



2016-12-01

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The Effects of Experimental Anterior Knee Pain on
Bilateral Ground Reaction Forces
During Running

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A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Master of Science

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ABSTRACT

The Effects of Experimental Anterior Knee Pain on Bilateral Ground Reaction Forces During Running

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The purpose of this study was to examine the independent effects of anterior knee pain (AKP) on bilateral ground reaction force (GRF) during running, with a focus on GRF applied to the uninvolved leg, which, prior to this study, had never been evaluated. Twelve volunteers completed three data collection sessions, that corresponded to one of three conditions (control, sham, and pain), in a counterbalanced order. For each session, subjects ran for five minutes. For the pain and sham sessions, respectively, hypertonic and isotonic saline were infused into the infrapatellar fat pad of the right leg during the running, while no infusion was involved in the control session. GRF data were collected during the final 30 seconds of running. Functional statistics were used to determine the effects of session and leg (right and left) on vertical and anterior-posterior GRF throughout the stance phase of running. A mixed model ANOVA was used to determine the effect of session and leg on vertical GRF load rate, impulse due to vertical, propulsive, and braking GRFs. A repeated measures ANOVA was used to determine the effect of session and time on subject-perceived pain. Alpha was set to 0.05 for all statistical comparisons. Unexpectedly, no significant session \times leg interaction existed for vertical GRF at any time point during stance phase of running. Similarly, the experimental AKP did not affect impulse due to vertical GRF or load rate for the vertical GRF. There was, however, a significant session \times leg interaction for anterior-posterior GRF. For the pain session, involved-leg braking GRF was 11% greater than uninvolved-leg braking GRF during the first 9% of stance phase. There was also a significant between-session difference for involved-leg braking impulse ($p = 0.023$) and uninvolved-leg propulsive impulse ($p = 0.027$). The mean involved-leg braking impulses were 11.3 Ns (± 0.6), 13.2 Ns (± 0.6) and 13.2 Ns (± 0.6) for the pain, control, and sham sessions, respectively. Mean uninvolved-leg propulsive impulses were 14.8 Ns (± 1.3), 13.6 Ns (± 1.3), and 13.5 Ns (± 1.3) for the pain, control, and sham sessions, respectively. These differences in anterior-posterior GRF might reflect a compensatory unloading of the involved leg due to AKP.

Key Words: anterior knee pain, ground reaction force, running

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Introduction

Knee pathology and the related knee pain are common.^{18,54,55} Knee pain affects one in four people over the age of 55,⁵⁴ and the knee is the most commonly injured joint for athletes.¹⁸ Specifically, anterior knee pain (AKP) is one of the most common knee problems,¹⁸ particularly in runners.⁵⁵ AKP etiology is unclear. Proposed causes of AKP include, but are not limited to, increased patellar mobility,⁶² elevated patellofemoral stress,²⁵ and decreased patellofemoral contact area.²⁵

It is thought that individuals who suffer from AKP employ various compensatory strategies to unload the involved (i.e., painful) leg, in an attempt to decrease pain.^{7,13,38,50} These compensatory strategies could involve altered joint kinetics^{24,27,36,42,50} and kinematics,^{38,60} as well as abnormal neural activation characteristics.^{14,29,42} Many of these neuromechanical alterations are reflected in altered ground reaction force (GRF) that is transmitted to the involved leg. GRF has been used to reflect altered neuromechanics for the entire lower extremity. For example, arthrogenic muscle inhibition, due to AKP, alters GRF.^{23,26,36,50} AKP inhibits quadriceps activation, thereby reducing knee extension torque, vertical GRF, and braking GRF.^{23,26,36,41,50} Other muscles that may also be inhibited by AKP include the gastrocnemius and gluteus medius, which contribute to the propulsive and vertical GRFs.^{20,24,36} If allowed to persist, abnormal GRF, applied to the involved leg, might lead to other pathologies (e.g., knee joint effusion, patellofemoral joint crepitus, and abnormal patellar tracking).⁴⁵

While the effects of AKP on the involved leg have been documented, effects of AKP on the uninvolved (i.e., pain-free) leg have not. Examining the effects of AKP on the uninvolved leg is important because as individuals unload the involved leg,^{7,13,38,50} a greater load is likely applied to the uninvolved leg, which could potentially cause pathologies for the uninvolved leg.

In addition to pain, other factors are also associated with knee pathology (e.g., joint effusion,⁵⁷ muscle weakness,³⁵ and inflammation⁴), which make the independent effects of AKP difficult to study. For this reason, experimental AKP models have been developed to learn more regarding the independent effects of AKP. These models involve hypertonic saline that is placed in the infrapatellar fat pad.^{7,20,28,50} Pain is produced by the hypertonic saline due to the higher concentration of salt in the saline compared to normal body fluids. It is thought that the AKP that results from these models effectively represents neuromechanical effects of AKP; for example, experimental AKP lowers frontal and sagittal plane knee joint moments,²⁶ impairs postural control,²⁸ and inhibits involuntary and voluntary quadriceps activation patterns.⁴² Other characteristics of experimental AKP (e.g., distribution patterns and quality of pain) have also been shown to represent clinical AKP.⁸

The purpose of this study was to examine the independent effects of AKP on bilateral GRF during running, with a focus on the GRF applied to the uninvolved leg. We hypothesized that, relative to pain-free running, experimental AKP would increase vertical GRF that is transmitted to the uninvolved leg, as well as increase vertical GRF load rate and impulse transmitted to the uninvolved leg (indicating increased load to the uninvolved leg). We also hypothesized that experimental AKP would decrease braking GRF for the involved leg, as well as increase propulsive GRF for the uninvolved leg.

Methods

Subjects

Twelve volunteers between the ages of 18 and 40 participated in this study. Several statistical power analyses, each based on a previously studied GRF characteristic, such as peak braking GRF and peak vertical GRF,⁵⁰ and $n = 12$ subjects who experienced three conditions,⁵⁰

suggested that sufficient power (> 0.80) could be expected with this sample size. The same previous research indicated that we could expect effect sizes that range from 0.8 to one.⁵⁰ This sample size and the associated expected statistical power are based on 95% confidence and rely on assumptions of normal distribution and consistent variance for the dependent variables.

