Nevertheless, it is possible that, in attempting to decrease trunk flexion, individuals may have increased activity in the hamstrings in order to overcome passive stiffness of the hip flexor muscles. This may explain why, despite an appreciable reduction in hip extensor moment (Figure 3), there were minimal changes in hamstring activation (Figure 5) in the decreased trunk flexion condition

The results of this study demonstrate a clear association between hip flexor stiffness and knee OA. relationship. While it is possible that OA leads directly to changes in hip muscle stiffness, it is equally possible that increased passive stiffness may result from other mechanisms and may play a role in the

aetiology of the disease. In a recent study we demonstrated a link between physical activity patterns and passive hip flexor stiffness in healthy people [18]. Specifically, we showed that healthy individuals who were inactive and sat for prolonged periods, exhibited a mean Thomas test angle of 1.4° (hip flexion), almost identical to the angle recorded from the healthy control group in this present study (Figure 1). In contrast, the active group in our previous study exhibited a Thomas test angle of -4.7°, which is over 10° smaller than the angle of the OA group in this study (Figure 1). Given these findings, and the fact that people with knee OA have reduced physical activity levels [24], it is possible that the difference in passive hip stiffness observed in this study is a result of physical activity avoidance.

If physical activity avoidance does underlie increased passive hip stiffness, then this may lead to increased trunk flexion, which may, in turn, increase hamstring activity and medial co-contraction [8]. Given the potential links between co-contraction and pain [25], loading [2] and disease progression [3, 4], it may be possible for patients with knee OA to find themselves trapped within a negative feedback loop. Specifically, pain may lead to activity avoidance which in turn may lead to increased passive stiffness, compromised postural alignment, altered loading, pain and further activity avoidance. If such a mechanism is at play in people with knee OA, then interventions are required which can improve postural alignment by reducing passive tissue stiffness and which also discourage activity avoidance behaviours. We are currently developing such an intervention [26].

There are some limitations to this study. Firstly, we used a clinical test, the Thomas test, to quantify passive stiffness of the hip flexor muscles. Although this test has been shown to have good reliability [27], research is required using more objective measurement techniques to quantify passive stiffness of musculotendinous structures around the hip. Secondly, we did not measure spinopelvic mobility. It was therefore not possible to explore whether between-subject differences in the alignment of the lumbopelvic-hip complex were related to the biomechanical response to altering trunk flexion. Thirdly, trunk flexion was measured using a system of reflective markers placed on the sternum and thoracic vertebrae. Using a single spinal upper body segment, we made inferences about the orientation of the trunk, a multiarticulate structure. While more complex kinematic modelling techniques are required in future studies, we feel that this current study brings new insights into the relationship between hip flexor

stiffness, trunk flexion and knee flexor muscle activity. Finally, we did not quantify the activity levels of the participants. Therefore, we cannot know definitively if the group with OA were more inactive than the healthy group. Further research is therefore needed to explore the relationship between passive hip flexor stiffness and activity patterns in people with knee OA.

Conclusions

Individuals with knee OA demonstrated increased passive stiffness of the hip flexor muscles which was linked to an increase in trunk flexion during walking. While a simple instruction to increase trunk flexion in walking led to a clear increase in hamstring activity, the instruction to decrease trunk flexion produced only small reductions in hamstring activation. We suggest that this is because pelvic alignment in the sagittal plane Is likely to be determined by passive stiffness of the hip flexor muscles and may therefore be difficult to modify through simple postural instruction. If this is the case, interventions are required which can bring about improvements in postural alignment by reducing muscle stiffness.

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Competing interest statement

The authors have no competing interesting to declare.

Ethical statement

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.

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Figures

Figure 1: Passive hip flexion measured in supine (left panel) and trunk flexion during walking (right panel) for the individuals with knee OA and the healthy group.

