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Patellofemoral Pain: Proximal, Distal, and Local Factors
2nd International Research Retreat

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Introduction

Patellofemoral pain (PFP) is one of the most common lower extremity conditions seen in orthopaedic practice.[6] While patellofemoral problems are evident in a wide range of individuals, PFP is particularly prevalent in younger persons who are physically active. Based on the data of Taunton et al,[5] approximately 2.5 million runners will be diagnosed with PFP in a given year. PFP also is a significant problem in the military, as it has been reported that 37% of recruits develop symptoms while participating in basic training.[3] Females are reported to be at higher risk for the development of PFP than their male counterparts.[2] The problem of PFP is highlighted by the fact that 70% to 90% of individuals with this condition have recurrent or chronic pain.[4]

While interventions for PFP have shown positive short-term outcomes, long-term clinical outcomes are less compelling. This is illustrated by the fact that 80% of individuals who completed a rehabilitation program for PFP still reported pain, and 74% had reduced their physical activity at a 5-year follow-up.[1] The apparent lack of long-term success in treating this condition may be due to the fact that the underlying factors that contribute to the development of PFP are not being addressed. While it is generally agreed that the etiology of PFP is multifactorial in nature, it is our contention that the root causes of this condition are not well understood.

The mission of the second International Patellofemoral Pain Research Retreat was to bring together scientists and clinicians from around the world who are conducting research aimed at understanding the factors that contribute to the development and, consequently, the treatment of PFP. The retreat was held in Ghent, Belgium and was hosted by the Department of Rehabilitation Sciences and Physiotherapy at Ghent University.

A call for abstracts was made in the fall of 2010. All abstracts were peer-reviewed for scientific merit and relevance to the retreat. In the end, 30 abstracts were accepted for podium presentations, and 19 were accepted as posters. In total, 50 individuals from 9 countries participated in the meeting.

The format of the 2.5-day retreat included 2 keynote presentations, interspersed with 6 podium and 4 poster sessions. The presentations were grouped into 3 mechanistic categories (local factors, distal factors, and proximal factors) and 1 clinical category (interventions). Presentations in the local factors session were studies that focused on the contri-

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bution of patellofemoral joint mechanics and surrounding tissues to PFP. Presentations in the distal factors session were dedicated to research on the contribution of foot and ankle mechanics to PFP. Presentations in the proximal factors session focused on understanding how the hip, pelvis, and trunk may contribute to patellofemoral joint dysfunction. Finally, presentations in the intervention session addressed research related to clinical outcomes associated with various interventions for PFP.

The keynote presenters for the retreat were chosen for their clinical and scientific contributions in the area of patellofemoral joint dysfunction. Christopher Powers from the University of Southern California gave the first keynote address, "Mechanisms Underlying Patellofemoral Joint Pain: Lessons Learned Over the Past 20 Years." The second keynote presenter was Jenny McConnell from Sydney, Australia, whose talk was titled "Knee Pain: Where Does It Come From?" These keynotes provided the platform for rich discussion and debate throughout the remainder of the meeting.

An important element of the retreat was the development of consensus statements that summarized the state of the research in each of the 4 presentation categories. Group leaders were selected, and these individuals were charged to take notes on points of consensus during the presentations and ensuing discussions. At the end of the meeting, participants were divided into 4 groups based on their area of interest. Each group was then asked to summarize the state of the research in their area by addressing 2 questions: What have we learned to date? Where do we need to go in the future? The groups also were instructed to include references to support their consensus statements. Following the individual group meetings, the consensus statements were discussed and debated with the entire group. Following the conclusion of the meeting, the consensus statements were refined and distributed to participants for final editing and approval.

In the following pages, you will find the consensus documents from the meeting. The statements should be viewed as the state of present thought based on current knowledge, with the realization that these documents will evolve with time. It is our hope that this summary will promote ideas for future research studies to advance our knowledge in this area.

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Consensus Statement

I. LOCAL FACTORS

The source of patellofemoral pain (PFP) is believed to be multifactorial. The obvious factors are those that can be defined as having a direct pathway to pain (eg, ligament tears, arthritis, acute trauma, bone bruise, stress fractures, patellar replacement, or total knee replacement). Once the standard sources of pain are ruled out, a large percentage of patients remain with what can only be termed as having "chronic idiopathic PFP." Due to the high prevalence of idiopathic PFP,[7,27,56] much research has been focused on trying to identify the true sources of this pain. At the current time, the primary theory is that patellofemoral malalignment and maltracking (pathomechanics) result in PFP. One potential pathway to pain is that patella malalignment/maltracking overloads the subchondral bone, resulting in pain. This theory has been substantiated by a recent study that demonstrated a direct correlation between the level of pain and patellofemoral kinematics.[53] Another potential pathway to PFP is that patella malalignment/maltracking leads to a shortened lateral retinaculum and/or ischemia, with eventual secondary nerve changes resulting in pain.[46] An alternative theory, the tissue homeostasis model, has been proposed by Dye.[17] This theory states that a loss of tissue homeostasis at the patellofemoral joint, resulting from pathophysiological processes (eg, an inflamed synovial lining and fat pad tissues, retinacular neuromas, increased intraosseous pressure, and increased osseous metabolic activity), accounts for idiopathic PFP. Although this theory has been presented as exclusive to potential patellofemoral pathomechanics, it remains an expert opinion (level of evidence, 5), and it is highly likely that patella malalignment/maltracking underlies the loss of tissue homeostasis leading to PFP.

What Have We Learned?

1. The patella acts as a dynamic lever[65] for the quadriceps musculature and experiences some of the highest loads of any structure in the human body (0.5 times body weight for walking[34,43] to over 7 times body weight for squatting[33]). Because this lever's fulcrum (center of patellofemoral contact)[50] changes with knee angle and activity, the relationship between the quadriceps forces and the torque it produces likewise changes with knee angle and activity.
2. PFP can arise from any innervated patellofemoral joint structure[18] and a combination of innervated tissues may be involved concurrently.[6,22,31] These structures include subchondral bone, infrapatellar fat pad, quadriceps tendon, patellar tendon, synovium, the medial and lateral retinaculum,[23,45] and patellar (medial and lateral) ligaments. Although cartilage is aneural, the forces applied are passed to the innervated subchondral bone.
3. Pain is subjective, thus the importance of psychological state cannot be overstated.[13,26,40]
4. Proprioception appears to play an important role in the dynamic stability of the patellofemoral and tibiofemoral joints,[11] and a decrease in proprioception has been noted in patients with posttraumatic patellar dislocation.[29]
5. Although there are competing theories as to the source of idiopathic PFP, few variables have been directly correlated with pain. A recent study demonstrated a correlation between patellofemoral kinematics (change in varus rotation during extension) and pain intensity on an average day (r = 0.56).[53] Another study documented a correlation between a measure of bone metabolic activity and the highest level of pain experienced in the previous year (r = 0.55).[15] The latter study excluded patients with pain who demonstrated no bone metabolic activity, thus the strength of the correlation is suspect. Last, 1 study documented a significant (P = .04) correlation between pain and mean innervated area in the lateral retinaculum.[44] The study size was small (n = 13) and a correlation coefficient was not provided, but with a P value close to .05, it is most likely that the correlation was weak.
6. Patellar and femoral bone shape and the amount of patellar engagement in the femoral trochlea sulcus influence patellofemoral kinematics. Specifically, a low lateral trochlea inclination angle has been associated with excessive lateral shift and patellar dislocation,[2] whereas a high lateral trochlea inclination angle has been correlated to medial patellofemoral shift and tilt in patients with PFP (r = 0.48 and 0.57, respectively) and controls (r = 0.35 and 0.61, respectively).[24] When the percentage of patellar to trochlear cartilage overlap is less than 30%, the patella tends to sublux.[36]
7. Increased subchondral bone metabolic activity has been demonstrated in individuals with idiopathic PFP.[15,37]

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The study with the largest population[37] found that 44% (48/109) of the knees experiencing patellofemoral pain had increased metabolic activity.

8. The medial patellofemoral ligament has been identified as the strongest static patellofemoral joint stabilizer in early knee flexion (0°-30°), contributing 50% to 60% of the passive resistance to lateral patellar motion in this range.[38]

Maltracking

9. Maltracking occurs in a subset of patients with PFP and potentially progresses toward patellofemoral osteoarthritis.[58] In certain patients, maltracking is likely the primary impairment leading to repetitive patellofemoral joint cartilage overload from the continuous impact load as the patella re-engages with the femur.[53] This, in turn, overloads the underlying subchondral bone, resulting in pain. Alternatively, maltracking can damage the ligaments of the patellofemoral joint, potentially leading to increased innervation and pain.[45]
10. There is a large amount of intersubject variability in patellofemoral joint kinematics. However, based on recent 3-dimensional studies, there is general agreement that the patella extends and moves proximally as the tibiofemoral joint extends.[4,32,48,61] One potential source for this intersubject variability is the likely presence of subgroups[47,53] within the general population of individuals experiencing maltracking. In addition, variability in the anatomical references used to define the kinematics can result in large inconsistencies (eg, measuring the patellar tilt angle in an axial image 10 mm above versus 10 mm below the patellar center results in a 27° change in this angle).[54]
11. The specific type of maltracking pattern likely alters the pathway to pain and can influence the effectiveness of interventions.[14]
12. Recent 3-dimensional patellofemoral kinematic studies[53,61] have documented that maltracking exists outside the axial plane (eg, patella alta, flexion, and varus rotation).

Loading of the Lower Limb

13. The manner in which the lower limb is loaded affects patellofemoral kinematics.[35] For example, there is evidence to suggest that in weight bearing, patellofemoral malalignment and/or maltracking may be the result of internal rotation of the femur as opposed to lateral tilt/displacement of the patella.[42,55] Conversely, patellofemoral malalignment/maltracking in non–weight bearing is the result of the patella moving on a relatively stable femur.[42,55]
14. Increased quadriceps force tends to exacerbate pathological patellofemoral kinematics.[9,28,42] For this reason, it has been stated that "radiographic examination under static conditions can be misleading."[47]
15. During weight-bearing exercises, the quadriceps force decreases as the knee extends into terminal extension, whereas the opposite occurs for non–weight-bearing exercises.[25] Specifically, when standing with the knee fully extended, there is minimal required active quadriceps force, and this force requirement increases with increasing flexion. In sitting, with the knee at 90° of flexion, there is no required active quadriceps force and this force requirement increases as the knee extends.[25] Thus, in weight-bearing terminal knee extension, there are minimal loads on the patella and patellar maltracking is often not observed.[42,61] Yet, in deep knee flexion (greater than 60°) during weight bearing, maltracking has been observed in individuals with chronic patellofemoral pain.[61] This is due to the distal widening of the femoral groove and the high quadriceps forces on the patella in this range of motion.
16. As the axial plane kinematics tend to "normalize" once the patella engages with the femoral sulcus,[53,61] documented patellar maltracking in full knee extension during non–weight-bearing exercises may serve as a marker of altered patellofemoral joint contact stress in deeper flexion.

Patellofemoral Cartilage Contact

17. Patellofemoral joint pressure distribution has been studied using pressure-sensor films in vitro, and it has been reported that patellofemoral contact force, contact area, and maximum peak pressure rise with increasing flexion angles in cadaveric specimens with loaded quadriceps.[64]
18. Peak cartilage thickness in healthy adults has been reported to range from 4.5 to 5.5 mm for the patella and 3.5 to 4.0 mm for the femur,[16,19] indicating that submillimeter accuracies are necessary to keep the errors in estimating patellofemoral contact kinematics within acceptable limits. Few techniques that can noninvasively quantify in vivo patellofemoral kinematics/alignment have reported accuracies to such a level.[3,5,21]
19. Based on modeling studies, patellofemoral joint and cartilage stress is significantly greater in individuals with PFP compared to controls.[20]

Bracing and Taping

20. Patellar taping has been shown to reduce pain[60] and alter patellofemoral kinematics.[14]
21. Taping has been shown to improve knee joint proprioception in individuals experiencing PFP who were rated as having poor proprioception,[11] whereas bracing has been shown to influence the somatosensory inflow from the skin around the knee.[57]
22. In individuals with PFP, an improvement in the control of the tibiofemoral joint with both bracing and neutral patella taping has been demonstrated.[51]
23. Taping has been shown to reduce the amount of superior translation[14] of the patella in extension, which would likely lead to increased contact area. Additionally, the change in lateral shift, lateral tilt, and varus rotation

with taping has been demonstrated to be dependent on the value of these kinematic parameters in the untaped state.[14]

Alterations in the Quadriceps

24. Quadriceps weakness and atrophy[30] and vastus medialis obliquus (VMO) atrophy[39] have been associated with idiopathic PFP, but evidence to the contrary has also been reported.[10]
25. Although impaired VMO function (as assessed by EMG signal magnitude and timing) has been implicated in PFP,[12,59,63] this finding has not been consistent across all studies.[8,41]
26. Recent in vivo work demonstrated that the largest component of the vastus medialis moment arm relative to the patella center of mass is in the anterior direction, with a secondary component in the superior direction.[62] Thus, for every unit force within this muscle, the largest torques will result in patella varus rotation, with the secondary torque resulting in medial tilt. The same holds true for the vastus lateralis, with the largest torque resulting in valgus rotation with secondary torque production causing lateral tilt.
27. Recent work comparing the in vivo patellofemoral kinematics before and after a motor branch block to the VMO demonstrated that a loss of force in the VMO could explain some, but not all, of the kinematic changes typically observed in patients with PFP.[52] This confirms the speculations of an earlier anatomical study.[1]

Where Do We Need to Go in the Future?

1. It is widely accepted that PFP is multifactorial and that individuals can arrive at a painful state through multiple mechanisms. Yet, there remains no consensus on causative relationships between chronic idiopathic PFP and any of these mechanisms. Thus, future studies must work at developing a direct link between tissue stress and pain. Specific attention should be given to tissues that are the likely sources of pain.
2. As part of providing this direct link, future studies evaluating potential factors leading to PFP should focus on obtaining a large database of individuals with pain (eg, greater than 50) along with an appropriate control group of the same size, to capture a true representation of the spectrum of individuals experiencing PFP. These studies should make every attempt to explain the pathway to pain for each subject (or subgroup of patients) within the study, as opposed to assuming that individuals who do not fit within the primary theories being tested are "outliers" and can be eliminated from the analysis or ignored in discussing the results.
3. It has been hypothesized that "periodic short episodes of ischemia due to vascular bending" could be 1 source of PFP.[46] Clinically, this may be related to a subgroup of patients whose pain is associated with low environmental temperatures and poor rehabilitation outcomes.[49] Further work is needed to substantiate these hypotheses.
4. Because PFP can arise from surgery and other injuries, the relationship of the pain experienced by these individuals to that of individuals with idiopathic PFP should be investigated.
5. Future studies need to include clear definitions of the eligibility criteria (eg, idiopathic pain, traumatic onset of pain, pain on activity, previous surgery, total knee replacement, previous history of dislocation, instability without any history of dislocation, PFP following other specific knee injuries), along with justifications for the inclusion/exclusion criteria.
6. Because it is known that the level of quadriceps force, as well as the knee angle, affects patellofemoral bone and cartilage contact kinematics, studies focused on exploring the pathomechanics of PFP should do so under dynamic conditions, with high quadriceps loads, in regions of the greatest patellofemoral instability.
7. Clinical diagnostic tests need to be developed that differentiate the potential pathological parameters that lead to PFP. Specifically, a system of relatively simple clinical tests for the classification of patients needs to be developed to facilitate targeted patient-specific treatment options. As part of this, the relationship between complex imaging and modeling techniques should be related to more available clinical measures.
8. Because in vivo measures of patellofemoral cartilage stress are unavailable, there is a need to develop neuromusculoskeletal computational models that are validated and accurate to provide greater assessment of contact mechanics under physiological loading conditions. As this area continues to advance, proper validation, accuracy, and sensitivity studies will be crucial to maintain clinical relevance.
9. The current image-based alignment and kinematics assessment methodologies need to be transferred to the clinic. Relationships must be developed that explain the variation across experimental paradigms (eg, weight bearing versus non–weight bearing, static versus dynamic). In each of these, a clear understanding of accuracy, precision, and repeatability is required. In addition, a clear, consistent definition of the anatomical references used to define the kinematics is essential for reducing variability and enhancing cross-study comparisons.[9,54]
10. Further development of imaging modalities (eg, MR spectroscopy, water-fat differentiating MRI, PET, CT), as well as other tools, that will enhance the diagnosis of underlying mechanisms of PFP is needed.
11. Metrics of maltracking should consider the underlying geometry of the articulating surfaces to infer the influence of maltracking on contact areas and joint stress.