Subjects were required to have no history of (1) lower extremity injury in the six months prior to participation in this study, and (2) knee-related surgery in their lifetime. Further, subjects were required to be running at least 10 miles per week at the time of data collection in order to increase the likelihood that they would be able to complete the data collection protocol. To ensure that subjects met these criteria, they were assessed by a questionnaire that involved all of the aforementioned criteria.

Experimental Protocol

Subjects completed three data collection sessions in the same biomechanics laboratory. Each session corresponded to one of three conditions (control, sham, and pain), with each session being held 48 hours apart. Conditions were administered in a counterbalanced order. Subjects were instructed to refrain from exercising before and throughout data collection, starting 48 hours prior to the first data collection session and lasting until they had completed all three sessions. Before any of the data collection sessions, subjects met with researchers once to become familiar with the research protocol and provide informed consent. Prior to the collection of any data, all data collection procedures were approved by the appropriate institutional review board.

For each data collection session, subjects reported to the laboratory in the same pair of their own athletic shoes. Subjects then changed into running shorts that were provided by the researchers. Female subjects completed each session in running shorts, shoes, and their own

sports bra, while male subjects completed each session in just running shorts and shoes. To warm-up, subjects walked on the instrumented treadmill (ATMI, Watertown, MA, USA) for five minutes at a self-selected walking speed, at a speed that was representative of their typical walking speed. Next subjects ran for five minutes on the treadmill at one of three speeds (3.0, 3.5, or 4.0 m/s). Subjects were instructed to select the highest of the three speeds that they could comfortably run for five continuous minutes. The same selected running speed was used for each session, as determined during the first data collection session.

For each data collection session, upon completion of the warm-up, subjects laid supine on a treatment table. For the sham and pain sessions, the infusions (sham: a continuous infusion of isotonic solution (0.9% NaCl) into the right infrapatellar fat pad; pain: a continuous infusion of hypertonic solution (5.0% NaCl)) were initiated at this point. Prior to each needle stick, the skin was shaved and prepared with an iodine swab and alcohol wipe. A 20-gauge flexible catheter (Becton Dixon Medical Systems, Sandy, UT) was then inserted into the infrapatellar fat pad of the right leg. The catheter was inserted from the lateral side of the leg in an inferio-medial direction to a depth of one cm. The catheter was placed immediately posterior to the patellar tendon, in the middle of the infrapatellar fat pad, as has been done previously.^{20,28,50} Diagnostic ultrasound (General Electric, Wauwatosa, WI) was used to evaluate correct placement of the catheter during pilot testing, though it was not used during data collection. The catheter was placed in the right leg for every subject for each session. A plastic 76-cm connection tube was used to connect the catheter to a 30-mL syringe and portable syringe pump. For the duration of the run during the sham and pain sessions, the pump was held in a miniature fanny pack provided for the subjects to wear during these data collection sessions. The fanny pack was not worn during the control session. Subjects were blinded about which solution had been prepared for

infusion. Once the infusion had begun, subjects laid supine for two minutes, sat for two minutes, and stood for two minutes. These six minutes helped subjects to become familiar with the infusion and consequent experimental knee pain, and minimize the chance of a vasovagal response (i.e., the subjects becoming light-headed and passing out). None of the subjects experienced a vasovagal response due to the infusion. During the control session, subjects followed the same familiarization protocol, although an infusion was not involved.

For each data collection session, following the six-minute familiarization period, subjects performed 10 minutes of exercise, five minutes of running and five minutes of walking, on the instrumented treadmill. Between subjects, the walk and the run were performed in a counterbalanced order, with each subject performing the walk and the run in their designated order for all three sessions. Three-dimensional GRFs were recorded (2000 Hz) during the final 30 seconds of the run. Although we observed walking, it is not presently reported or discussed, due to questionable accuracy of the data for several subjects. At the conclusion of the walking and running, subjects returned to the treatment table, the catheter was removed, and the subjects rested for 30 minutes. The control session was identical to the sham and pain sessions, except that there was no involved infusion. For each session, subject-perceived pain was measured every three minutes, from immediately prior to the needle stick to 30 minutes after the completion of the exercise, using a 10 cm visual analog scale (VAS). On the left end of the visual analog scale, 'no pain' was typed, while 'worst possible pain' was typed on the right end of the scale. The subject-perceived pain data was observed to ensure that the experimental AKP was perceived consistently by the subjects. Ten consecutive stance phases from the run were analyzed. Figure 1 is a schematic that describes the timeline that was followed for each data collection session.

Statistical Analysis

The independent variables for this study were session, leg, and time. The continuous dependent variables were vertical and anterior-posterior GRF. The discrete dependent variables were subject-perceived pain, load rate between initial contact and impact peak vertical GRF, and impulse due to the vertical GRF (throughout stance), and propulsive and braking GRF. Vertical and anterior-posterior GRF, across the entire stance phase, were compared between sessions (control, sham, and pain) and legs (right and left) using a functional analysis approach. This approach allows for the comparison of GRF across the entire stance phase, rather than only at discrete times. Subject-perceived pain was compared between sessions (16 times, from pre-needle stick to 48 minutes post-needle stick), using a mixed model repeated measures ANOVA. Vertical GRF load rate and impulses (due to vertical, braking, and propulsive GRF) were compared between sessions and legs using a mixed models ANOVA. Alpha levels for all the statistical comparisons were set, *a priori*, at 0.05.

Results

Subject-Perceived Pain

We observed significant between-session differences for subject-perceived pain, at each VAS measurement time point, between minutes 9 and 36 ($p < 0.05$; Figure 2). During this duration, subject-perceived pain was greater during the pain session, relative to the sham and control sessions. There was no significant difference, at any time point, between the sham and control sessions.