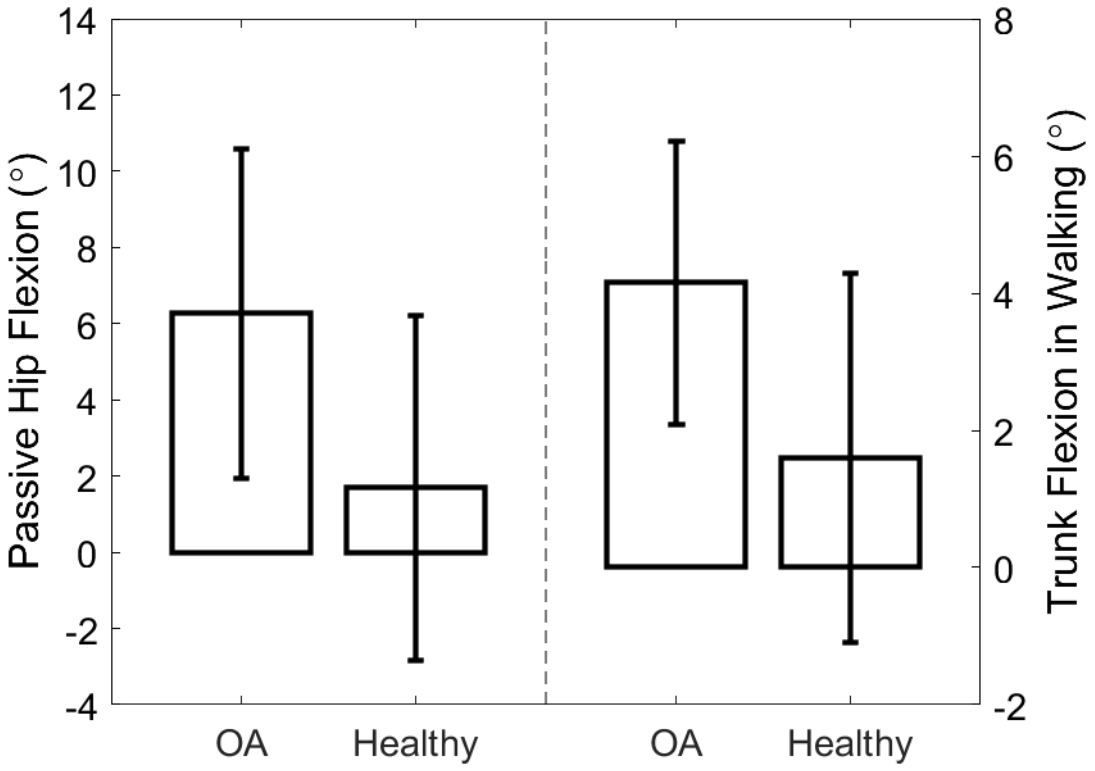
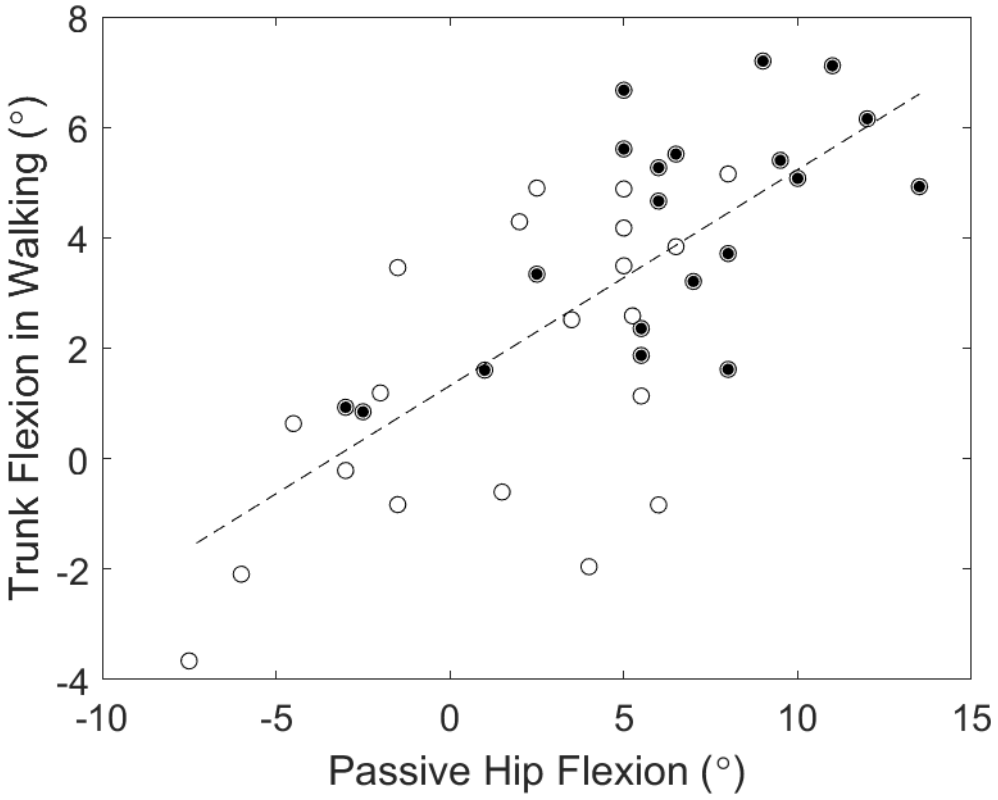


Figure 2: Scatter plot illustrating the association between passive hip flexion measured in supine and trunk flexion angle in walking. Healthy data are shown as unfilled and knee OA data as filled circles.