12. Long-term, prospective studies are needed to investigate the long-term sequelae of PFP.
13. The interrelationships between proximal, distal, and local factors need to be better understood.
14. Taping and bracing have been shown to improve proprioception at the knee.[11] Future work is needed to determine if this improvement in proprioception can be directly correlated with improved knee function or a reduction in PFP.

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II. DISTAL FACTORS

What Have We Learned?

Relationship Between Distal Factors and PFP

1. Since the first consensus statement, there has been an increase in the amount of research investigating distal contributions to PFP. However, knowledge regarding causative relationships remains limited due to a dearth of prospective studies.[5]
2. A systematic review of 24 case-control studies found that individuals with PFP tend to have delayed peak rearfoot eversion and a greater amount of rearfoot eversion at heel strike during walking and running, as well as less rearfoot eversion range during running.[5] However, it is unclear how these kinematic variations relate to patellofemoral joint loading and subsequent pathology, and whether they represent etiological factors or compensatory strategies in response to pain.
3. A prospective study identified a non–gender-specific increase in midfoot mobility when moving from subtalar joint neutral to static relaxed stance (navicular drop) as a risk factor for developing PFP in military trainees.[9,10]

Case-control studies have also reported greater midfoot mobility in patients with PFP, measured when moving from non–weight bearing to static relaxed stance (midfoot height),[19] and as navicular drop.[3]

4. There is emerging evidence of a relationship between rearfoot eversion and tibia and hip motion in PFP. Peak rearfoot eversion has been shown to be positively correlated with peak tibia internal rotation in PFP (but not controls), while greater rearfoot eversion range of motion was also positively correlated with hip adduction range in both PFP and controls.[2] This has implications for patellofemoral joint loading because both tibia internal rotation and hip adduction are likely to increase dynamic knee valgus (medial knee collapse) and patellofemoral joint stress.
5. Static measures of foot posture appear to be an inadequate representation of dynamic foot function.[4] Static alignment measures have not been identified as risk factors for PFP development.[25,27] However, measures of foot mobility can distinguish between PFP and controls.[19]
6. Based on recent findings of reduced dorsiflexion in runners with a history of PFP, it has been proposed that increased rearfoot eversion during running may be a mechanism to unlock the midfoot and allow a compensatory increase in midfoot dorsiflexion.[18] The resulting reduced ability to resupinate the foot during late stance to form a rigid lever may prolong dynamic knee valgus.
7. While research has continued to focus on foot pronation, other variations in foot posture may be associated with PFP, particularly in different at-risk populations. A prospective study reported that a more lateral rollover pattern of plantar pressure was a risk factor for PFP development in predominantly male military recruits,[25] which may suggest a predominant gait pattern in these individuals.

Foot Interventions for PFP

8. There is growing evidence that foot orthoses are efficacious for PFP in the short term (ie, 6-8 weeks).[1,7,12,15,17,20,23,24] However, the mechanism by which orthotics reduces PFP remains unclear.
9. Foot orthoses have been shown to improve functional performance immediately[8] and in the medium term (12 weeks)[7] in persons with PFP, and appear to have similar immediate effects in patellofemoral joint osteoarthritis.[13]
10. Those with PFP tend to report that soft foot orthoses are more comfortable,[20,21] which may have implications for compliance and, therefore, efficacy.[16,22]
11. Clinical predictors of successful outcome with foot orthoses for PFP include greater midfoot (width) mobility under load,[20,26] lower baseline pain severity,[6,26] as well as less ankle dorsiflexion and wearing of less supportive shoes.[6] Furthermore, the strongest predictor of success at 12 weeks was found by 1 study to be an immediate reduction in pain during a single-leg squat,[6] suggesting that modifying orthoses to enhance functional performance should be a consideration during prescription.
12. A recent case series trained rearfoot-strike runners with PFP to land with a nonrearfoot-strike pattern, and found changes in their running foot-strike pattern for 3 months, reduced vertical impact peak and loading rates, and improved PFP symptoms.[11]

What Are Some of the Challenges in Furthering Our Knowledge of Distal Factors?

1. Accurate 3-dimensional measurement of foot motion remains one of the greatest challenges. Inconsistent definitions of segments within multisegment foot models may, in part, explain some of the discrepancies between studies.
2. Advances in imaging techniques that have enabled more sophisticated evaluation of hip and knee motion have limited applicability at the foot and ankle, due to the complex anatomy of multiple bones and joints of varying orientations. Two-dimensional imaging is, therefore, insufficient. Biplanar fluoroscopy equipment may not adequately capture joint motion.
3. Current techniques limit accurate measurement of intrinsic foot muscles, which may be important in controlling foot motion. Unlike the knee and hip, insertion of fine-wire electrodes into intrinsic foot muscles is likely to cause discomfort and alter gait patterns.
4. Challenges in recruiting large samples for prospective studies have led to predominantly military cohorts, which may not be generalizable to the general population due to higher proportions of males, screening for musculoskeletal anomalies, and excessive loading.
5. The traditional paradigm that foot orthoses work via mechanical alterations has expanded to include alternative paradigms of shock attenuation, neuromuscular effects, proprioceptive input, or placebo. Evaluation is limited by difficulties with measuring these entities, particularly while wearing foot orthoses.
6. In light of this, placebo interventions for foot orthoses that have been utilized in previous studies might have been ineffective due to their primary goal of controlling for mechanical effects of orthoses. However, it is difficult to develop an effective placebo for foot orthoses when their mechanism of effect is unclear.

Where Do We Need to Go in the Future?

1. There remains a strong need for the development of a model of how altered foot function affects the patellofemoral joint.
2. Patterns of coupling and variability between the foot and ankle and more proximal kinetic-chain components (knee, hip) need to be established with respect to the development of PFP.
3. The contribution of the midfoot should be investigated

further, particularly with respect to midfoot mobility (midfoot height and width).

4. The focus of future studies should expand beyond pronation and consider how a more supinated foot type or a lack of ankle dorsiflexion may contribute to PFP.
5. There needs to be ongoing development of simple, reliable, and valid clinical measures of foot alignment and function that represent dynamic foot function. This will help bridge the gap between the laboratory and clinic, and aid decision making regarding foot orthosis prescription for patients with PFP.
6. To determine the influence of dynamic foot function on the patellofemoral joint, laboratory-based gait analysis requires development of validated multisegment kinematic foot models that consider the forefoot and midfoot, and methods of measuring patellar motion. Technologies such as standing MRI would allow more accurate visualization of bony movement (eg, during single-leg squat). Collaboration with biomechanists and engineers would be beneficial to enhance foot measurement.
7. More sophisticated methods of measuring intrinsic foot muscles, as well as tibialis posterior, are required.
8. Future studies should focus on prospective designs in at-risk populations and the general community, to strengthen knowledge regarding distal risk factors for PFP and to provide evidence for whether foot interventions (eg, foot orthoses, intrinsic exercises) are worthwhile to prevent PFP in those at risk. More studies utilizing both male and female participants, and evaluating the effect of age on the foot's role in PFP, are also required.
9. Additional work is required to re-evaluate how foot orthoses are prescribed for PFP. Given known short-term effects on pain and their relatively low expense, orthoses may be best used as an interim modality (eg, to facilitate pain-free exercise), and may produce greater effects if used as an adjunct intervention (eg, with exercise)[27] or in a different capacity (eg, as a proprioceptive tool).
10. More emphasis needs to be placed on the orthosis-footwear interaction, and whether orthoses are redundant if good supportive footwear is worn.
11. Published clinical prediction rules for foot orthosis success, which are post hoc and preliminary in nature, need to be validated.
12. Future research should focus on perceptions and characteristics of the person, rather than the device, particularly characteristics of those with PFP (eg, kinematics, kinetics) who respond to foot orthoses. This may help enhance orthosis design.
13. The clinical efficacy of custom foot orthoses and methods of retraining foot function (eg, intrinsic foot exercises, barefoot running, gait retraining) needs to be further evaluated, ideally in randomized clinical trials with a natural history control.

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III. PROXIMAL FACTORS

What Have We Learned?

1. Findings from the current literature suggest that females with PFP demonstrate altered hip kinematics during more demanding tasks like running, jumping, and landing. Data from investigations that used either 2-dimensional or 3-dimensional analysis showed increased frontal plane hip motion.[8,13,22] Furthermore, findings from investigations that used 3-dimensional analysis showed altered transverse plane motion. Some researchers reported increased hip internal rotation,[18] whereas others found greater external rotation.[22,23] Inconsistent data may reflect compensatory strategies among tasks or different measurement techniques.
2. Results from cadaveric[12] and magnetic resonance imaging[14,16,17] studies have shown that excessive femoral internal rotation increases lateral patella displacement/tilt and patellofemoral joint stress.
3. Females with PFP demonstrate hip abductor and external rotator weakness compared with healthy females.[15] However, findings from prospective studies have not identified hip weakness as a possible risk factor.[5,11,20] It remains elusive if males with PFP exhibit a similar pattern of hip weakness.
4. Preliminary data suggest that hip abductor and extensor endurance and fatigue may be a more important contributor to altered hip kinematics during demanding tasks like running.[8,19,21] However, the confounding nature of pain during fatiguing tasks requires further investigation.[8,21]
5. Emerging evidence suggests that individuals with PFP have altered gluteus medius and gluteus maximus neuromuscular activity during different activities like running, landing, and stair stepping.[1,4,6,7,18,24]
6. Individuals with PFP may benefit from hip strengthening exercise.[3,9,10] However, additional data are needed to understand if benefits result from improvements in hip strength or neuromuscular activity.[13,25]

Where Do We Need to Go in the Future?

1. Comprehensive studies are needed to better understand the interrelationships among hip muscle performance (eg, strength, endurance, and neuromuscular activity), kinematics, and kinetics (collectively referred to as neuromechanics) in individuals with and without PFP.
2. Altered trunk function may adversely affect lower extremity mechanics.[7,23] However, additional studies are needed to determine the effect that altered trunk function may have on patellofemoral joint loading.
3. Researchers need to establish a standard method for assessing hip and trunk muscle strength and endurance to allow more meaningful comparisons between study results. The chosen method should account for the following: type of muscle contraction, use of a static or dynamic test, subject position, measurement device (eg, a handheld or an isokinetic dynamometer), type of applied resistance, and normalization method.[2]
4. Further work is needed to better understand trunk and hip neuromuscular activity during functional tasks (eg, stair stepping, running, jumping, landing) in individuals with PFP. This includes a need to establish a standard method for collecting, processing, and reducing electromyographic data.
5. Clinical prediction rules are needed to identify a subgroup of individuals who may have developed PFP from altered hip neuromechanics.
6. Investigations are needed to examine changes in hip and trunk strength and neuromuscular activity during functional activities following rehabilitation exercise. Findings from these studies will provide important information as to whether clinicians develop and implement interventions focusing on hip and trunk strength, neuromuscular re-education, or a combination of both.
7. Comprehensive (ie, simultaneous assessment of strength, neuromuscular activity, kinetics, and kinematics) prospective studies are necessary to identify trunk and hip risk factors that may contribute to patellofemoral joint pathology.

8. Additional information is needed to determine the influence altered hip, pelvic, and lumbar spine range of motion and flexibility may have on PFP.
9. Future research should examine sex differences in hip and knee neuromechanics in individuals with PFP. If sex differences exist, then sex-specific interventions may be indicated for this patient population.

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IV. INTERVENTIONS

What Have We Learned?

1. There have been approximately 11 published randomized clinical trials (RCTs) on therapeutic interventions for PFP since 2009. There have been several other nonrandomized clinical trials also assessing the efficacy of therapeutic interventions.
2. On the ISRCTN clinical trials register, there are 5 trials studying bracing, taping, advice, exercise, and insoles on PFP.
3. There remain several difficulties in conducting robust clinical intervention trials on PFP. These are primarily small sample size, threat of bias from participants, and assessors or therapists not being blinded to treatment allocation. The most difficult of these is blinding of the treating clinician. Although the RCT is usually regarded as the most robust way to evaluate a therapeutic intervention, it is not necessarily ideal to address the problems associated with complex therapeutic intervention trials.
4. Despite the myriad biomechanical, electromyographic,

and gait outcome measures in intervention trials, there remains a lack of knowledge of normative data, and the known range of abnormal values in PFP. Furthermore, there are many variations in the data collection procedures, limiting the interpretation of outcome data.

5. Standardizing outcome measures in terms of the measures themselves and the time points at which the intervention is evaluated remains problematic. For example, although there are many clinical trials evaluating patellar taping, it is difficult to get an overall picture of treatment efficacy due to our inability to pool data.[1]
6. There is evidence that treating hip muscle weakness can reduce PFP.[3,4,6]
7. There is limited evidence that interventions, such as taping,[8] bracing of the knee,[5,7] exercise,[3,8] orthotics in combination with physiotherapy,[2] can help PFP when measured by self-reported questionnaires or a visual analog scale. The relationship between improved pain and altered lower-limb biomechanics, kinetics, and kinematics or muscle activity is still not clear. Bracing may help prevent PFP.[9]

Where Do We Need to Go in the Future?

1. Gait training and muscle re-education are new areas for intervention research.
2. New intervention trials may need to consider broad subgrouping of subjects, so that the intervention is more targeted and may increase the power of the trial.
3. These subgroups might include foot posture, patellar alignment, muscle weakness and flexibility, as well as patient fear, psychology, and the menstrual cycle, which may all affect treatment efficacy. Stratifying by gender may also provide a clearer picture of the efficacy of treatment.
4. Prior to intervention trials, cohort studies will need to establish if these subgroups exist and if they can be identified in clinical practice.
5. Finally, there are effective nonsurgical interventions, but they are not as effective as we would like them to be. The pooled data of Collins et al[2] and van Linschoten et al[8] (n = 310) showed that 40% of the patients still reported persistent complaints 1 year postintervention. Therefore, studies with at least a 1-year follow-up may give a different impression of treatment efficacy.

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Keynote Addresses

Mechanisms Underlying Patellofemoral Pain: Lessons Learned Over the Past 20 Years

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Although patellofemoral pain (PFP) is recognized as one of the most common lower extremity disorders encountered in the general population, the etiology and treatment of PFP remain controversial. Over the past 20 years, our group has taken a multidisciplinary approach to better understand the pathogenesis of patellofemoral joint dysfunction. To gain insight into this complicated clinical condition, our studies have included the use of dynamic imaging, kinematic and kinetic analyses, cadaveric measurements, biomechanical modeling, and clinical assessments. This lecture will touch upon some of our contributions in these areas.