Vertical GRF Characteristics

There was no session \times leg interaction for vertical GRF at any time point of stance (Figure 3). Further, no significant session \times leg interaction was observed for vertical GRF load

rate ($p = 0.961$; Table 1) or impulse due to vertical GRF ($p = 0.073$; Table 1). There was, however, a significant main effect of leg ($p = 0.049$) for the impulse due to vertical GRF: when data were compared bilaterally, pooled from all three sessions, mean vertical GRF impulse was 1.1% less for the involved leg, relative to the uninvolved leg. The effect size when comparing legs for the impulse due to vertical GRF was 0.06, which is a very small effect size.

Anterior-Posterior GRF Characteristics

A significant session \times leg interaction was observed for anterior-posterior GRF. For the pain session only, involved-leg anterior-posterior GRF was about 11% greater, relative to uninvolved-leg anterior-posterior GRF, during the first 9% of stance ($p < 0.05$; Figure 4D). Further, there was a significant between-session difference for involved-leg braking impulse ($p = 0.023$). Mean involved-leg braking impulse for the pain session ($11.3 \text{ Ns} \pm 0.6$) was 14% less than for the control ($13.2 \text{ Ns} \pm 0.6$; 0.73) and sham ($13.1 \text{ Ns} \pm 0.6$; 0.63) sessions. There was also a significant between-session difference for uninvolved-leg propulsive impulse ($p = 0.027$). Mean uninvolved-leg propulsive impulse for the pain session ($14.8 \text{ Ns} \pm 1.3$) was 8% greater than for the control ($13.6 \text{ Ns} \pm 1.3$; 0.24) and sham ($13.5 \text{ Ns} \pm 1.3$; 0.28) sessions.

Summary

As expected, the hypertonic saline infusion of the pain session significantly increased subject-perceived pain. However, the experimental AKP did not significantly affect vertical GRF during running. The experimental AKP did, however, significantly influence observed characteristics of anterior-posterior GRF, during running, in three ways. First, during the first 9% of the stance phase, for the pain session only, anterior-posterior GRF was about 11% greater for the involved leg, relative to the uninvolved leg. Second, the braking impulse for the involved leg was less for the pain session, relative to the control and sham sessions. Third, the propulsive

impulse for the uninvolved leg was greater for the pain session, relative to the control and sham sessions.

Discussion

We conducted this study to evaluate the independent effects of AKP on bilateral GRF during running, with a focus on the uninvolved leg (i.e., the leg that did not experience pain). Numerous studies exist regarding the effects of AKP on the involved leg;^{1,30,38,39,41,60} however, the effects of AKP on the uninvolved leg have yet to be documented. Further, few studies have looked at the effects of AKP during running.^{6,9,20,50} Since AKP is one of the most common knee problems,¹⁸ especially in runners,⁵⁵ this is an important area of study. This novel study is important because it is the first to quantify independent effects of AKP on bilateral GRF during running (we did so using the experimental AKP model).

We hypothesized that, relative to pain-free running, experimental AKP would increase vertical GRF transmitted to the uninvolved leg, throughout stance, as well as increase the vertical GRF load rate and impulse due to vertical GRF for the uninvolved leg. We also hypothesized that experimental AKP would decrease braking GRF for the involved leg, as well as increase propulsive GRF for the uninvolved leg. The present results failed to support the first hypothesis related to vertical GRF: AKP did not independently increase vertical GRF applied to the uninvolved leg, nor did AKP increase vertical GRF load rate or impulse due to vertical GRF for the uninvolved leg. Although we did see a significant main effect of leg for the impulse due to vertical GRF, it is not likely clinically or functionally significant due to the small effect size of 0.06 that is associated with it. The present results did, however, support both of the hypotheses regarding anterior-posterior GRF: AKP independently decreased braking GRF for the involved leg. Further, AKP independently increased propulsive GRF for the uninvolved leg. For the

braking GRF, when comparing the pain session to the control and sham sessions, we saw effect sizes of 0.73 and 0.63, respectively. These are moderate effect sizes. We also saw effect sizes of 0.24 and 0.28 when comparing the pain session to the control and sham sessions, respectively, for the propulsive GRF. These are small effect sizes.

Although the present data cannot definitively identify specific causes of the observed alterations in anterior-posterior GRF, neuromechanical compensations that have previously been associated with AKP fit with the observed anterior-posterior GRF alterations. Previous researchers have described several compensatory strategies that are associated with unloading of the involved leg during AKP,^{1,38,44,60} including altered neural activation patterns^{14,24,27,42} which can alter anterior-posterior GRF. For example, several studies have indicated that AKP inhibits quadriceps activation, and decreased quadriceps activation would likely reduce braking GRF.^{24,27,36} As stated previously, the present results indicate a decrease in braking GRF for the involved leg and an increase in propulsive GRF for the uninvolved leg during the pain session. The decrease in braking GRF for the involved leg could be due to AKP inhibiting quadriceps activation.

This study adds to the existing body of literature regarding the use of experimental AKP to examine the independent effects of AKP on various knee pathologies. As there are numerous other factors associated with knee pathology in addition to pain (e.g., joint effusion⁵⁷ and inflammation⁴), the study of the independent effects of AKP is difficult. As such, experimental AKP models have been developed to study the independent effects of AKP. While some studies have used an injection technique to administer hypertonic saline into the infrapatellar fat pad,^{7,27,28,29} fewer studies have used a continuous infusion of hypertonic saline.^{20,50} This is the first study that utilized the continuous infusion method to examine bilateral running GRF. The

present results strengthen the idea that this infusion model can effectively induce experimental AKP during dynamic physical activity, including running. This study also corroborates previous research^{20,50} in supporting the idea that the continuous infusion model can be effectively used to induce experimental AKP for a relatively extended duration: for the present pain session, AKP levels were consistent during the entire infusion (17 minutes; Figure 2).