372 Figure 3: Trunk flexion angles in the healthy and knee OA groups for the three walking conditions, normal
373 walking (solid), increased trunk flexion (dashed) and decreased trunk flexion (dotted) across the stance
374 phase of walking.

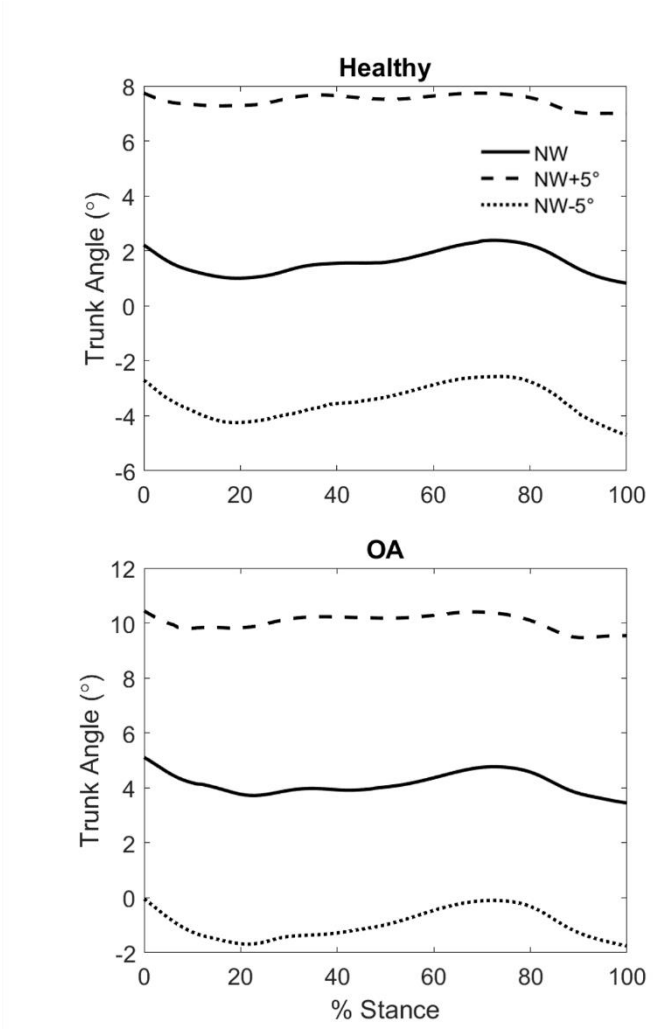


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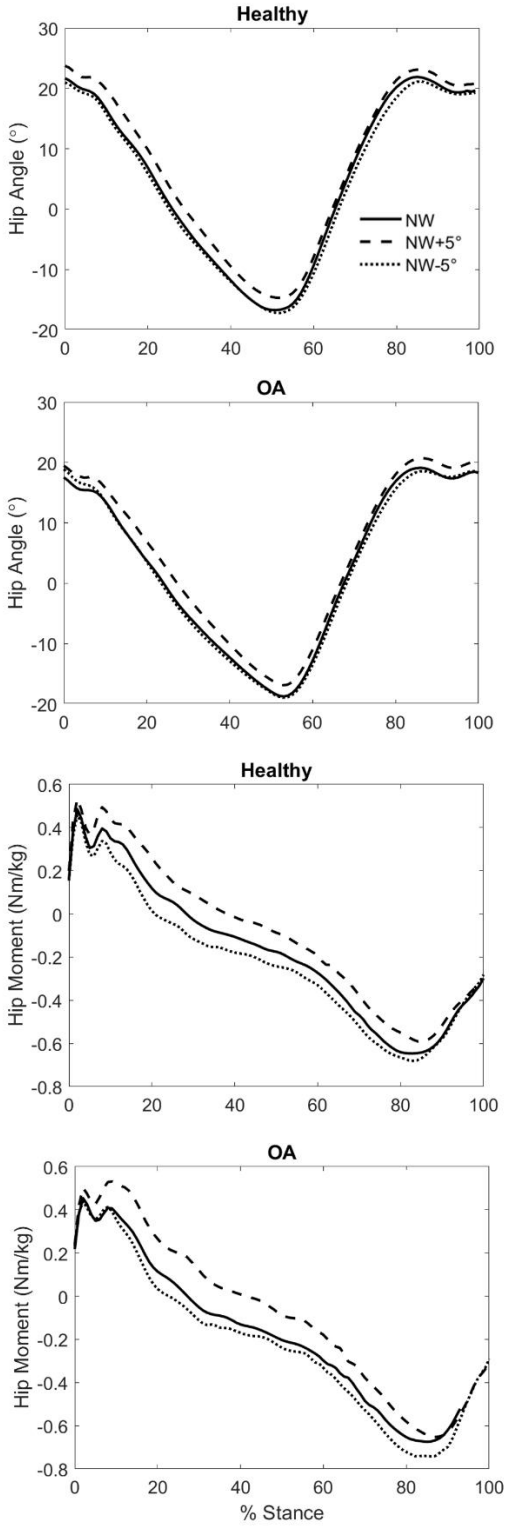
Figure 4: Hip flexion angles and (internal) hip extensor moments for the healthy and knee OA groups across the three walking conditions, normal walking (solid), increased trunk flexion (dashed) and decreased trunk flexion (dotted) across the stance phase of walking.