It has been hypothesized that abnormal loading of the patellofemoral joint is an important factor with respect to the genesis of PFP.[7] This premise is supported by the clinical observation that PFP typically is reproduced with activities that require quadriceps contraction (ie, squatting, stair ascent/descent, etc). To test this hypothesis, we developed a patient-specific, imaging-based biomechanical model of the patellofemoral joint to quantify patellofemoral stress during functional tasks.[13] We were able to demonstrate that females with PFP exhibit higher patellofemoral stress during walking when compared to pain-free controls. Interestingly, the higher patellofemoral stress in the PFP group was the result of diminished contact area as opposed to an increase in the joint reaction force.[13] Based on these findings, we concluded that diminished contact area may be an important etiologic factor underlying PFP.

Evidence in support of the concept that reduced contact area may play a contributory role with respect to elevated patellofemoral stress and PFP is provided by previous research evaluating the mechanism by which patellofemoral braces reduce patellofemoral symptoms.[24,25] Given the fact that kinematic MRI has revealed that patellofemoral bracing does not improve patella tracking under dynamic, loaded conditions,[23] we proposed that patellofemoral braces may achieve symptom reduction by increasing contact area. In theory, the compressive forces applied to the patella by means of sleeves and stabilizing straps may serve to better seat the patella in the femoral tochlea, thereby increasing contact area. We were able to demonstrate that application of patellofemoral bracing resulted in significant increases in contact area (30%-40%), without an appreciable change in patella alignment.[24] Importantly, this translated to a 27% reduction in patellofemoral stress during walking and a 56% reduction in pain.[25]

Over the past 5 years, our modeling efforts have expanded to examine the influence of excessive joint loading on patella cartilage stress. Using a subject-specific 3-D model to quantify patellofemoral joint reaction forces,[4] and finite-element methods to quantify stress,[10] we have been able to show that females with PFP exhibit elevated hydrostatic and shear stress in articular cartilage. We also have reported that females with PFP exhibit thinner cartilage and reduced deformational behavior following an acute bout of loading.[9] Given the cross-sectional nature of these studies, however, it is not possible to ascertain if reduced cartilage thickness was a cause or an effect of high cartilage stress. However, these findings suggest that elevated joint stress is associated with cartilage changes consistent with initial pathologic findings in the development of patellofemoral osteoarthritis.

Given the lack of nerve fibers, articular cartilage cannot be a source of PFP. However, subchondral bone is innervated, and this is thought to be the primary source of retropatellar pain. Ongoing research in our laboratory is focused on evaluating the transfer of stress from the articular cartilage to subchondral bone. Preliminary findings are showing that persons with PFP and high cartilage stress also exhibit high bone stress.[15] We are now evaluating the responses of bone to abnormal loading, including elevated patella water content (ie, bone marrow edema) and the presence of bone marrow lesions.[14]

Given that elevated joint stress appears to underlie, at least in part, the development of PFP, it is important to identify the biomechanical factors that contribute to elevated patellofemoral stress. As mentioned above, stress is defined as force per unit area. As such, increased patellofemoral stress could be the result of diminished contact area, elevated joint reaction force, or a combination of both.

The factors that influence contact area are those that contribute to patella malalignment and/or maltracking. It is well established that patella tracking in non–weight bearing is dictated by bony structure, particularly the depth of the trochlear groove,[22] and the angle of inclination of the lateral anterior femoral condyle.[12] Conditions such as trochlear dysplasia (abnormal shape or depth of the trochlea) or patella alta (high-riding patella) can lead to excessive lateral tilt and lateral displacement of the patella,[32] decreased contact area,[32] and a subsequent increase in patellofemoral stress.[31]

Historically, patella tracking has been viewed as the relative motion of the patella on a fixed femur. This assumption, however, is based on kinematic studies that were performed non–weight bearing or under conditions in which the femur motion was constrained.

Recent evidence from our group suggests that patellofemoral joint kinematics may be different during weight-bearing tasks. For example, we have provided evidence that the primary contributor to lateral patella tilt and displacement during weight bearing is internal rotation of the femur underneath a stable patella.[26,29] These findings suggest that the control of femur rotation may be important in restoring normal patellofemoral joint kinematics. In addition, minimizing femoral rotation may impact patellofemoral joint stress, as it has been shown that excessive internal rotation of the femur can result in decreased patellofemoral contact area and increased joint stress.[17]

Apart from the influence of diminished contact area on patellofemoral joint stress, a recent publication by our group has revealed that patients with PFP exhibit higher than normal laterally directed patellofemoral joint forces.[2] Importantly, we identified the main contributors to the lateral forces as frontal and transverse plane motions at the knee.[3] This led to the conceptual framework of the dynamic quadriceps angle or Q-angle.[21] As the Q-angle reflects the frontal plane forces acting on the patella, frontal plane motion of the lower extremity would be expected to adversely affect patellofemoral joint loading. As noted in a previously published review article,[21] there are distal factors (ie, those related to the foot and ankle) and proximal factors (ie, those related to the hip and pelvis) that can influence the dynamic Q-angle.

With respect to the distal factors that can influence the patellofemoral joint, it is commonly believed that foot pronation and resulting tibia rotation contribute to PFP. This premise has formed the basis for the use of foot orthoses as a treatment for this condition. When relating excessive pronation to PFP, however, an assumption is made that abnormal pronation results in excessive tibia internal rotation. This relationship, however, has been shown to be inconsistent at best.[28] More important is the fact that excessive tibia internal rotation caused by abnormal foot pronation would actually decrease the Q-angle, as the tibial tuberosity would move medially.[21] Indeed, in vitro studies have shown that tibia internal rotation has no influence on patellofemoral joint contact area or pressures.[18] Nonetheless, there is growing evidence that foot orthoses are efficacious for PFP, at least in the short term (ie, 6-8 weeks).[1,5] However, the mechanisms by which these devices reduce PFP remain unclear.

With respect to proximal factors, excessive knee valgus resulting from hip adduction would be expected to have the largest influence on the dynamic Q-angle, as this motion influences the frontal plane alignment of the lower extremity.[21] Excessive hip internal rotation also would contribute to an increase in the dynamic Q-angle; however, its influence on lower-limb alignment would not be as great. Indeed, previous work by our group and others has shown that females with PFP exhibit greater degrees of hip adduction and internal rotation during dynamic tasks when compared to pain-free controls.[6,20,30,33]

There are an increasing number of studies suggesting that impaired hip strength (extensors, abductors, and external rotators) may underlie the tendency of females with PFP to exhibit altered hip kinematics. In fact, a systematic review of the literature in this area concluded that there is strong evidence that these individuals exhibit impaired strength of the hip extensors, abductors, and external rotators.[27] From a treatment standpoint, the focus on hip strength/control is logical from a biomechanical perspective, because control of hip internal rotation can improve patella tracking, thereby improving contact area, and the control of hip adduction can reduce the laterally directed forces on the patellofemoral joint by minimizing the dynamic Q-angle. Indeed, clinical trials evaluating the influence of hip strength on pain and function in persons with PFP are emerging to support this premise.[8,11,16,19]

In summary, several important advances have emerged over the last decade that have advanced our understanding of the potential factors that may underlie PFP:

- Elevated patellofemoral stress appears to be an important biomechanical variable associated with PFP and perhaps patellofemoral joint osteoarthritis.
- The combination of reduced contact area and elevated joint reaction forces is most detrimental with respect to patellofemoral joint loading.
- There is evidence to suggest that in weight bearing, patellofemoral malalignment and/or maltracking may be the result of internal rotation of the femur as opposed to lateral tilt/displacement of the patella.
- The lateral forces acting on the patella are largely influenced by abnormal motions of the lower extremity.
- Compared to abnormal foot pronation, altered hip kinematics (ie, excessive hip adduction and internal rotation) appear to have the greatest influence on the dynamic Q-angle.
- Individuals with PFP may benefit from interventions aimed at improving hip muscle performance.

Future work should be directed toward understanding whether hip strengthening is superior to traditional quadriceps strengthening for the treatment of PFP. Although not addressed specifically in this lecture, emerging research in the area of patellofemoral joint pathomechanics calls into question the practice of vastus medialis obliquus (VMO) strengthening as the gold standard treatment for PFP. It is my hope that this keynote address will promote new ideas for future research and advancements in clinical practice.

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Knee Pain: Where Does It Come From?

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Patellofemoral pain (PFP) is a multifactorial problem with weighting which factors (local, proximal, or distal) have the greatest significance with respect to causing the symptoms.[6] However, what seems to be missing in the debate is the source of the pain. Foot pronation, femoral anteversion, poor gluteal control, or the delay in the onset timing of vastus medialis obliquus (VMO) are not in themselves the source of PFP, even though their presence is associated with pain. The presence of pain will certainly decrease muscle activity, timing, and endurance, as well as alter movement patterns.[12] As we know, however, pain is very much a cortical experience, so extrinsic factors, such as fear of pain, stress, and anxiety,[15,20] can amplify the pain experience for the patient, and the contribution of these factors must be understood if we are to satisfactorily improve the rehabilitation of individuals with PFP. This lecture will examine some of these issues in the context of PFP.

Hodges et al[12] have reported that inducing pain in the knee of asymptomatic individuals decreases both VMO and vastus lateralis (VL) activity. But when a painful electric shock is randomly and intermittently applied to the knee of the same individuals (ie, mimicking the fear-of-pain state experienced by patients with PFP), only VMO activity is decreased. Exposure to fear and stress initiates the secretion of several hormones, including corticosterone/cortisol, catecholamines, prolactin, oxytocin, and rennin. This is part of the survival mechanism. Such conditions are often referred to as "stressors" and can be divided into 3 categories: external conditions resulting in pain or discomfort, internal homeostatic disturbances, and learned or associative responses to the perception of impending endangerment, pain, or discomfort.[20] Release of cortisol can be detrimental to a patient's recovery. In fact, it has been found that stress-related hormones can alter inner ear fluid homeostasis and auditory function.[13] This could have implications for the balance of individuals exhibiting fear/avoidance behavior, so instead of just blaming poor gluteal function for balance problems in patients with PFP, other factors may need to be considered.

The structures that may be the possible source of PFP are the synovium, lateral retinaculum, subchondral bone, and the infrapatellar fat pad. Articular cartilage is aneural and thus provides only an indirect source of pain, perhaps either through synovial irritation or increasing subchondral bone stress. Interestingly, there is no correlation between amount of articular cartilage degeneration and pain experienced by patients with osteoarthritis (OA) of the knee, with many patients with knee OA having episodic bouts of pain for years before requiring surgical intervention. The severity of OA knee pain is associated with bone marrow lesions (edema) with subarticular bone attrition,[16,18] synovitis/effusion, and degenerative meniscal tears, but is not associated with the presence of osteophytes or reduction in joint space.[16] Hill and colleagues[11] followed 270 subjects with tibiofemoral and patellofemoral OA for 30 months and found no correlation between baseline synovitis and baseline pain; however, a decrease in synovitis at follow-up correlated with a reduction in pain. These investigators found synovitis in 3 locations: superior, medial, and inferior patella, with infrapatellar synovitis being the most strongly correlated with pain severity. Synovitis was not associated with cartilage loss in either the tibiofemoral or patellofemoral compartments.[11]

Free nerve endings (IVa) are present in the synovium.[9] As such, peripatellar synovitis is a possible source of PFP. Despite the evidence supporting the synovium as a potential pain source, histological changes in the synovium of patients with PFP are only moderate.[1] However, there is evidence of histological changes in the lateral retinaculum in some patients with PFP, as shown by increased numbers of myelinated and unmyelinated nerve fibers, neuroma formation, and nerve fibrosis.[10,15,19] Additionally, increased intraosseous pressure of the patella has been found in patients with PFP who complain of pain when sitting with a bent knee ("moviegoer's knee"), possibly secondary to a transient venous outflow obstruction.[9,10] However, the structure that has largely been ignored by the orthopaedic community, even though it was first identified as a potent source of pain by Hoffa in 1904, is the infrapatellar fat pad.

The fat pad covers the extra-articular posterior patellar surface, merges superiorly with the peripatellar fold, extends into the ligamentum mucosum posteriorly, and is lined by synovium.[7] The fat pad attaches to the proximal patellar tendon, inferior pole of the patella, transverse meniscal ligament, medial and lateral meniscal horns, the retinaculum, and the periosteum of the tibia.[7] The fat pad is highly vascular and richly innervated, so it is one of the most pain-sensitive structures in the knee.[7,8] The innervation of the fat pad is linked to the entire knee joint structure, so the fat pad may be affected by pathology in various knee joint components.[7] To simulate early knee OA change, Clements and colleagues[5] injected monoiodoacetate into the right knee of 150 rats and, after 21 days of weight-bearing asymmetry, found marked inflammatory changes in the fat pad. These authors concluded that the infrapatellar fat pad may contribute to pain in the early stages of knee OA. Experimentally induced chemical irritation of the fat pad in asymptomatic individuals confirms that the pain is not just confined to the infrapatellar region but can refer to the proximal thigh as far as the groin.[2] In fact, the fat pad and medial retinaculum of patients with PFP contain a higher number of substance-P nerve fibers than the same structures in individuals without PFP.[21]

The fat pad facilitates distribution of synovial fluid, stabilizes the patella in the extremes of knee motion (ie, less than

20° and greater than 100° of knee flexion), and increases tibial external rotation.[4] A total resection of the fat pad decreases patellofemoral contact area.[4] Inflammatory changes in the fat pad seen on MRI are most commonly the consequence of trauma and degeneration, with the commonest traumatic lesions following arthroscopy, which in 50% of cases fibrous scarring can still be present 12 months later.[17] Impingement of the fat pad with diffuse edema occurs following patellar dislocation, often mimicking a loose body.[14]

An ongoing clinical trial being conducted by our group currently consists of 65 patients (mean age, 41 years; range, 14-79 years) presenting to an outpatient setting for treatment of knee pain. The patients completed a KOOS questionnaire, as well as provided detailed information about the area of their knee pain. Visual analog pain scales at rest and during activities such as walking, stair climbing, squatting, quadriceps setting, and passive knee extension were obtained. The widest diameter of the fat pad of both knees was measured with a tape measure. MRI scans were performed on a random subset of the subjects, prepatella and postpatella tape, and pretreatment and posttreatment. Treatment consisted of taping to unload the fat pad so that the patients were pain free, stretching the anterior hip structures, and weight-bearing gluteal training, and was provided weekly for the first 2 weeks, once every 2 weeks for the next 2 treatments, then once a month for the next 2 treatments. Follow-up assessments were made at 3, 6, and 12 months.

At initial presentation, 46% of the 65 patients reported pain in the inferior patellar region, 43% medial knee pain, 23% retropatellar pain, and 20% lateral pain. Fifty-three percent of patients reporting retropatellar pain also reported inferior or medial knee pain. Sixty-one percent of patients complained of pain doing a quadriceps contraction, ranging from 1 to 8 on the visual analog scale, and 45% of patients experienced pain on extension overpressure ranging from 1 to 9 on the visual analog scale. The size of the fat pad on the affected side averaged 10 cm and 8.5 cm on the unaffected side. These findings support the hypothesis that the fat pad was the source of the symptoms. At the time of entry into the trial, scans were available for 20 subjects. In 12 of these scans, the radiologists had commented on fat pad inflammation. The diagnoses of these 20 patients included ACL injury (n = 1), meniscal tear (n = 2), tricompartmental OA (n = 2), patellar tendinopathy (n = 1), patellar dislocation (n = 3), medial femoral softening (n = 3), chondromalacia patella (n = 1), and patellofemoral arthrosis (n = 7).