The magnitude of subject-perceived pain appears to sufficiently represent clinical AKP (Figure 2); however, it is unclear whether other characteristics of this experimental AKP are representative of clinical AKP. Previous research supports the idea that experimental AKP induced by hypertonic saline is representative of joint pain related to knee pathology by indicating that the quality and distribution of pain elicited by hypertonic saline are similar to clinical AKP.⁸

The results of this study have potential clinical implications. It is important for rehabilitative clinicians to clearly understand how bilateral mechanics can be altered as a result of AKP; such alterations can potentially cause pathologies for the uninvolved leg. With this knowledge, clinicians could potentially treat AKP more effectively and comprehensively. Several studies have shown that at the conclusion of a bilateral strengthening program, individuals with AKP experienced a decrease in pain, as well as an increase in strength and function.^{19,22} The present results support the idea that clinicians should consider strengthening exercises for the involved and uninvolved legs during AKP rehabilitation. In addition to the idea of strengthening an impaired involved-leg quadriceps, which has previously been associated with AKP, clinicians might also consider treating the muscles that are associated with anterior-posterior GRF. For example, the gastrocnemius has been shown to be inhibited by AKP, which contributes to propulsive GRF.^{20,24} As AKP patients increase certain loads applied to the

uninvolved leg (e.g., increased propulsive GRF) while compensating for involved-leg AKP, some uninvolved-leg musculoskeletal structures (e.g., the gastrocnemius) may be overloaded and experience related chronic injury. Strengthening the muscles that could be overworked may help prevent pathologies that could arise.

In conclusion, this novel study is important for several reasons. It is the first study to observe the independent effects of AKP on bilateral GRFs during running. The findings of this study indicate that there was a decrease in braking GRF for the involved leg and an increase in propulsive GRF for the uninvolved leg as a result of the experimental AKP. The experimental AKP did not influence any of the vertical GRF characteristics. This study adds to the current body of literature by elucidating some of the independent, bilateral effects of running in the presence of unilateral AKP.

References

1. Aliberti, S., Costa, M. d. S. X., Passaro, A. d. C., Arnone, A. C., Hirata, R., & Sacco, I. C. N. (2011). Influence of patellofemoral pain syndrome on plantar pressure in the foot rollover process during gait. *Clinics*, 66, 367-372.
2. Aminaka, N., Pietrosimone, B. G., Armstrong, C. W., Meszaros, A., & Gribble, P. A. (2011). Patellofemoral pain syndrome alters neuromuscular control and kinetics during stair ambulation. *Journal of Electromyography and Kinesiology*, 21(4), 645-651.
3. Anderson, F. C., & Pandy, M. G. (2003). Individual muscle contributions to support in normal walking. *Gait & Posture*, 17(2), 159-169.
4. Baker, V., Bennell, K., Stillman, B., Cowan, S., & Crossley, K. (2002). Abnormal knee joint position sense in individuals with patellofemoral pain syndrome. *Journal of Orthopaedic Research*, 20(2), 208-214.
5. Barton, C. J., Levinger, P., Webster, K. E., & Menz, H. B. (2011). Walking kinematics in individuals with patellofemoral pain syndrome: A case-control study. *Gait & Posture*, 33(2), 286-291.
6. Bazett-Jones, D. M., Cobb, S. C., Huddleston, W. E., O'Connor, K. M., Armstrong, B. S. R., & Earl-Boehm, J. E. (2013). Effect of patellofemoral pain on strength and mechanics after an exhaustive run. *Medicine & Science in Sports & Exercise*, 45(7), 1331-1339 1310.
7. Bennell, K. L., & Hinman, R. S. (2005). Effect of experimentally induced knee pain on standing balance in healthy older individuals. *Rheumatology*, 44(3), 378-381.
8. Bennell, K., Hodges, P., Mellor, R., Bexander, C., & Souvlis, T. (2004). The nature of anterior knee pain following injection of hypertonic saline into the infrapatellar fat pad. *Journal of Orthopaedic Research*, 22(1), 116-121.
9. Besier, T. F., Fredericson, M., Gold, G. E., Beaupré, G. S., & Delp, S. L. (2009). Knee muscle forces during walking and running in patellofemoral pain patients and pain-free controls. *Journal of Biomechanics*, 42(7), 898-905.
10. Bohnsack, M., Hurschler, C., Demirtas, T., Rühmann, O., Stukenborg-Colsman, C., & Wirth, C.-J. (2005). Infrapatellar fat pad pressure and volume changes of the anterior compartment during knee motion: possible clinical consequences to the anterior knee pain syndrome. *Knee Surgery, Sports Traumatology, Arthroscopy*, 13(2), 135-141.
11. Bohnsack, M., Klages, P., Hurschler, C., Halcour, A., Wilharm, A., Ostermeier, S., Rühmann, O., & Wirth, C.-J. (2009). Influence of an infrapatellar fat pad edema on patellofemoral biomechanics and knee kinematics: a possible relation to the anterior knee pain syndrome. *Archives of Orthopaedic and Trauma Surgery*, 129(8), 1025-1030.
12. Boling, M., Padua, D., Marshall, S., Guskiewicz, K., Pyne, S., & Beutler, A. (2010). Gender differences in the incidence and prevalence of patellofemoral pain syndrome. *Scandinavian Journal of Medicine & Science in Sports*, 20(5), 725-730.
13. Brechter, J. H., & Powers, C. M. (2002). Patellofemoral joint stress during stair ascent and descent in persons with and without patellofemoral pain. *Gait & Posture*, 16(2), 115-123.
14. Brindle, T., Mattacola, C., & McCrory, J. (2003). Electromyographic changes in the gluteus medius during stair ascent and descent in subjects with anterior knee pain. *Knee Surgery, Sports Traumatology, Arthroscopy*, 11(4), 244-251.