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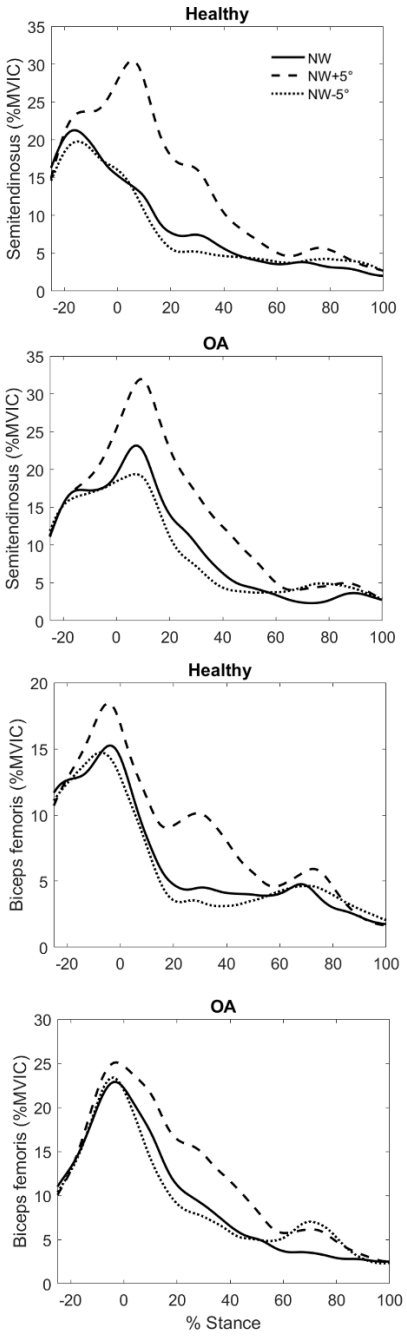
382 Figure 5: Muscle activation profiles for biceps femoris and semitendinosus for the healthy and knee OA
383 groups across the three walking conditions, normal walking (solid), increased trunk flexion (dashed) and
384 decreased trunk flexion (dotted) across the stance phase of walking.



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Tables

Table 1: ANOVA testing for the effect of trunk flexion and group x trunk flexion interaction. Post hoc test results are given for the comparison between decreased trunk flexion and normal walking and for the comparison between increased trunk flexion and normal walking. Statistical significance at p<0.01 with Bonferroni adjustment is denoted by *.

			Decreased trunk flexion vs normal walking		**Increased trunk flexion vs normal walking**	
	Effect of flexion	**interaction**	**Confidence interval**	**Change**	**Confidence interval**	**Change**
Sagittal hip angle (Nm/Kg)	p<0.001	p=0.50	(-1.4, 0.3)	-11%	(1.6, 4.6)*	60%
Sagittal hip moment (°)	p<0.001	p=0.55	(-0.12, -0.05)*	-64%	(0.09, 0.19)*	103%
Biceps femoris (%MVIC)	P<0.001	P=0.17	(-3.8, 0.2)	-18%	(2.1, 6.2)*	41%
Semitendinosus (%MVIC)	p<0.001	p=0.80	(-6.3, 1.6)	-16%	(3.9, 18.2)*	79%

W