Ten subjects received an MRI scan prepatella and immediately postpatella taping, in which the taping had to decrease the symptoms on stairs or squatting by at least 80%. The patella was tilted out of the fat pad and the fat pad was unloaded anteriorly and posteriorly. The MRI results showed various areas of fat pad inflammation, depending on the underlying pathology. The posttaping MRI scans demonstrated an inferior patellar shift in all cases, an increase in fat pad depth, and either an anterior or posterior tibial shift, compared with the pretape condition. MRI results of another subset of subjects 6 to 12 months following cessation of treatment revealed a decrease in fat pad volume, an increase in patellar height, a medial patellar drift, and an increase in patella varus alignment, suggesting an improvement in VMO strength.

In summary, the infrapatellar fat pad is highly innervated and a probable source of knee pain. Knee pain inhibits quadriceps activity, and fear of pain specifically inhibits the VMO. Taping to unload the infrapatellar fat pad significantly reduces pain and causes an inferior patellar shift and a slight increase in fat pad depth. Physical therapy treatment results in an increase in patellar varus alignment, an increase in patellar height, a medial drift of the patella, and decreased fat pad volume, suggestive of an improved resting VMO tone.

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Podium and Poster Presentation Abstracts

PODIUM PRESENTATIONS

INCREASED PATELLA CARTILAGE STRESS WITH INTERNAL ROTATION OF THE FEMUR: EVALUATION USING FINITE ELEMENT ANALYSIS

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INTRODUCTION: Disorders of the patellofemoral joint (PFJ) are among the most common and clinically challenging conditions encountered in orthopaedic practice. Patellofemoral pain (PFP) affects a wide range of individuals, with higher incidence rates among women and those who are physically active.[1] The most commonly cited hypothesis as to the cause of PFP is related to abnormal patella alignment and/or tracking.[2] Recent studies have shown that altered PFJ kinematics is the result of abnormal femoral internal rotation as opposed to abnormal patellar motion.[3] Internal rotation of the femur with respect to the patella has been shown to decrease the contact area and increase the stress at the PFJ in cadaver knees.[4] Using finite element analysis methods, the objective of this study was to compare the hydrostatic pressure at the patella cartilage-bone interface at 15° and 45° of knee flexion at different degrees of internal rotation of the femur.

METHODS: Subject-specific PFJ geometry of 7 females with PFP was obtained from high-resolution, sagittal plane MR images acquired with a 3.0 T MR scanner (General Electric Healthcare). Weight-bearing PFJ kinematics was acquired using sagittal plane MR sequence while the knee joint was loaded with 25% of body weight at 15° and 45° of knee flexion. Quadriceps muscle morphology was assessed from thigh MR images in coronal and axial planes. For biomechanical testing, lower extremity kinematics were collected using a Vicon (Oxford Metrics LTD) 8-camera motion analysis system at 60 Hz. Ground reaction forces were recorded at 1560 Hz using 2 AMTI force plates. EMG signals of knee musculature were recorded at 1560 Hz, using preamplified, bipolar surface electrodes (Motion Lab Systems). Input parameters for the FE model included: (1) joint geometry, (2) weight-bearing PFJ kinematics, and (3) quadriceps muscle forces. To estimate quadriceps forces, a previously described subject-specific model of the PFJ was used.[5] A previously described method was used to perform quasi-static loading simulations using a nonlinear FE solver (Abaqus, SIMULIA).[6] A mixed-model (hip angle by knee angle) analysis of variance (ANOVA) was performed to examine the difference in peak and average hydrostatic pressure of the patella cartilage elements at the cartilage-bone interface. The significance level was set at .05.

TABLE Peak and Average Hydrostatic Pressure at the Cartilage-Bone Interface*

Internal Rotation	Peak Pressure	Average Pressure
Neutral	2.14 ± 0.65	0.80 ± 0.19
5°	2.88 ± 0.83†	1.14 ± 0.27†
10°	3.25 ± 1.03†	1.15 ± 0.29†

**Values are presented as mean ± SD in MPa.*
†Indicates a significant difference in hydrostatic pressure from neutral position of femur.

RESULTS AND DISCUSSION: Significant group main effects (no interactions) were found for both stress variables of interest. When averaged across knee flexion angles, 5° and 10° of femoral internal rotation resulted in significant increases in peak and average hydrostatic pressure when compared to the neutral position (**TABLE**). Besier et al[7] have reported that femoral rotation with the knee flexed at 60° increased PFJ stresses in one-third of their subjects. However, when simulating internal rotation, the patella was allowed to move with the femur. Kinematic MRI studies have shown the patella does not move with the femur as it rotates in weight bearing.[3] Our results agree with conclusions of cadaveric[4] and imaging studies[3] that femoral internal rotation is a contributor of altered PFJ mechanics in persons with PFP.

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UTILIZING A FORWARD TRUNK LEAN DURING RUNNING DECREASES PATELLOFEMORAL JOINT STRESS

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INTRODUCTION: Patellofemoral pain (PFP) is one of the most common knee joint problems among runners.[1] A commonly accepted cause of PFP is elevated patellofemoral joint (PFJ) stress.[2] As stress is defined as force per unit area, elevated stress could occur as a result of an increase in the PFJ reaction force and/or a decrease in contact area. In turn, an increase in the PFJ reaction force could occur with an increase in the knee flexion angle and/or an increase in the knee extensor moment. Recent literature suggests that sagittal plane trunk posture can have a significant influence on knee joint kinematics and quadriceps muscle activation during landing from a jump.[3] Therefore, modifying sagittal plane trunk posture may be a potential strategy to reduce PFJ stress during running. The purpose of this study was to investigate whether an increased trunk forward lean posture would lead to a reduction of PFJ stress as compared to a self-selected posture during overground running. We hypothesized that increased trunk flexion would result in a decrease in PFJ stress.

METHODS: Six healthy adults (3 females, 3 males) participated in this study. Trunk and knee kinematics (11-camera Qualysis motion-capture system; 250 Hz) as well as knee kinetics (AMTI force plate; 1250 Hz) were obtained while subjects ran over ground with self-selected and forward-flexed trunk postures at a controlled velocity of 3.4 m/s. The PFJ

TABLE Trunk and Knee Kinematics and Knee Kinetics at the Time of Peak PFJ Stress (*P* Value Based on Wilcoxon Signed Rank Test)

	Self	Flex	*P* Value
Trunk flexion angle, deg	4.9 ± 5.3	11.8 ± 5.4	.028
Knee flexion angle, deg	42.4 ± 3.2	43.3 ± 3.7	.173
Knee extensor moment,* Nm/kg	2.8 ± 0.4	2.6 ± 0.4	.046

*Normalized by subject's body weight.

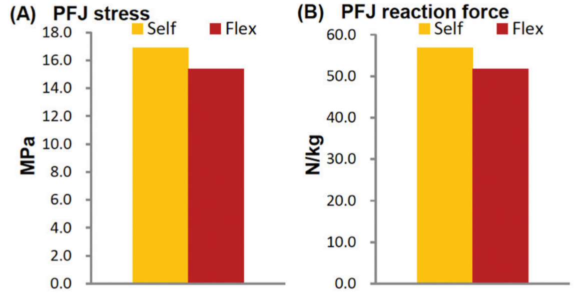

FIGURE. Peak patellofemoral joint stress (A) and joint reaction force (at the time of peak stress) (B) when running with self-selected (Self) and forward-flexed (Flex) trunk postures.

stress was estimated using a previously described biomechanical model.[2] Model input variables included subject-specific parameters (ie, knee joint kinematics, net knee joint moment) and data from the literature (ie, knee moment arms, quadriceps force/patella ligament force ratios, and joint contact area). The model outputs were PFJ reaction force and PFJ stress. Variables of interest consisted of peak PFJ stress and the PFJ reaction force, as well as the trunk and knee flexion angles and knee extensor moment at the time of peak PFJ stress. Wilcoxon signed rank tests were used to compare the differences between the 2 conditions.

RESULTS: Significant decreases in PFJ stress (P = .028) as well as reaction force (P = .028) were observed when subjects ran with the forward-flexed posture as compared to the self-selected trunk posture. The changes in flexed trunk condition were accompanied by a significant increase in trunk flexion angle (6.9°) and a significant decrease in knee extensor moment (0.24 Nm/kg). No significant changes in the knee flexion angle were observed (**TABLE**).

DISCUSSION: Our results indicate that a slight increase in trunk flexion (6.9°) can lead to a significant reduction of peak PFJ stress during running. The 9% decrease in peak PFJ stress was the result of a 9% decrease in the PFJ reaction force. In turn, the decrease in the PFJ reaction force was the result of an 8% decrease in the knee extensor moment, as no changes in the knee flexion angle were observed. Because trunk orientation can have a significant influence on the location of the center of mass and center of pressure, it is logical that a slight increase in trunk flexion can lead to a significant reduction of knee extensor moment and, consequently, PFJ stress. Future studies are needed to determine whether running with a forward trunk posture can be used to reduce PFP in symptomatic runners.

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GENDER DIFFERENCES IN HIP AND KNEE MECHANICS OF PATELLOFEMORAL PAIN SYNDROME DURING RUNNING

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INTRODUCTION: Previous studies have reported abnormal hip and knee mechanics with PFPS in female runners (F-PFPS). These include excessive peak contralateral pelvic drop (CPD), peak hip adduction (HADD), and decreased peak knee adduction (KADD). To date, there are few investigations of the mechanics of males with PFPS (M-PFPS). Previously, Dierks et al[1] found that the few males in a mixed-gender cohort demonstrated hip abduction during running, whereas F-PFPS displayed increased HADD. Similarly, in a prospective study of a male-dominant cohort, those who went on to develop PFPS exhibited increased lateral plantar pressure during gait when compared to healthy matched controls.[2] Taken together, the results of these 2 studies suggest that M-PFPS may move in greater KADD than healthy males and females with PFPS during dynamic activities. The purpose of this study was to compare the lower extremity mechanics and alignment in M-PFPS with healthy male controls (M-CON) and with F-PFPS. We hypothesized that M-PFPS would run in less CPD, less HADD, greater KADD, and greater peak knee external adduction moment (KEAM) when compared to M-CON and F-PFPS. Further, it was hypothesized that M-PFPS would demonstrate greater frontal plane mechanical axis of the tibia than both M-CON and F-PFPS.

METHODS: Data for 18 subjects between the ages of 18 and 35 years were collected per group. To qualify, M-PFPS and F-PFPS were required to have PFPS with duration ⩾3 months. All subjects were running ⩾10 km/wk. An instrumented gait analysis was conducted (Vicon, Oxford, UK; Bertec Corp, Worthington, OH) during overground running at 3.35 m/s. Data were processed using Visual 3D (C-Motion, Bethesda, MD). The frontal plane mechanical axis of the tibia (TMA) was measured using a caliper-inclinometer device.[3] Analysis of variance (ANOVA) was used to analyze the data.

RESULTS: M-PFPS (pain, 5.5 ± 2.0 out of 10; 21.7 ± 10.3 km/wk; 24.7 ± 4.9 years old), M-CON (29.5 ± 20.0 km/wk; 23.9 ± 2.7 years old), F-PFPS (pain, 4.9 ± 1.0; 24.6 ± 14.2 km/wk; 22.1 ± 3.9 years old). As hypothesized, M-PFPS ran in greater KADD and demonstrated greater KEAM than M-CON (**TABLE**). Contrary to our hypothesis, M-PFPS demonstrated greater CPD than M-PFPS. TMA was not different between groups. However, a post hoc analysis revealed that M-PFPS ran with greater peak shank segment adduction (referenced to the lab). Compared with M-PFPS, F-PFPS ran in greater HADD and less KADD.

DISCUSSION: M-PFPS exhibited greater KADD, whereas F-PFPS demonstrated increased HADD and less KADD. Greater KADD will likely decrease dynamic Q-angle, whereas increased hip adduction will likely increase dynamic Q-angle. Cadaveric studies have demonstrated that increasing and decreasing the Q-angle may both have a deleterious effect on patellofemoral joint contact area and joint stress.[4] M-PFPS also demonstrated greater CPD. Increases in both KADD and CPD have been associated with increased KEAM,[3,5] as was observed in these subjects. While the dynamic measure of peak shank segment adduction was greater in the M-PFPS group, the static measure (TMA) was not. This suggests that the greater peak KADD exhibited by M-PFPS was more related to dynamic rather than static alignment. Based on the results of this study, therapies for PFPS may need to be gender specific. Interventions for F-PFPS often focus on strengthening and neuromuscular re-education to reduce excessive HADD and increase KADD. However, interventions for M-PFPS may need to focus on decreasing KADD and KEAM. Additionally, therapies focused on decreasing CPD may provide a means to decrease KEAM and KADD in M-PFPS. Understanding gender differ-

TABLE Mean ± SD Running Mechanics, Mechanical Axis Angle of the Tibia, and Results of ANOVA

Variable	M-PFPS	*P* Value	M-CON	*P* Value	F-PFPS
CPD, deg	–3.9 ± 2.3	.002	–6.5 ± 2.2	.19	–7.7 ± 2.2
HADD, deg	11.9 ± 3.0	.39	12.9 ± 3.4	<.001	19.2 ± 3.0
KADD, deg	2.7 ± 3.2	.029	5.7 ± 1.0	.018	2.2 ± 4.0
KEAM, N·m/kg·m	0.688 ± 0.240	.041	0.543 ± 0.162	.34	0.613 ± 0.227
TMA, deg	7.8 ± 2.4	.89	7.7 ± 2.4	.429	6.5 ± 3.4
Shank adduction, deg	7.1 ± 3.1	.046	5.3 ± 1.9	.115	5.5 ± 2.5

ences will lead to the development of more optimal therapies for PFPS.

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THE EFFECT OF FATIGUE ON JOINT KINEMATICS IN FEMALE RUNNERS WITH PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) is one of the most common injuries in female runners.[1] Emerging evidence suggests that hip mechanics may be altered in this population.[2] Fatigue while running may exacerbate poor hip mechanics and lead to the development of pain. To date, though, no study has found differences in hip mechanics between those with and without PFPS when comparing between a fresh and fatigued state.[3,4] These studies, however, included a number of factors such as both genders, and different fatigue levels between groups, which may have resulted in differences in mechanics that were independent of having PFPS. Therefore, the purpose of this study was to determine the alterations in hip and knee kinematics between a group of female runners with PFPS as compared to healthy control female runners during a fatiguing run. We hypothesized that female runners with PFPS would have greater hip adduction, hip internal rotation, knee abduction, and knee external rotation in an exerted state than healthy control runners.

METHODS: Thirty-two female runners (16 PFPS, 16 healthy controls) participated in this study. Subjects in the PFPS group must have reported pain during running for at least the past 2 months and their diagnosis was confirmed by a medical professional. Subjects underwent an instrumented gait analysis while running on a treadmill for 30 minutes at their typical training pace. During every minute of the run, the subject reported their pain on a verbal analog scale, their Borg rating, and their marker trajectories and forces were recorded. The first minute of running after an initial warm-up was compared to the first point of the maximum Borg rating during the run. Data were compared using a 2-factor ANOVA (group by time).

RESULTS: Fatigue levels were similar for the control group (14.5) and the PFPS group (15.2) at the end of the run. By comparison, the PFPS group reported pain levels on average of 4.76 (range, 3-7) as compared to 0 for the control group. The results of the kinematic variables of interest are summarized in the **TABLE**. Contrary to our hypothesis, we found that the control group had greater shift in hip adduction at the end of the run as compared to the PFPS group (**FIGURE**). No significant differences were found in the remaining kinematic variables.