15. Callaghan, M. J., & Baltzopoulos, V. (1994). Gait analysis in patients with anterior knee pain. *Clinical Biomechanics*, 9(2), 79-84.
16. Cheung, R. T. H., & Davis, I. S. (2011). Landing pattern modification to improve patellofemoral pain in runners: A case series. *Journal of Orthopaedic & Sports Physical Therapy*, 41(12), 914-919.
17. Das, S. K., & Farooqi, A. (2008). Osteoarthritis. *Best Practice & Research Clinical Rheumatology*, 22(4), 657-675.
18. DeHaven, K. E., & Lintner, D. M. (1986). Athletic injuries: Comparison by age, sport, and gender. *The American Journal of Sports Medicine*, 14(3), 218-224.
19. De Marche Baldon, R., Serrao, F. V., Silva, R. S., Piva, S. R. (2014). Effects of functional stabilization training on pain, function, and lower extremity biomechanics in women with patellofemoral pain: A randomized clinical trial. *Journal of Orthopaedic & Sports Physical Therapy*, 44(4), 240-251.
20. Denning, W. M., Woodland, S., Winward, J. G., Leavitt, M. G., Parcell, A. C., Hopkins, J. T., Francom, D., & Seeley, M. K. (2014). The influence of experimental anterior knee pain during running on electromyography and articular cartilage metabolism. *Osteoarthritis and Cartilage*, 22(8), 1111-1119.
21. Fairbank, J., Pynsent, P., van Poortvliet, J., & Phillips, H. (1984). Mechanical factors in the incidence of knee pain in adolescents and young adults. *Journal of Bone & Joint Surgery, British Volume*, 66-B(5), 685-693.
22. Ferber, R., Kendall, K. D., & Farr, L. (2011). Changes in knee biomechanics after a hip-abductor strengthening protocol for runners with patellofemoral pain syndrome. *The Journal of Athletic Training*, 46(2), 142-149.
23. Flynn, T. W., & Soutas-Little, R. W. (1995). Patellofemoral joint compressive forces in forward and backward running. *Journal of Orthopaedic & Sports Physical Therapy*, 21(5), 277-282.
24. Hamner, S. R., Seth, A., & Delp, S. L. (2010). Muscle contributions to propulsion and support during running. *Journal of Biomechanics*, 43(14), 2709-2716.
25. Heino Brechter, J., & Powers, C. M. (2002). Patellofemoral stress during walking in persons with and without patellofemoral pain. *Medicine and science in sports and exercise*, 34(10), 1582-1593.
26. Henriksen, M., Graven-Nielsen, T., Aaboe, J., Andriacchi, T. P., & Bliddal, H. (2010). Gait changes in patients with knee osteoarthritis are replicated by experimental knee pain. *Arthritis Care & Research*, 62(4), 501-509.
27. Henriksen, M., Rosager, S., Aaboe, J., Graven-Nielsen, T., & Bliddal, H. (2011). Experimental Knee Pain Reduces Muscle Strength. *The Journal of Pain*, 12(4), 460-467.
28. Hirata, R., Arendt-Nielsen, L., Shiozawa, S., & Graven-Nielsen, T. (2012). Experimental knee pain impairs postural stability during quiet stance but not after perturbations. *European Journal of Applied Physiology*, 112(7), 2511-2521.
29. Hodges, P. W., Mellor, R., Crossley, K., & Bennell, K. (2009). Pain induced by injection of hypertonic saline into the infrapatellar fat pad and effect on coordination of the quadriceps muscles. *Arthritis Care & Research*, 61(1), 70-77.
30. Ireland, M. L., Wilson, J. D., Ballantyne, B. T., & Davis, I. M. (2003). Hip strength in females with and without patellofemoral pain. *Journal of Orthopaedic & Sports Physical Therapy*, 33(11), 671-676.

31. Karst, G. M., & Willett, G. M. (1995). Onset timing of electromyographic activity in the vastus medialis oblique and vastus lateralis muscles in subjects with and without patellofemoral pain syndrome. *Physical Therapy*, 75(9), 813-823.
32. King, C. D., Sibille, K. T., Goodin, B. R., Cruz-Almeida, Y., Glover, T. L., Bartley, E., Riley, J. L., Herbert, M. S., Sotolongo, A., Schmidt, J., Fessler, B. J., Redden, D. T., Staud, R., Bradley, L. A., & Fillingim, R. B. (2013). Experimental pain sensitivity differs as a function of clinical pain severity in symptomatic knee osteoarthritis. *Osteoarthritis and Cartilage*, 21(9), 1243-1252.
33. Levinger, P., & Gilleard, W. (2005). The heel strike transient during walking in subjects with patellofemoral pain syndrome. *Physical Therapy in Sport*, 6(2), 83-88.
34. Levinger, P., & Gilleard, W. (2007). Tibia and rearfoot motion and ground reaction forces in subjects with patellofemoral pain syndrome during walking. *Gait & Posture*, 25(1), 2-8.
35. Lewek, M., Rudolph, K., Axe, M., & Snyder-Mackler, L. (2002). The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. *Clinical Biomechanics*, 17(1), 56-63.
36. Liu, M. Q., Anderson, F. C., Schwartz, M. H., & Delp, S. L. (2008). Muscle contributions to support and progression over a range of walking speeds. *Journal of Biomechanics*, 41(15), 3243-3252.
37. Murphy, L., Schwartz, T. A., Helmick, C. G., Renner, J. B., Tudor, G., Koch, G., Jordan, J. M. (2008). Lifetime risk of symptomatic knee osteoarthritis. *Arthritis Care & Research*, 59(9), 1207-1213.
38. Nadeau, S., Gravel, D., Hébert, L. J., Arsenault, A. B., & Lepage, Y. (1997). Gait study of patients with patellofemoral pain syndrome. *Gait & Posture*, 5(1), 21-27.
39. Naseri, N., & Pourkazemi, F. (2012). Difference in knee joint position sense in athletes with and without patellofemoral pain syndrome. *Knee Surgery, Sports Traumatology, Arthroscopy*, 20(10), 2071-2076.
40. Palmieri-Smith, R. M., Kreinbrink, J., Ashton-Miller, J. A., & Wojtys, E. M. (2007). Quadriceps inhibition induced by an experimental knee joint effusion affects knee joint mechanics during a single-legged drop landing. *The American Journal of Sports Medicine*, 35(8), 1269-1275.
41. Paoloni, M., Mangone, M., Fracocchi, G., Murgia, M., Maria Saraceni, V., & Santilli, V. (2010). Kinematic and kinetic features of normal level walking in patellofemoral pain syndrome: More than a sagittal plane alteration. *Journal of Biomechanics*, 43(9), 1794-1798.
42. Park, J., & Hopkins, J. T. (2013). Induced anterior knee pain immediately reduces involuntary and voluntary quadriceps activation. *Clinical journal of sport medicine: official journal of the Canadian Academy of Sport Medicine*, 23(1), 19-24.
43. Piva, S. R., Fitzgerald, G. K., Irrgang, J. J., Fritz, J. M., Wisniewski, S., McGinty, G. T., Childs, J. D., Domenech, M. A., Jones, S., & Delitto, A. (2009). Associates of physical function and pain in patients with patellofemoral pain syndrome. *Archives of Physical Medicine and Rehabilitation*, 90(2), 285-295.
44. Powers, C. M., Heino, J. G., Rao, S., & Perry, J. (1999). The influence of patellofemoral pain on lower limb loading during gait. *Clinical Biomechanics*, 14(10), 722-728.
45. Price, A. J., Jones, J., & Allum, R. (2000). Chronic traumatic anterior knee pain. *Injury*, 31(5), 373-378.