DISCUSSION: The purpose of this study was to determine the effect of fatigue on running mechanics between those with and without PFPS. We found that the control group increased in hip adduction at the end of the run compared to their prefatigue measures. Perhaps because the PFPS

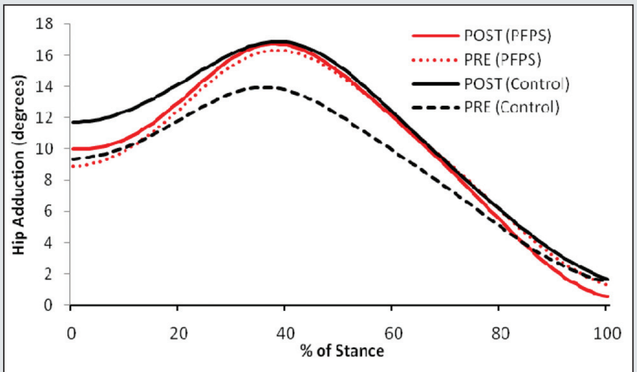

FIGURE. Ensemble hip adduction curves for the PFPS and control groups in fresh and fatigued states.

TABLE Comparison of Peak Angles (SD) Between Female Runners With PFPS and Healthy Control Runners Before and After a Fatiguing Run

	PFPS Pre	CON Pre	PFPS Post	CON Post	*P* Value
KER	1.0° (4.9)	1.6° (4.1)	3.0° (5.5)	2.9° (3.5)	.225
KABD	0.4° (3.5)	–0.3° (2.8)	0.1° (3.6)	–0.7° (2.4)	.716
HIR	9.7° (3.9)	5.1° (3.9)	8.9° (4.4)	5.0° (4.3)	.259
HADD	16.7° (3.2)	14.4° (3.4)	17.1° (4.0)	17.4° (2.9)	.006*

**Fatigue-by-group interaction.*

group started the run in greater hip adduction, they did not have the adduction shift of the control group. The PFPS group, in the absence of such a shift, also reported increased pain at the end of the run. The alteration in frontal plane hip kinematics in the contol group may help redistribute patellofemoral contact stress as the run progresses. By comparison, the PFPS group did not change their hip mechanics during the run and likely repetitively stressed the same portion of the patellofemoral joint, which may have contributed to the development of pain. Although the PFPS group also had greater hip internal rotation at the beginning of the run, neither group substantially changed at the end of the run. Perhaps, as an individual fatigues, frontal plane mechanics may be more directly affected than transverse plane, as the frontal plane is resisting gravitational forces versus rotational forces. Interestingly, we did not find differences between groups in the fresh and fatigued states at the knee. This is consistent with reports from other investigators who have examined the knee in those with PFPS.[3,4] The lack of hip adduction shift in the PFPS group suggests that these individuals lack the ability to adapt to changing internal conditions. These results suggest that rehabilitation specialists should consider focusing on hip neuromuscular control to develop effective strategies to cope with change versus standard hip strengthening and endurance exercises for those with PFPS.

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KINEMATICS AND MUSCLE ACTIVITY DURING SINGLE- AND DOUBLE-LEG SQUATS IN PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Patella malalignment is one of the most common etiological theories for patellofemoral pain syndrome (PFPS).[1] Changes in kinematics (eg, excessive hip internal rotation) and alterations in muscle activation such as decreased vastus medialis obliquus (VMO) activity are proposed to lead to patella malalignment and PFPS symptoms. However, studies have found variable alterations in kinematics and muscle activity in subjects with PFPS.[2-5] The aim of this study was to investigate whether subjects with PFPS have altered hip, knee, or ankle joint motion or EMG signal in the quadriceps, hamstrings, gluteus medius (GMed), and gluteus maximus (GMax) muscles during single- and double-leg squats.
METHODS: A convenience sample of 23 healthy controls (15 females; mean ± SD age, 30.6 ± 6.6 years; height, 1.70 ± 0.08 m; mass, 66.5 ± 10.1 kg) and 25 subjects with PFPS (20 females; age, 30.0 ± 7.6 years; height, 1.70 ± 0.06 m; mass, 71.3 ± 12.7 kg) were recruited. The subjects with PFPS had no coexistent pathology. Other inclusion criteria were subjective reports of knee pain on at least 2 of the following activities: prolonged sitting, squatting, kneeling, ascending/descending stairs, or running. Light-emitting diodes were placed on the lower limb and data were acquired using a 3-D movement analysis system (CODA mpx30). Measurements of joint motion were taken while the subjects performed 8 trials of single- and double-leg squats. Surface EMG from the VMO, vastus lateralis (VL), rectus femoris (RF), hamstrings, GMax, and GMed was recorded simultaneously, using pairs of active silver/silver chloride electrodes (Biopac). The EMG data were rectified, smoothed, averaged across trials, and normalized to the maximum EMG data during each squat. The data were reported at 10° intervals of knee flexion and an average EMG value for descent and ascent was calculated. Using Mathcad software, the data were analyzed and group differences in joint range and normalized EMG signal were assessed with either independent *t* or Mann-Whitney tests using SPSS software ($P \leqslant .01$).
RESULTS: No group differences were found in joint range during single- and double-leg squats. During single-leg squats, normalized GMax activity was higher in subjects with PFPS (**FIGURE**), reaching significance for average descent and at joint angles of 20°, 50°, and 60° of knee flexion during descent and at 50° during ascent ($P \leqslant .01$). Hamstrings activity was lower in PFPS, significantly for average descent and at 10° and 20° of knee flexion during descent ($P < .01$). No other group differences were identified in muscle activity during single- and double-leg squats.

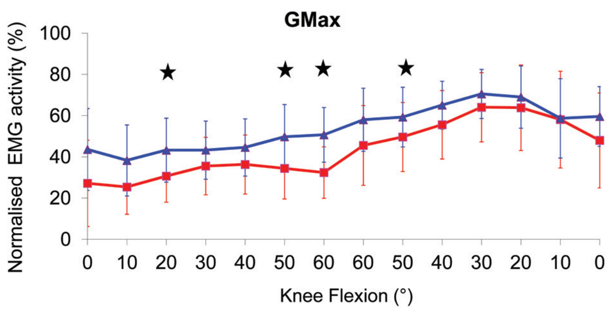

FIGURE. Mean normalized ± SD GMax EMG activity during descent and ascent of single leg squats in PFPS and healthy subjects (PFPS and control groups). In PFPS subjects GMax activity was higher at several joint angles of knee flexion ($P \leqslant .01$).

DISCUSSION: Although PFPS is a knee disorder, the changes in muscle activity were found at the hip. Subjects with PFPS had increased GMax, which may be an adaptive strategy to decelerate hip flexion during descent, compensate for the decreased hamstrings activity, and facilitate extension during ascent of a single-leg squat. No group difference in joint range was identified. This is in conflict with previous studies of similar tasks, one of which reported decreased hip internal rotation and greater adduction[2] and another that found greater internal rotation but similar adduction in females with PFPS.[3] It appears that kinematic alterations are a variable feature of PFPS.
ACKNOWLEDGEMENTS: Professor R. Woledge for his assistance in data analysis.

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CHANGES IN HIP AND KNEE KINETICS AND KINEMATICS FOLLOWING AN EXHAUSTIVE RUN IN INDIVIDUALS WITH AND WITHOUT PFPS

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) is one of the most common injuries occurring in runners. Running is typically performed until an individual feels tired or is exhausted, possibly leading to injury. Healthy individuals demonstrate increased hip internal rotation following an exhaustive run,[1,2] a pattern that is also seen in individuals with PFPS. While previous research indicates kinematic differences between PFPS and uninjured participants following an exhaustive run,[1,3] changes in joint moments have not been examined. The purpose of this project was to determine if there are changes in hip and knee kinetics and kinematics following an exhaustive run, and if those changes differ between individuals with PFPS and uninjured individuals.
METHODS: Fifteen persons with PFPS (7 men, 8 women; mean ± SD age, 27.3 ± 6.4 years; mass, 76.5 ± 12.1 kg; height, 1.73 ± 0.7 m) participated. The participants met inclusion criteria that are common for PFPS research (pain 3/10 for a minimum of 4 weeks, pain during physical activ-

ity, prolonged sitting, stair climbing, squatting). Nine persons (5 men, 4 women; 23.6 ± 4.6 years; 69.3 ± 13.0 kg; 1.73 ± 0.1 m) acted as healthy controls (CON). All participants were active a minimum of 30 minutes at least 3 times per week in running activities. The visual analog scale (VAS) was used to assess knee pain before, during, and after the running trials. The most painful knee was tested, and this was matched for the control participants. Three-dimensional kinematic data were collected at 200 Hz and ground reaction force data were collected at 1000 Hz. Participants ran on a runway at a consistent speed (3.5-4.5 m/s) wearing standardized footwear, and after several practice trials, 5 trials were recorded. Participants then wore their typical running shoes and ran on a treadmill until they reached 1 of the stopping conditions: heart rate >85% of maximum or rating of perceived exertion of 17/20.[1,3] Immediately after concluding the exhaustive run, participants returned to the standard shoe and completed an additional 5 overground running trials. Hip and trunk strength, measured in kilograms normalized to body mass (kg), was also tested before and after the exhaustion protocol for the hip abductors (ABD) and external rotators (HER) to explain changes in mechanics. Internal joint moments were calculated using an inverse dynamics approach. Knee joint moments were reported in the leg reference frame. Peak joint angle and moment data were extracted from the stance phase. The dependent variables analyzed were frontal and transverse plane peak angles and moments at the hip and knee. The independent variable was exhaustion state (Pre-EXH, Post-EXH) and injury status group (PFPS, CON). Data were analyzed using a 2-by-2 repeated-measures ANOVA ($P<.05$).

RESULTS: One female PFPS and 1 male CON participant withdrew from the study prior to testing. No significant interactions between group and exhaustion state were found for moments (P = .061-.739) or angles (P = .099-.869). A trend toward significance (P = .061) was found for the knee abduction moment interaction as those with PFPS increased and CON decreased. Significant main effects for exhaustion were found: decreased peak knee external rotation moment (P = .006) and trends for decreased peak hip abduction moment (P = .075) and increased peak knee internal rotation angle (P = .075). Group main effects included a greater peak hip abduction angle in those with PFPS and trends for greater peak hip internal rotation angle (P = .071) and lesser peak knee internal rotation angle (P = .065). Both ABD and HER strength was significantly decreased following the exhaustion protocol ($P<.001$).

DISCUSSION: The results of the study are in agreement with previous studies regarding mechanical changes following an exhaustive run. The finding of increased knee internal rotation is in agreement with the data from Dierks et al[3]; however, other changes in kinematics were not found. This study found changes in knee and hip moments that are in contrast with Truebenbach,[2] who did not report any differences following exhaustion. The reduced moments can be explained by decreased ABD and HER strength, which is consistent with previous studies.[1,3] This study provides preliminary evidence regarding the importance of understanding the changes in lower extremity mechanics due to exhaustion.

ACKNOWLEDGEMENTS: Support provided by the UW-Milwaukee College of Health Sciences.

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A COMPARISON OF HIP STRENGTH AND CORE ENDURANCE IN MALES AND FEMALES WITH A HISTORY OF PATELLOFEMORAL PAIN SYNDROME

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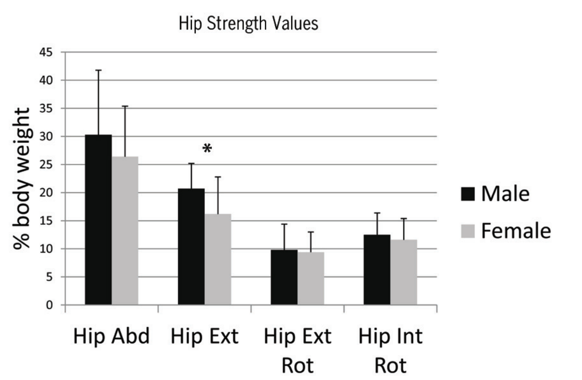
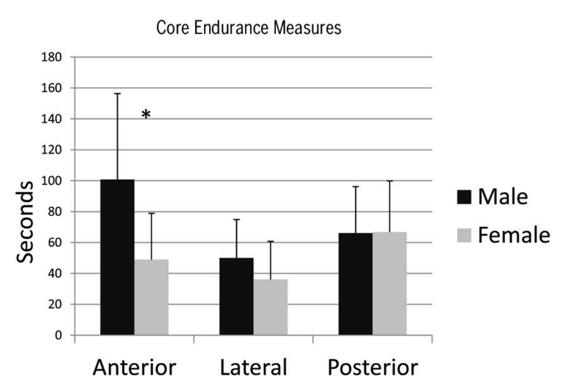
INTRODUCTION: Patellofemoral pain syndrome (PFPS) is a common knee pathology experienced by active individuals, and an emerging body of evidence supports an association between hip weakness and PFPS in females.[1] Researchers also have begun to examine the importance of core endurance in females with PFPS.[2] Although more prevalent in females, PFPS also occurs in males. To date, investigators have not determined if males with PFPS have similar hip strength and core endurance measures to females. Identification of these differences may provide important clinical insight when developing sex-specific interventions for PFPS. Therefore, the purpose of this study was to determine potential sex differences in hip muscle strength and core endurance. Our null hypothesis was that there would be no between-sex differences for any measure.

METHODS: As part of a larger RCT, data for 12 males and 18 females were analyzed (PFPS symptoms for a minimum of 4 weeks and participation in running, jumping, or cutting activities at least 30 minutes 3 times a week). Subjects initially completed a 10-cm visual analog scale (VAS), assessing average pain during activity for the week prior, and the Anterior Knee Pain Scale (AKPS). Subjects then performed 3 maximum voluntary isometric contractions of the hip abductor, extensor, external rotator, and internal rotator muscles against a force dynamometer. For each trial, subjects generated maximal force according to the "make test" against a stabilization strap. The values for the 3 trials were averaged and expressed as a percentage of body weight for data analysis. Next, subjects performed the front plank, side bridge exercise, and horizontal extension test to assess anterior, lateral (affected side), and posterior core endurance, respectively. The time that subjects maintained each position was recorded to the nearest one-tenth of a second and used for data analysis. Separate independent *t* tests were used to determine any group differences in hip strength and core endurance values. All statistical analyses were performed using SPSS Version 18.0 with a level of significance at .05.

RESULTS: No differences existed between males and females with respect to VAS (4.7 ± 1.6 cm versus 4.9 ± 1.3 cm; P = .69) or AKPS (75.4 ± 9.3 versus 71.6 ± 10.1; P = .29) scores. Males demonstrated greater hip ex-





tensor force output (P = .04) than females. All other hip force values were similar (P>.05). Males also exhibited greater anterior core endurance (P = .01) but similar lateral (P = .15) and posterior (P = .96) endurance values.

DISCUSSION: While current evidence has shown reduced hip muscle force output in females with PFPS,[1] it has remained unknown if males would exhibit a similar pattern of hip dysfunction. Overall, hip strength values for all subjects agreed with prior works.[1] These findings are clinically relevant because they provide preliminary evidence that males with PFPS may have similar hip weakness as females. Furthermore, females generated significantly less hip extensor force output than males. This finding further highlights the importance of gluteus maximus strength for this cohort.[3] We also assessed core endurance, as trunk function can influence hip function.[4] Contrary to our null hypothesis, males demonstrated increased anterior, but similar lateral and posterior, core endurance compared to females. To date, investigators have not determined if sex differences exist in core endurance for this patient population. Emerging evidence has suggested that females with PFPS may benefit from interventions aimed to improve anterior core endurance.[2] However, additional investigations are needed to better understand the influence of core endurance on the etiology and management of PFPS.