46. Rathleff, M. S., Samani, A., Olesen, J. L., Roos, E. M., Rasmussen, S., Christensen, B. H., & Madeleine, P. (2013). Neuromuscular activity and knee kinematics in adolescents with patellofemoral pain. *Medicine & Science in Sports & Exercise*, 45(9), 1730-1739.
47. Sanchis-Alfonso, V. (2010). Pathophysiology of anterior knee pain. *Patellofemoral Pain, Instability, and Arthritis*, 1 - 16.
48. Sanchis-Alfonso, V., & Roselló-Sastre, E. (2000). Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment: A neuroanatomic basis for anterior knee pain in the active young patient. *The American Journal of Sports Medicine*, 28(5), 725-731.
49. Sasaki, K., & Neptune, R. R. (2010). Individual muscle contributions to the axial knee joint contact force during normal walking. *Journal of Biomechanics*, 43(14), 2780-2784.
50. Seeley, M. K., Park, J., King, D., & Hopkins, J. T. (2013). A novel experimental knee-pain model affects perceived pain and movement biomechanics. *Journal of Athletic Training*, 48(3), 337-345.
51. Simon, S. R., Paul, I. L., Mansour, J., Munro, M., Abernethy, P. J., & Radin, E. L. (1981). Peak dynamic force in human gait. *Journal of Biomechanics*, 14(12), 817-822.
52. Sørensen, T. J., Langberg, H., Hodges, P. W., Bliddal, H., & Henriksen, M. (2012). Experimental knee joint pain during strength training and muscle strength gain in healthy subjects: A randomized controlled trial. *Arthritis Care & Research*, 64(1), 108-116.
53. Steinberg, N., Siev-Ner, I., Peleg, S., Dar, G., Masharawi, Y., Zeev, A., & HersHKovitz, I. (2012). Joint range of motion and patellofemoral pain in dancers. *International Journal of Sports Medicine*, 33(07), 561-566.
54. Symmons, D. P. (2001). Knee pain in older adults: the latest musculoskeletal "epidemic". *Annals of the Rheumatic Diseases*, 60(2), 89-90.
55. Taunton, J. E., Ryan, M. B., Clement, D. B., McKenzie, D. C., Lloyd-Smith, D. R., & Zumbo, B. D. (2002). A retrospective case-control analysis of 2002 running injuries. *British Journal of Sports Medicine*, 36(2), 95-101.
56. Thijs, Y., De Clercq, D., Roosen, P., & Witvrouw, E. (2008). Gait-related intrinsic risk factors for patellofemoral pain in novice recreational runners. *British Journal of Sports Medicine*, 42(6), 466-471.
57. Torry, M. R., Decker, M. J., Viola, R. W., O'Connor, D. D., & Richard Steadman, J. (2000). Intra-articular knee joint effusion induces quadriceps avoidance gait patterns. *Clinical Biomechanics*, 15(3), 147-159.
58. Tucker, K. J., & Hodges, P. W. (2010). Changes in motor unit recruitment strategy during pain alters force direction. *European Journal of Pain*, 14(9), 932-938.
59. Tucker, K. J., & Hodges, P. W. (2009). Motoneuron recruitment is altered with pain induced in non-muscular tissue. *PAIN*, 141(1-2), 151-155.
60. Willson, J. D., & Davis, I. S. (2008). Lower extremity mechanics of females with and without patellofemoral pain across activities with progressively greater task demands. *Clinical Biomechanics*, 23(2), 203-211.
61. Wirtz, A. D., Willson, J. D., Kernozek, T. W., & Hong, D.-A. (2012). Patellofemoral joint stress during running in females with and without patellofemoral pain. *The Knee*, 19(5), 703-708.
62. Witvrouw, E., Lysens, R., Bellemans, J., Cambier, D., & Vanderstraeten, G. (2000). Intrinsic risk factors for the development of anterior knee pain in an athletic population: A two-year prospective study. *The American Journal of Sports Medicine*, 28(4), 480-489.

Table 1. Vertical GRF Load Rate and Impulse.

	Control		Pain		Sham	
	Uninvolved Leg	Involved Leg	Uninvolved Leg	Involved Leg	Uninvolved Leg	Involved Leg
Load Rate	23293.7 N/s (948.4)	23437.7 N/s (790.4)	23287.1 N/s (1114.5)	23643.1 N/s (987.8)	23655.1 N/s (870.8)	23667.9 N/s (895.9)
Impulse	252.9 Ns (6.1)	252.4 Ns (5.6)	253.3 Ns (6.0)	246 Ns (5.7)	251.1 Ns (6.1)	250.7 Ns (5.7)

Vertical GRF load rate during the early loading phase of stance, as well as comparisons for the vertical GRF impulse. No significant session \times leg interaction existed for the load rate, nor was there any difference between sessions (control, pain, and sham) or legs (involved and uninvolved) for the load rate. There was no significant difference between the control, pain, and sham sessions ($p = 0.073$). Values in the table are reported as mean (standard error).

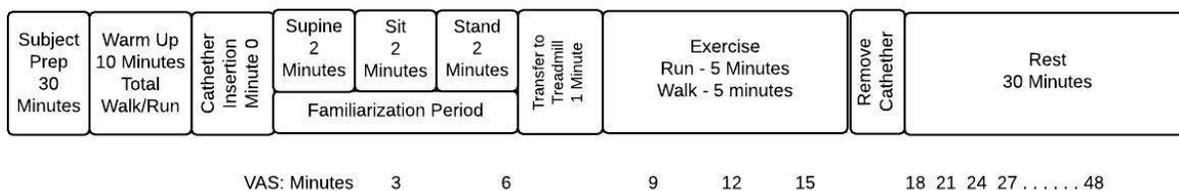


Figure 1. Timeline of events for data collection, for all sessions.

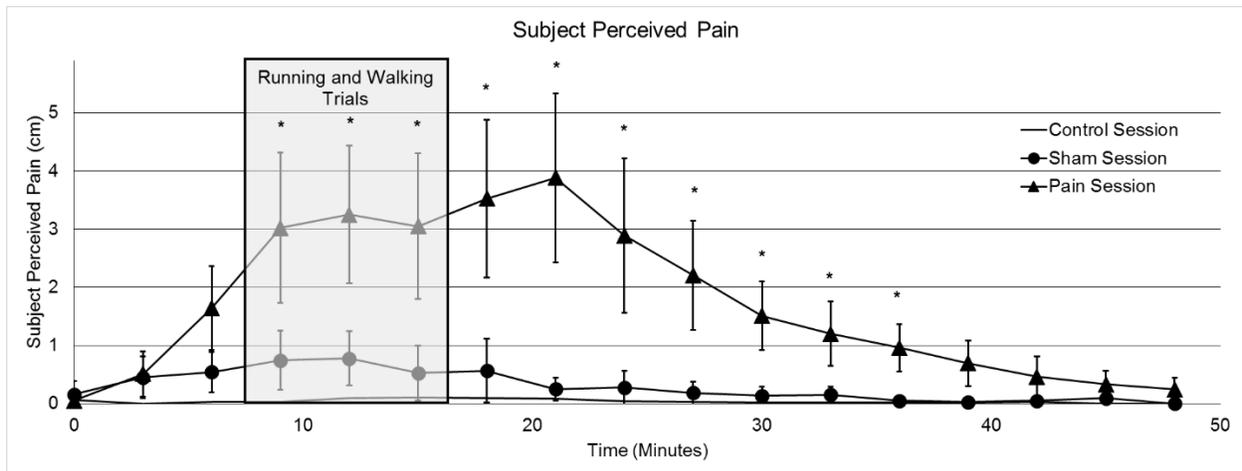


Figure 2. Subject-perceived anterior knee pain, across time for each session, from immediately prior to needle stick (Time Zero) to 30 minutes post-exercise. Each point indicates average subject-perceived pain at a specific time, with vertical bars representing 95% confidence intervals (control session confidence intervals were not included, to increase clarity). Asterisks indicate points in time where the subject-perceived pain for the pain session was significantly greater than for the control and sham sessions.