ACKNOWLEDGEMENTS: Funding support from the National Athletic Trainers' Association Research and Education Foundation.

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RELATIONSHIPS BETWEEN LOWER-LIMB BIOMECHANICS DURING A SINGLE-LEG SQUAT WITH CUTTING AND CHANGING-DIRECTION TASKS: A PRELIMINARY INVESTIGATION

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INTRODUCTION: Valgus knee motion has been associated with patellofemoral dysfunction[1] as well as acute noncontact anterior cruciate ligament injuries in female athletes.[2] Dynamic valgus is a combination of frontal and transverse plane hip and knee motion.[3] The need to develop screening tests to find athletes who may be predisposed to such injuries through repetitively loading the knee in this manner is of prime importance to design individualized knee injury prevention programs. Previous literature has found 3-D joint kinematics of the hip and knee during a single-leg squat to be related to those during jogging[4] and thus warrants further investigation as a potential screening test. No studies have attempted to relate lower-limb motion during a single-leg squat to that during cutting or other changing-direction maneuvers. Therefore, the aim of this study was to investigate the relationship between 3-D kinematic variables during a single-leg squat with lower-limb motion and moments during a 90° cut and 180° turn.

METHODS: Ten female football players (mean ± SD age, height, and mass: 20.9 ± 3.6 years, 1.70 ± 0.07 m, 59.7 ± 7.2 kg, respectively), who were all injury free, participated in the study. Each subject performed 6 trials each of a single-leg squat (SLSQT) on both limbs, 90° cut (90C), and 180° turn (180T). Each changing-direction maneuver was performed using the right leg as the turning limb. Testing took place on an indoor Mondo running surface. 3-D motion analysis was performed using Qualysis Pro reflex infrared cameras (240 Hz) operating through Qualysis Track Manager software (Version 1.10.282). Ground reaction forces were collected from 2 AMTI force platforms (1200 Hz) embedded into the running track. Knee joint moments during the change-of-direction tasks were calculated using an inverse dynamics approach through Visual3D software (C-Motion, Version 3.90.21). Joint coordinate data and force data were smoothed with a Butterworth low-pass digital filter with cutoff frequencies of 12 Hz and 25 Hz, respectively. Due to the low sample size and the absence of normality of some of the variables, Spearman rho was used to explore relationships between right lower-limb variables performing each of the tasks. The significance level was set at P<.05.

RESULTS: For the 180T, absolute and normalized valgus moments were strongly correlated to peak internal hip rotation (ρ = .770, P = .009; ρ = .794, P = .006, respectively) and hip rotation range of motion (ROM) (ρ = .636, P = .048) during the SLSQT. Hip internal rotation angle at maximum knee flexion was also significantly correlated to absolute knee valgus moment during the 180T (ρ = .646, P = .044). Peak knee valgus angle during the 180T was significantly correlated to hip rotation (ρ = .758, P = .011) ROM during the SLSQT. For the 90C, peak knee valgus angle was found to be significantly related to hip rotation ROM during a SLSQT (ρ = .661, P = .038). However, no other SLSQT variables related to 90C lower-limb joint motions or moments.

DISCUSSION: The results illustrate that hip rotation motion during a SLSQT is related to knee valgus motion and moments during 180° turns, but not 90° cuts. This could be due to biomechanical differences between the 2 maneuvers and the potential technique variance observed with the 90° cuts. Previous research has shown such relationships between running and SLSQT[4] and, along with these findings, provides some support for the use of the SLSQT to identify athletes who display poor dynamic knee control during a variety of athletic tasks. As previous research has shown that 2-D measures of frontal plane projection angle during a single-leg squat show significant correlations with 3-D estimates of hip and tibial rotations (2 major components of dynamic valgus),[3] then potentially a 2-D frontal plane analysis of a SLSQT could be a viable option for knee injury risk screening in female athletes. However, further investigation is warranted.

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IS HIP MUSCLE WEAKNESS A PREDISPOSING FACTOR FOR PATELLOFEMORAL PAIN IN FEMALE NOVICE RUNNERS? A PROSPECTIVE STUDY

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INTRODUCTION: The kinetic chain theory suggests that a dysfunction of a certain joint can manifest injuries in other joints, particularly in those distal to the affected joint. Hence, it has been recognized that the mechanics of the patellofemoral joint is also influenced by segmental interactions of the lower extremity.[1] Hip muscle weakness has been proposed to contribute to patellofemoral malalignment and the development of the patellofemoral pain syndrome (PFPS).[2] However, from retrospective studies that have been addressing this issue, it is still unclear if hip muscle weakness is a cause or a consequence of PFPS.[3-6,11,12] Epidemiological studies have found that PFPS is the most prevalent injury in runners.[7,8] Research has shown that during locomotion women exhibit significantly greater external knee valgus movement and hip internal rotation compared to their male counterparts.[9,10] The ability of women to control these motions may depend on the strength of proximal muscle groups that are antagonistic to these movement tendencies. Repetitive activities with this malalignment may make female runners more vulnerable to the development of PFPS. The purpose of this study was to prospectively investigate in female recreational runners if hip muscle weakness, measured in an isometric way, is a predisposing factor for the development of PFPS.

METHODS: Before the start of a 10-week "start to run" program, the isometric strength of the hip flexor, extensor, abductor, adductor, and external and internal rotator muscles was measured in 77 healthy female novice runners. All subjects were asymptomatic before the initiation of the "start to run" program. The isometric strength of the hip muscles was evaluated with a Microfet handheld dynamometer (Hoggan Health Industries, West Jordan, UT). The recorded strength measurements, in Newton, were normalized to body weight and the peak force from the 3 trials was used for statistical analysis. During the 10-week training period, patellofemoral pain was diagnosed and registered by an orthopaedic surgeon. A binary logistic regression analysis was used to identify the intrinsic risk factors for PFPS in this study. Statistical significance was accepted at the level of $\alpha \leq .05$.

RESULTS: During the 10-week "start to run" program, PFPS was diagnosed in 16 of the 77 runners. No significant differences in age, height, weight, BMI, or Q-angle were found between the runners who sustained PFPS and those who did not. Statistical analysis revealed that there was no significant difference in strength of any of the assessed hip muscle groups between the runners who did and those who did not develop PFPS. Logistic regression analysis did not identify a deviation in strength of any of the assessed hip muscle groups as a risk factor for PFPS.

DISCUSSION: Based on the results of this study, we cannot conclude that weak isometric strength of the hip muscles predisposes people to the development of PFPS. Although this might seem contradictory to the results of former studies, which found an association between decreased hip muscle strength and PFPS,[3,11,12] the outcome of this study suggests that the observed weakness of the hip abductor, external rotator, and extensor muscles in those with PFPS in previously reported retrospective studies might be the result of, rather than a predisposing factor for, PFPS. Although hip muscle weakness has been demonstrated in patients with PFPS, care should be taken in considering hip muscle weakness as a causal factor.

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ATROPHY OF THE VASTUS MEDIALIS OBLIQUUS IN PATIENTS WITH PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: One of the main suggested contributing factors of patellofemoral pain syndrome (PFPS) is abnormal patellar tracking due to an imbalance in the activity of the vastus medialis obliquus (VMO) relative to the vastus lateralis (VL).[1,2] Accordingly, it has often been suggested that PFPS is associated with decreased VMO muscle mass. Strikingly, there are few studies evaluating VMO atrophy in patients with PFPS compared with healthy controls using valid techniques. The present study is the first to examine with magnetic resonance imaging (MRI) if patients with PFPS exhibit a smaller size of the muscles (VMO and VL) that play a significant role in the dynamic balance of the patella.

METHODS: Forty-six patients with PFPS (21 male and 25 female; mean ± SD age, 25.0 ± 7.4 years) and a control group of 30 healthy subjects (13 male and 17 female; age, 21.6 ± 4.5 years) underwent MRI of the quadriceps. Muscle size was determined by calculating the anatomic cross-sectional area (CSA) of the total quadriceps and its components (**FIGURE**).

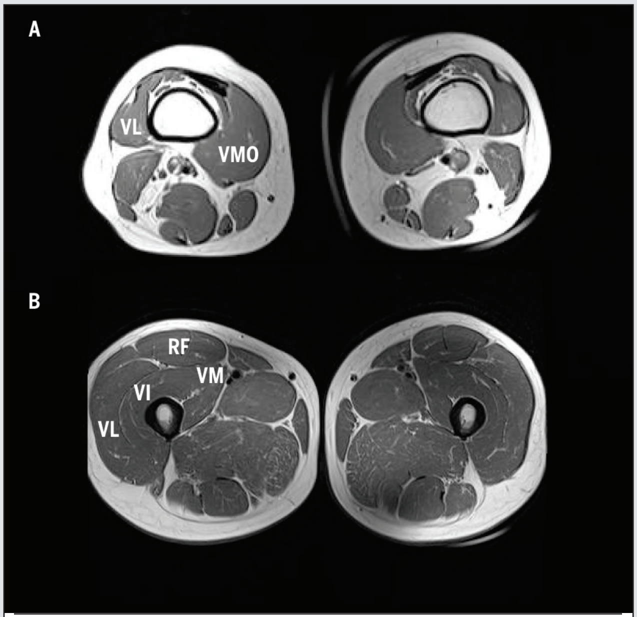

FIGURE. MRIs of the quadriceps. (A) Female with PFPS, image 2 cm above the superior border of the patella. (B) Male with PFPS, image midthigh level. VMO, vastus medialis obliquus; VL, vastus lateralis; VM, vastus medialis; VI, vastus intermedius; RF, rectus femoris.

RESULTS: The CSA of the VMO was significantly smaller in the PFPS group than in the control group (16.67 ± 4.97 versus 18.36 ± 5.25 cm^2; P = .040), while there was no significant difference in the CSA of the VL at patellar level. A tendency was noted for a smaller total quadriceps CSA for the PFPS patients at midthigh level (66.99 ± 15.06 versus 70.83 ± 15.30 cm^2; P = .074).

DISCUSSION: This is the first study to examine VMO size in patients with PFPS by MRI. The main results indicated that patients with PFPS show a less-developed quadriceps, and in particular a significantly smaller CSA of the VMO compared to healthy controls. Because there was no significant difference in the CSA of the VL at patellar level, it seems that the VMO muscle was disproportionately smaller in comparison with the VL. As the current study was cross-sectional, it is impossible to draw conclusions about VMO atrophy being the effect or the cause of PFPS. Longitudinal, prospective studies are needed to establish the cause-effect relation of VMO atrophy and PFPS. Moreover, it has not been demonstrated that the patients also had a smaller VMO size before their patellofemoral complaints started. Consequently, it may be possible that within the patients with PFPS, the VMO did not atrophy but was just developmentally smaller compared to those of the healthy controls. In conclusion, although it is not clear whether this atrophy is a result or a cause of PFPS, the results of this study do show that atrophy of the VMO is a contributing factor in PFPS.

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ALTERATIONS IN IN VIVO KNEE JOINT KINEMATICS FOLLOWING THE LOSS OF VASTI MEDIALIS FUNCTION: IMPLICATIONS FOR PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: A widely accepted theory in regard to the source of patellofemoral (PF) pain syndrome (PFPS) is that a force imbalance around the knee leads to PF static malalignment and dynamic maltracking. In turn, this causes elevated joint contact stresses, which ultimately results in PF pain. The source of this force imbalance is still open to debate, with some postulating the cause to be delayed timing or loss of strength in the vasti medialis (VM) or an imbalance in the passive structures.[1] Yet, numerous studies refute these claims as well.[2] Unfortunately, a direct in vivo relationship between knee joint kinematics and loss of VM force has not been established in patients with PFPS. Thus, the purpose of this study was to determine how the loss of VM force alters the dynamic control of 3-D in vivo tibiofemoral (TF) and PF kinematics during a volitional extension task.

METHODS: To date, 13 females with no history of knee pain, trauma, or surgery have participated in this IRB approved study (mean ± SD age, 28.6 ± 9.8 years; height, 165.3 ± 7.7 cm; mass, 58.9 ± 7.3 kg). Signed consent was obtained and a history and physical was performed during the first visit. Next, subjects were placed supine in an MR imager (3.0 T, Philips Medical Systems, Best, the Netherlands). For dynamic scanning, the knee was supported in a bent position on a cushioned block within a customized coil holder. Subjects were asked to cyclically flex/extend their knee while a dynamic cinephase contrast (CPC) magnetic resonance (MR) image set (x, y, z velocity and anatomic images) was acquired.[3] The scanning protocol was saved so that the identical protocol could be used for the second visit. During the second visit, scanning began immediately after administering a motor branch block to the VM. Using ultrasound (US) guidance and electrical stimulation, the femoral nerve motor branch to the VM was localized and then 3 cc of 1% lidocaine was injected. Evaluation of the effectiveness of the block was assessed by absence of visible twitch (visual surface inspection and US) with percutaneus electrical stimulation of the motor nerve. Using reference marks on both the coil holder and the skin over the subject's knee, the subject was placed in as similar a position as possible to the first-visit scan. All scanning occurred within 20 minutes of completing the muscle block. The PF and TF kinematics, both preinjection and postinjection (pre-I and post-I), were quantified through integration of the CPC data. All kinematic data were interpolated to single knee angle (KA) increments. The pre-I and post-I kinematics were compared using a paired Student t test. Correlations between the change in kinematics post-I and the pre-I kinematics were quantified at the KA of maximum post-I change in PF medial shift (KA, 15°).

RESULTS: Postinjection, the patella shifted laterally (**FIGURE**) (max, 1.7 ± 1.7 mm; $P = .004$; KA, 15°), whereas the tibia rotated (max, 3.7° ± 3.5°; $P = .003$; KA, 15°) and shifted laterally (max, 2.6 ± 2.5 mm; $P = .04$; KA, 12°). These changes were 4.1 to 4.7 times greater than the average subject repeatability.[3] An insignificant trend of PF lateral tilt was seen post-I. Post-I PF lateral shift was correlated with pre-I PF superior displacement ($r = 0.48$) and valgus rotation ($r = 0.59$). TF external rotation and lateral shift were not correlated with any pre-I kinematics, but were correlated with each other ($r = 0.81$).

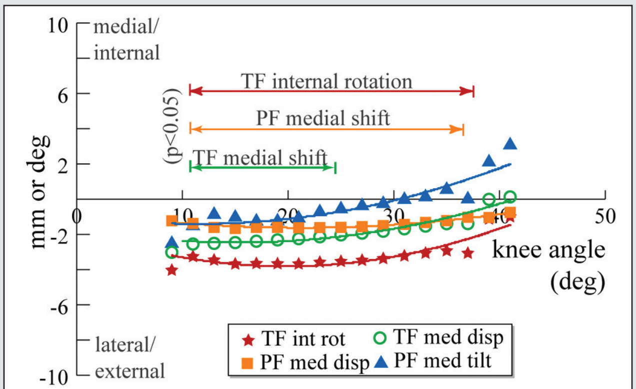

FIGURE. Postinjection differences in PF kinematics. A second-order polynomial is fit to the data and symbols are provided at 2° KA increments. The KA ranges where significant differences were found are shown using double-arrow lines.