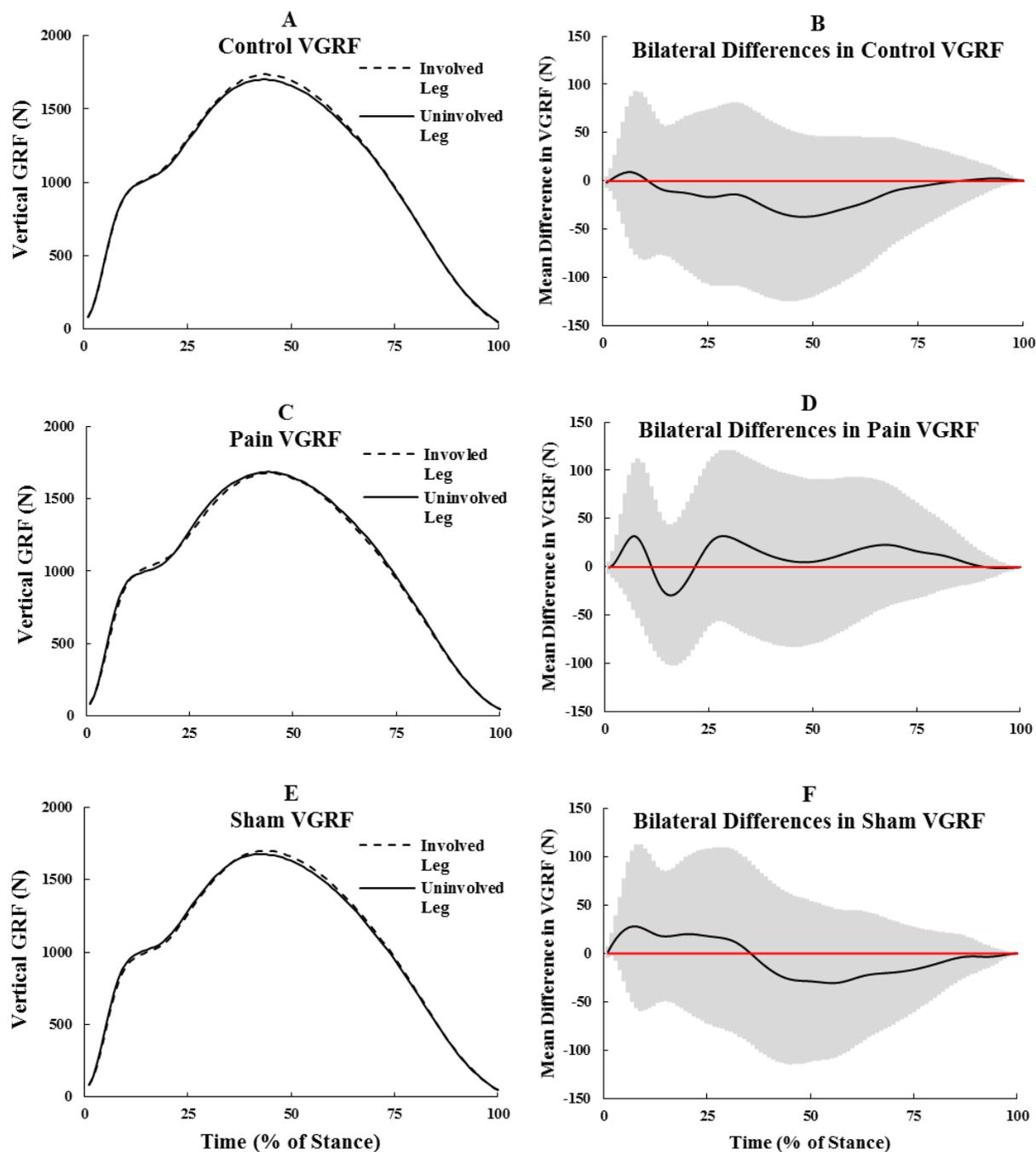


Figure 3. Ensemble means (all subjects), for the right and left legs, for smoothed vertical GRF across the entire stance phase of running, for the control (3A), pain (3C), and sham (3E) data collection sessions. Although not shown here, there was no significant session \times leg interaction for vertical GRF, at any point in time. Further, there was no significant bilateral difference due to the experimental anterior knee pain, for vertical GRF, for any session (3B – control; 3D – pain; and 3F – sham).

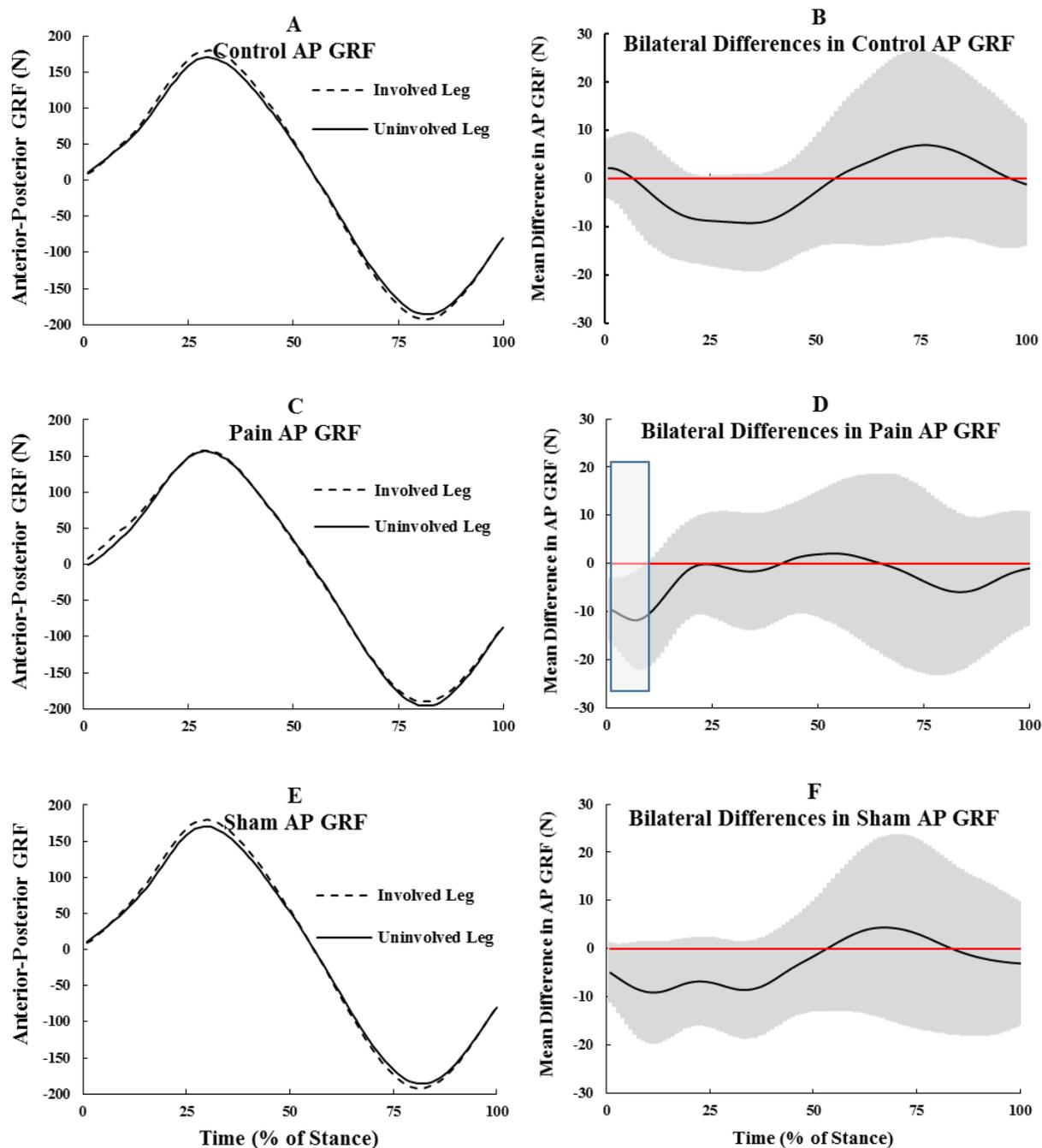


Figure 4. Ensemble means (all subjects), for the right and left legs, for smoothed anterior-posterior GRF across the entire stance phase of running, for the control (4A), pain (4C), and sham (4E) data collection sessions. Although not shown here, there was a significant session \times leg interaction between the control and pain sessions during the first 5% of stance phase; however, there were no other significant session \times leg interactions for any other sessions for the anterior-posterior GRF. Further, there was a significant bilateral difference due to experimental anterior knee pain, for anterior-posterior GRF, during the first 5% of stance phase, for the pain session, as indicated by the shaded box (4D). For the control and sham sessions, there was no significant bilateral difference due to experimental anterior knee pain, for anterior-posterior GRF (4B – control; and 4F – sham).