DISCUSSION: In the current study, a loss of VM function led to increased lateral PF shift and external TF rotation. This supports the fact that the VM exerts a medially directed force on the patella[4] and an internal rotation moment on the tibia via the patellar tendon. These kinematic changes mirrored the difference in axial plane kinematics seen between patients diagnosed with PFPS and controls.[5] Although the muscle block likely produced a greater loss in VM strength than that experienced by patients with PFPS, the post-I change in PF lateral shift was only 59% of the difference between the PFPS and control cohorts. In addition, the PFPS cohort also had increased PF superior displacement, flexion, valgus, and TF external rotation. Thus, the loss in VM function cannot explain all the kinematics changes in the PFPS cohort, and it is most likely that VM weakness is a major factor in, but not the sole source of, PF maltracking.

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THE EFFECT OF PATELLA HEIGHT AND TROCHLEAR GROOVE DEPTH ON PATELLA LATERAL TILT

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INTRODUCTION: Excessive lateral tilting of the patella has been linked to lateral patella compression syndrome and is thought to be 1 factor that may underlie the development of patellofemoral pain (PFP). Although excessive lateral tilting of the patella is a common finding in persons with PFP, the underlying cause of this anomaly is not fully understood. From a structural standpoint, 2 structural predispositions have been discussed in the literature: patella alta and trochlear dysplasia. Both of these conditions result in diminished patella stability typically afforded by the lateral femoral condyle of the distal femur. Additionally, Hvid et al[1] have proposed that during development a higher-riding patella would result in a shallower trochlear groove. However, few reports have assessed this relationship. To date, a clear understanding of how altered bony structure of the distal femur relates to lateral patella tilt is lacking. The purpose of this study was to assess the association between patella height, trochlear groove depth, and lateral patella tilt (LPT). We hypothesized that patella height and trochlear groove depth would significantly predict patellar tilt. As a secondary aim, we also assessed the relationship between patella height and trochlear groove depth.

METHODS: Thirty-two subjects participated in this study. Subjects could not have any previous history of patellar dislocations or instability. Preoperative radiographs for patients scheduled to undergo either an anterior cruciate ligament reconstruction or cartilage repair surgery were used. Axial and lateral radiographs of the patellofemoral joint were obtained in weight bearing with the knee flexed to 30°. The vertical height of the patella was quantified using the Insall-Salvati ratio (ISR), which measures the length of the patella tendon relative to the length of the patella. The depth of the trochlear groove was assessed by measuring the sulcus angle (SA), which was quantified as the angle formed by the intersection of lines defining the medial and lateral slopes of the trochlear groove. LPT was quantified as the angle formed between a line drawn along the lateral facet of the patella and a line along the posterior aspects of the medial and lateral femoral condyles. Using this measure, a smaller angle was

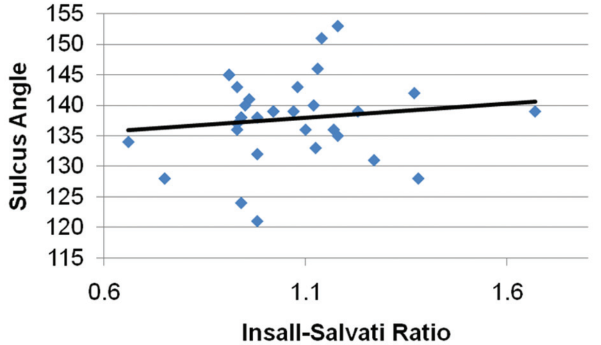

FIGURE. Correlation plot with the best linear fit line for the Insall-Salvati ratio and the sulcus angle.

representative of greater LPT. The relationship between the ISR, SA, and LPT was assessed using a stepwise linear regression model. The relationship between the ISR and the SA was assessed using Pearson product moment correlation coefficient.

RESULTS: The ISR and SA predicted 54% of the variance in patellar tilt. The model with SA and ISR resulted in $r = 0.734$ and $P<.001$. There was also a nonsignificant correlation between SA and ISR ($r = -0.25$, $P = .17$).

DISCUSSION: The purpose of this study was to evaluate if patella tilt could be predicted by the ISR and the SA. We found that these 2 measures predicted over 50% of the variance in patellar tilt, which has implications for various populations with PFP. A higher-riding patella and a shallower trochlear groove lessen the ability of the osseous structures of the distal femur to resist the lateral pull of the quadriceps and the iliotibial band. This could lead to greater shear across the patellofemoral joint and result in pain. Additionally, the effectiveness of bracing and taping to alter lateral patellar tilt may be restricted by osseous structures of the patellofemoral joint. Future studies should consider the effectiveness of these treatments with different types of bony alignment. Interestingly, 1 of the greatest causes of revisions for total knee arthoplasty is PFP.[2] Good patellofemoral articulation has previously been cited to be an important but overlooked contributor to reducing PFP after surgery.[3] Establishing a proper patellar-to-trochlear relationship is key to a successful revision knee arthroplasty. With newer-generation knee implants, deeper trochlear grooves have been designed to help promote improved patellar tracking. We also found no relationship between the ISR and the SA. The differences in the current study as compared to the one by Hvid et al[1] could be in part due to the participants in their study having patella instability whereas ours did not. The relationship between these variables may be stronger in more pronounced cases. However, within our sample we had individuals with a high-riding patella (**FIGURE**) that was not associated with a shallow trochlear groove.

ACKNOWLEDGEMENTS: These results were in part presented at the 2011 combined sections meeting of the American Physical Therapy Association.

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THE INFLUENCE OF RUNNING ON PATELLA WATER CONTENT AND BONE MARROW LESIONS IN FEMALES WITH AND WITHOUT PATELLOFEMORAL PAIN

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INTRODUCTION: It has been suggested that patellofemoral pain (PFP) is the result of increased pressure on highly innervated subchondral bone.[1] Current literature also suggests that individuals with PFP exhibit greater patellofemoral joint stress during functional activities.[2] Repetitive overloading of the patellofemoral joint is thought to result in articular cartilage breakdown, increased subchondral bone thickness and stiffness, and bone marrow lesions (BMLs).[3] BMLs are associated with the accumulation of extracellular fluid within bone marrow and have been suggested as the source of pain in degenerated joints.[4] The purpose of this pilot study was to quantify the influence of running on bone marrow water content changes in individuals with and without PFP. To accomplish this goal, we used an iterative decomposition of water and fat with echo asymmetry and least-squares estimation (IDEAL) magnetic resonance imaging (MRI) protocol.[5,6]

METHODS: Two female subjects with PFP (34.5 ± 6.4 years; 1.68 ± 0.0 m; 60.3 ± 4.6 kg) and 2 pain-free female controls (28.0 ± 4.2 years; 1.64 ± 0.0 m; 55.5 ± 5.0 kg) have participated in this study thus far. Study procedures included (1) a prerunning MR scan, (2) a 40-minute running session, and (3) a postrunning MR scan immediately following running. Each subject performed treadmill running to a perceived exertion level of 13 (moderate) based on the Borg scale. A 10-cm visual analog scale (VAS) was used to assess pain levels before and after running. MRI assessments were performed on a GE 3 Tesla scanner with an 8-element knee coil. A spoiled-gradient-echo IDEAL pulse sequence was utilized: TR, 20.2 milliseconds; TE = {1.68 2.67 3.65 4.63 5.62 6.61} milliseconds; slice thickness, 2 mm; FOV, 160 × 160 mm; matrix, 256 × 256; BW, 125 kHz. The reconstructed fat-fraction images were used for analysis. Water fraction was defined as 100-fat fraction (%). Local BMLs were defined as the diffuse dark signals adjacent to the articular cartilage. To compare the water signal before and after exercise, the patella water fraction and the volume of local BMLs were manually contoured and measured.

RESULTS: Prior to running, the subjects with PFP demonstrated greater patella water fraction compared to the pain-free controls (**FIGURE 1**). Additionally, both subjects with PFP demonstrated local BMLs on the lateral facet of the patella. After running, PFP subjects reported an average increase in pain of 4.7 ± 4.4. No pain was reported in the control subjects postrunning. The increased pain in the PFP subjects was accompanied by elevated patella water fraction (**FIGURE 1**) and increased volume of local BMLs (**FIGURE 2**).

DISCUSSION: Our data reveal that in persons with PFP, patella water fraction and volume of local BMLs increase in response to 40 minutes of moderate-effort running. The higher water fraction postrunning may result in elevated intraosseous pressure, thereby creating pain. As only 2 PFP and 2 control subjects were studied in this preliminary investigation, future efforts will focus on increasing the sample size to better understand the influence of loading on bone marrow water signal in per-

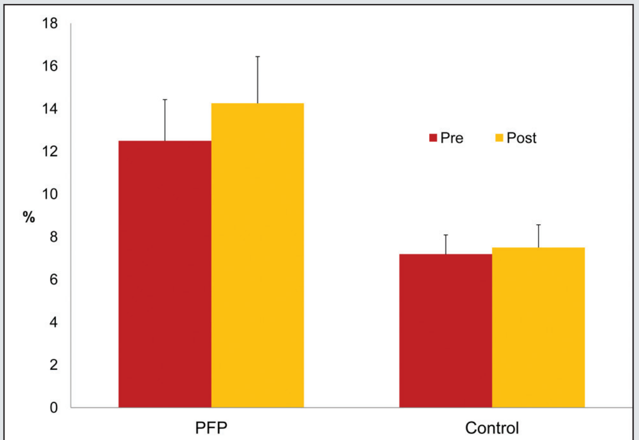

FIGURE 1. Average patella water fraction in PFP and control subjects prerunning and postrunning.

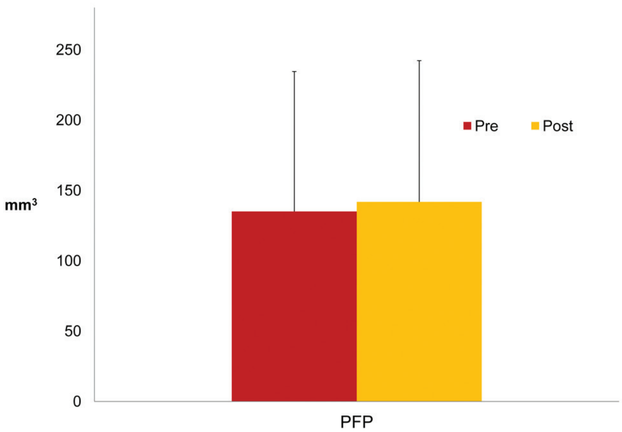

FIGURE 2. Average BML volume in PFP subjects prerunning and postrunning.

sons with PFP.

ACKNOWLEDGEMENTS: The authors thank Huanzhou Yu and Ann Shimakawa from GE Healthcare Applied Science Laboratory for their technical support.

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PATELLOFEMORAL JOINT COMPRESSION FORCES IN BACKWARD RUNNING

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INTRODUCTION: Backward running (BR) is used in rehabilitation of patients with patellofemoral pain syndrome (PFPS). It has been reported to have reduced peak patellofemoral joint compression forces (PPFJCF) compared to forward running (FR).[1] This may be due to slower speeds of BR. This study investigated if BR had reduced PPFJCF compared to FR at similar speed, and if so, whether this was due to kinematics or kinetics.

METHODS: Seventeen healthy volunteers (7 male, 10 female; mean ± SD age, 27 ± 6 years; height, 1.7 ± 0.1 m; mass, 72 ± 20 kg) performed FR and BR at a speed of 2.8 to 3.4 m/s, for 3 trials each. Kinematics were collected using a Vicon system (Oxford Metrics Group Ltd), and ground reaction force data using 2 force plates (Kistler Instruments Ltd). Kinetics were calculated with Vicon and data were further analyzed in Matlab (Mathworks Inc). PPFJCF was calculated combining experimental data with values for patella tendon moment arm (dPT) and patella mechanism angle from the literature[2]: $PPFJCF = (R_{Fq\text{-}Fpl})/F_q$, with $Q_{TForce}(F_q) = M_k/dPT$, where $R_{Fq\text{-}Fpl}$ is the ratio between quadriceps and patella tendon force,[3] F_q is patella tendon force, Q_{TForce} is quadriceps tendon force, and M_k is the peak knee extensor moment. The role of kinematics and kinetics was investigated with a telescopic inverted pendulum (TIP) model. Statistical differences between FR and BR were calculated with an independent *t* test (with P<.001 as significant difference).

RESULTS: Running speed was not significantly different between FR and BR (3.0 ± 0.2 and 3.0 ± 0.2 m/s). PPFJCF was significantly higher in FR than in BR (4.5 ± 1.5 and 3.4 ± 1.4 BW). M_k was significantly higher in FR than in BR (158 ± 54 and 124 ± 51 Nm), while knee angle at M_k was not significantly different (44° and 41°). This indicates that kinetics (moments) and not kinematics (knee angle) caused the reduced PPFJCF in BR. TIP model calculations (**FIGURE**) showed that the stance leg shortened during initial deceleration and extended during push-off in FR and BR. In FR, the stance leg extended more during the push-off phase than in BR (**FIGURE**). In both FR and BR, M_k occurred at similar approach angles of the contact leg (θ) (80° ± 4° and 82° ± 3°) (**FIGURE**). The body was upright and leaning forward (as θ was close to but smaller than 90°) at M_k. M_k therefore resulted in a loading response in both FR and BR, but a push-off response in FR only (a push-off response in BR requires a backward lean, θ>90°). As M_k in BR did not provide push-off, we propose BR seems generated more by pendular movement, while FR has a predominantly telescopic motion.[4] Pendular movement does not require high knee extensor moments, but high hip flexor moments to generate push-off. This was confirmed by the significantly higher peak hip flexor moments in BR compared to FR (113 ± 54 and 76 ± 43 Nm). Interestingly, for some participants (7 in total), PPFJCFs were similar in BR and FR. This is related to FR style.

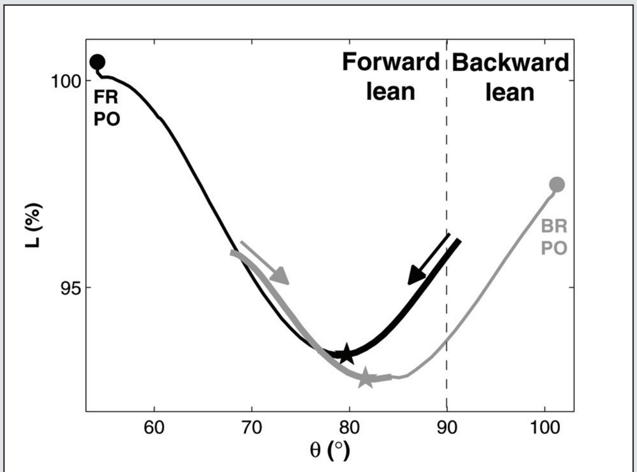

FIGURE. TIP model calculations with stance leg length (L) against θ. The gray lines are average data for BR and the black lines for FR, with the thinner parts for the push-off phase. Stars indicate where M_k occurred. FRPO and BRPO are push-off in FR and BR, respectively.

DISCUSSION: PPFJCF was lower in BR than in FR and this was not due to a difference in speed. The knee angles at the peak knee extensor moment were similar in BR and FR; kinetics differed, however, with higher peak knee extensor moments in FR and higher peak hip flexor moments in BR. This increased peak knee extensor moment was therefore related to the increased PPFJCF in FR. These differences were not consistent in all participants; further research is required to investigate whether it is the BR style that resulted in a reduced PPFJCF or whether an adapted FR style could also be advised to PFPS patients to exercise with reduced knee pain.

ACKNOWLEDGEMENTS: This study was also presented at the ISB 2011 meeting, Brussels, Belgium, July 3-7.

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INDIVIDUALS WITH PATELLOFEMORAL JOINT OSTEOARTHRITIS AFTER ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION HAVE WORSE FUNCTION THAN THOSE WITHOUT OSTEOARTHRITIS

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INTRODUCTION: Early knee osteoarthritis (OA) frequently develops secondary to anterior cruciate ligament reconstruction (ACLR).[1] The few studies that evaluated patellofemoral joint (PFJ) reported a high prevalence of PFJ OA (approximately 46%) >7 years postsurgery.[2,3] Notably, these studies only followed people who had a patellar tendon autograft, which is known to be associated with PFJ morbidity. Lower-limb function is frequently impaired after ACLR, with only 44% of people having normal function on a hop test battery at 2 to 5 years post-ACLR.[4] Importantly, performance on a hop test[5] and impaired function on a patient-reported outcome (PRO)[6] predicted knee OA development after ACLR. No studies have investigated lower-limb function in people with PFJ OA after ACLR. This study aimed to: (1) describe the prevalence of radiographic PFJ and TFJ OA 5 to 10 years after ACLR using an arthroscopic hamstring tendon autograft (HT), (2) compare patient-reported functional limitations, including activity levels between people with PFJ OA and those free of OA, and (3) compare the range of knee motion and functional performance between people with and without PFJ OA.

METHODS: 70 people were recruited and performed: (1) standard radiographs (posteroanterior for TFJ, skyline for PFJ) to grade compartment-specific OA using established criteria[7]; (2) PROs for knee function including: Knee Osteoarthritis Outcome Score (KOOS),[8] Anterior Knee Pain Scale (AKPS),[9] Tegner Activity Scale,[10] International Knee Documentation Committee score (IKDC)[11]; and (3) objective measures of function: range of knee movement and functional performance on hop distance and side hop tests.[4]

RESULTS: Radiographic PFJ OA was evident in 47% (33/70) and radiographic TFJ OA was evident in 33% (23/70). In total, 48% (34/70) exhibited no radiographic evidence of either TFJ or PFJ OA. Of the 36 (51%) people with radiographic OA, isolated PFJ OA was the most common distribution (41%), followed by tricompartmental distribution (31%), then lateral TFJ and PFJ distribution (13%), combined medial TFJ and PFJ (8%), and isolated TFJ (8%). There were no differences in age, height, or weight for people with PFJ OA and those with no radiographic OA. For the PROs, individuals with radiographic PFJ OA had significantly worse scores on the AKPS, IKDC (**TABLE**), and most scales of the KOOS (**FIGURE**) than those without OA. No differences were observed for the Tegner Activity Scale. For objective measures of function, there was no difference in the knee extension range between groups. However, participants with PFJ OA performed worse on functional tests (hop distance and side hop test) than those who were free of OA ($P<.04$).

TABLE Comparison of PRO Between Those With and Without PFJ OA

	PFJ OA	No OA	*P* Value
AKPS	87 ± 17	95 ± 5	.014
IKDC	78 ± 18	90 ± 8	.003

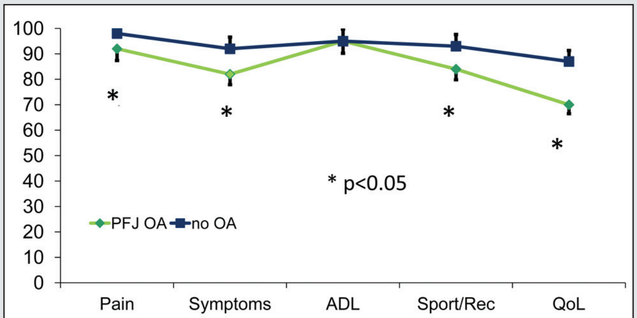

FIGURE. Comparison of KOOS between those with and without PFJ OA.

DISCUSSION: PFJ OA is relatively common approximately 7 years after HT ACLR (prevalence of 47%), and this rate is alarmingly greater than the prevalence of radiographic knee OA (15%) observed in the contralateral knee 10 to 15 years post-ACLR.[1] Our findings, combined with prior studies, suggest that PFJ OA is more of a problem than previously considered and that more research is needed to investigate the likely causes of its development after ACLR. Furthermore, function measured using PRO and performance on hop tests was significantly lower in the PFJ OA group than those with no OA. While the relationship between OA development and loss of function is not known, it appears restoration of function in people with PFJ OA after ACLR should be a high priority.

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LOWER-LIMB AND FOOT KINEMATICS IN DISTANCE RUNNERS WITH PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) is the most common overuse injury in distance runners. Prolonged rearfoot eversion is believed to cause prolonged tibial internal rotation and excessive femoral internal rotation, and predispose runners to PFPS.[1] The aim of this investigation was to compare hip rotation, knee rotation, tibial rotation, and rearfoot and forefoot joint angles between runners predisposed to PFPS and normal controls during barefoot treadmill running.

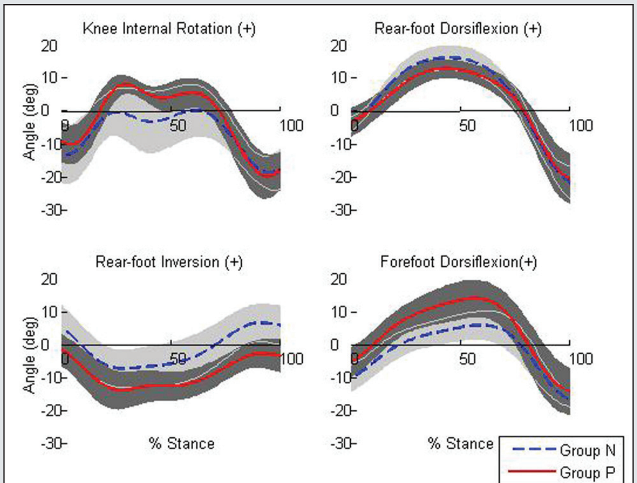

FIGURE. Knee rotation, rearfoot inversion/eversion, and rearfoot and forefoot plantar/dorsiflexion for runners with (Group P) and without (Group N) a history of PFPS during barefoot treadmill running.

METHODS: Twelve female midfoot-strike runners participated in the study. Six runners had a previous history of PFPS, but were asymptomatic at testing. Six runners had no history of PFPS or any other knee injury. Spherical reflective markers of 9-mm diameter were attached to known anatomical landmarks of both lower limbs.[2] A 12-camera Vicon MX System (Vicon Motion Systems, Oxford, UK) was used to collect 3-D spatial data at 200 Hz as the subject ran barefoot on a treadmill at 3.56 m/s. The kinematics of the dominant leg of the normal subject group and the injured leg of the patellofemoral subject group were assessed. Hip joint angles were calculated using the Vicon Plug-in Gait lower-limb model. Knee and multisegment foot joint angles were calculated using the Oxford Foot Model.[2] The timings of foot-strike and toe-off were calculated using kinematic methods.[3] Five strides of each subject were normalized to the stance period using cubic spline interpolation. Joint angles at foot-strike and toe-off, peak angular values, times to peak angular values and angular excursions for forefoot and rearfoot plantar/dorsiflexion, rearfoot inversion/eversion, knee rotation, tibial rotation, and hip rotation were identified for each of the 5 strides of each subject. Subject means of the discrete kinematic variables were calculated across the 5 strides of each subject. Group means and standard deviations were calculated across subjects with and without a history of PFPS.

RESULTS: Rearfoot eversion (peak and angle at toe-off) was significantly greater in runners with a previous history of PFPS compared to healthy controls (**FIGURE**). Runners with a history of PFPS exhibited decreased rearfoot dorsiflexion (peak and excursion) compared to healthy controls, although these differences were not significant. Knee internal rotation (peak and excursion) and forefoot dorsiflexion (peak) were higher for runners with a history of PFPS compared to healthy controls, although these differences were not significant.

DISCUSSION: Dorsiflexion at the midfoot can only occur when the subtalar joint is everted.[4] We propose a sequence of events whereby the increased rearfoot eversion that was observed in subjects with a history of PFPS was secondary to the reduced dorsiflexion at the rearfoot that was also observed in these subjects. This mechanism allowed the runners to gain additional dorsiflexion at the forefoot. Rearfoot eversion is believed to be coupled with internal rotation of the tibia.[5] The increased knee internal rotation that was observed in runners with a history of PFPS therefore corresponds well with the increased eversion that was also observed. The results of the study did not support the theory that prolonged rearfoot eversion causes prolonged internal rotation of the tibia and excessive internal rotation at the hip, and predisposes runners to PFPS.[1]

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THE RELATIONSHIP BETWEEN REARFOOT, TIBIAL, AND FEMORAL KINEMATICS IN INDIVIDUALS WITH PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) development is considered to be multifactorial, with various local, proximal, and distal biomechanical factors proposed to be associated. One factor frequently dis-

TABLE Correlation Coefficient Results

	Peak Rearfoot Eversion		Rearfoot Eversion ROM	
	PFPS	CON	PFPS	CON
Peak tibial IR				
r	0.469*	0.344	0.063	0.022
P	0.018	0.162	0.766	0.931
Tibial rotation ROM				
r	0.001	0.246	0.091	0.280
P	0.995	0.326	0.664	0.260
Peak hip adduction				
r	−0.184	0.550*	0.394	0.488*
P	0.377	0.022	0.051	0.047
Hip adduction ROM				
r	0.341	0.164	0.527†	0.501*
P	0.096	0.529	0.007	0.040
Peak hip IR				
r	−0.188	0.219	−0.158	−0.167
P	0.369	0.399	0.452	0.522
Hip rotation ROM				
r	0.285	0.324	0.220	0.432
P	0.168	0.204	0.290	0.083

Abbreviations: CON, control group; IR, internal rotation; PFPS, patellofemoral pain syndrome group; ROM, range of motion.
**P<.05.*
†P<.01.

cussed and evaluated in the literature is excessive or prolonged rearfoot eversion, thought to increase tibial internal rotation (IR). Moving more proximally, greater tibial IR is hypothesized to produce medial collapse of the knee due to coupling with hip (femoral) IR and adduction. This aberrant movement pattern is thought to be detrimental to the patellofemoral joint (PFJ) due to associated increases in lateral PFJ stress. Despite sound theoretical rationale, previous research evaluating rearfoot motion has not identified any consistent links between rearfoot kinematics during gait and the presence of PFPS,[1,2] possibly due to the condition's heterogeneity. Additionally, no previous research has attempted to validate the theoretical link between rearfoot, tibial, and femoral motion in individuals with PFPS. This study aimed to evaluate the relationship of rearfoot eversion with tibial and femoral (hip) kinematics theoretically linked to PFPS development.

METHODS: Twenty-six individuals (5 males, 21 females) with PFPS aged 25.1 ± 4.6 years and 20 control participants (4 males, 16 females) aged 23.4 ± 2.3 years participated. Each participant underwent 3-D kinematic analysis during overground walking using a 10-camera Vicon motion analysis system. Specifically, peak angles and range of motion (ROM) for rearfoot eversion (relative to the laboratory), tibial IR (relative to the laboratory), hip adduction, and hip IR were measured using standardized marker sets (Oxford Foot Model combined with Plug-in Gait). The association of rearfoot motion with tibial and hip motion was evaluated using partial correlation coefficient statistics, entering gait velocity as a covariate.

RESULTS: Correlation coefficient results are presented in the **TABLE**. Greater peak rearfoot eversion was found to be associated with greater tibial IR in the PFPS group, explaining 22% of its variance, and greater peak hip adduction in the control group, explaining 30% of its variance. Greater rearfoot eversion ROM was found to be associated with greater hip adduction ROM in the PFPS and control groups, explaining 28% and 25% of variance, respectively, and greater peak hip adduction in the control group, explaining 24% of variance. Additionally, there was a trend toward an association between rearfoot eversion ROM and peak hip adduction in the PFPS group ($P = .051$).

DISCUSSION: Results from this study indicate differences in the relationship of peak rearfoot eversion with tibial and femoral (hip) kinematics between individuals with and without PFPS. Consistent with theoretical rationale linking rearfoot motion with PFPS development, peak rearfoot eversion was associated with peak tibial IR in individuals with PFPS. This relationship was not found in the control group, indicating it may play a causative role. However, prospective research is needed to confirm this. The opposite was found for peak hip adduction (ie, significantly associated with greater peak rearfoot eversion in controls but not PFPS). Further prospective research may shed light on the significance of this finding to PFPS pathology. More consistent relationships were found between rearfoot eversion ROM and proximal kinematics. Specifically, associations were found between greater rearfoot eversion ROM and greater hip adduction peak and ROM in both groups. This finding may be particularly important when considering treatment or prevention strategies for PFPS. Theoretically, treatment strategies aimed at either end of the kinetic chain (ie, foot orthoses to reduce rearfoot eversion or hip strengthening to reduce hip adduction) may have similar overall effects on lower-limb motion and, therefore, clinical outcomes. Further research evaluating this possibility is needed.

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A PRELIMINARY ANALYSIS OF GENDER-SPECIFIC RISK FACTORS FOR PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Females are reported to be 2 to 3 times more likely to develop patellofemoral pain syndrome (PFPS) compared to their male counterparts[1,2]; however, females' predisposition to PFPS is not well understood. Prospective investigations have been performed to determine biomechanical risk factors for PFPS,[3-7] but none of these investigations has determined if biomechanical risk factors for PFPS differ between males and females. Gaining an understanding of gender-specific risk factors may help to explain the predisposition of females to PFPS. Therefore, the purpose of this investigation was to determine the association between selected biomechanical variables and risk of incident PFPS separately for males and females.

METHODS: The cohort consisted of 1319 cadets (513 females, 806 males) from the United States Naval Academy (USNA). This cohort is part of a larger investigation that is assessing risk factors for ACL injury (JUMP-ACL). At the time of enrollment in this investigation, all participants were incoming freshmen and free from lower extremity injury that would limit their participation in a jump-landing task and/or lower extremity strength tests. Each participant underwent a baseline biomechanical assessment during his/her first summer of enrollment at the USNA. A Flock of Birds (Ascension Technologies, Inc, Burlington, VT) was utilized to collect 3-D kinematics of the hip and knee during 3 trials of a jump-landing task. The jump-landing task consisted of individuals jumping from a 30-cm-high box set at a horizontal distance of 50% of their height, down to a force platform, and upon landing, jumping vertically for maximum height. Peak isometric strength of the hip extensors, abductors, internal and external rotators, and knee flexors and extensors was collected over 2 consecutive trials. Postural measures (Q-angle and navicular drop) were assessed over 3 consecutive trials. Following baseline data collection, participants were followed prospectively for a maximum of 4 years to determine those diagnosed with PFPS. Diagnosis of PFPS was determined based on a manual review of medical records by the principal investigator. The criteria that needed to be met for inclusion in the PFPS group included: retropatellar knee pain with physical activity, pain on palpation of either the patellar facets or femoral condyles, and negative findings on examination of the knee ligaments, menisci, bursae, and synovial plica. Peak 3-D hip and knee kinematic data were determined during the stance phase of the jump-landing task (initial contact to toe-off) using a custom Matlab software program (The MathWorks, Inc, Natick, MA). All kinematic data were averaged over the 3 trials of the jump-landing task. All strength data were normalized to the mass of the subject and averaged over 2 trials. Postural measures were averaged over 3 trials. Separate multivariate Poisson regression analyses were performed to determine the risk of PFPS in males and females for the following biomechanical variables: hip flexion, internal rotation, and adduction angles; knee flexion, internal rotation, and valgus angles; hip extension, abduction, internal rotation, and external rotation strength; knee flexion and extension strength; Q-angle; and navicular drop. All analyses were performed using SAS 9.2 (SAS Institute, Inc, Cary, NC) with an a priori alpha level set at .05.

RESULTS: Sixty-three cadets were diagnosed with PFPS during the follow-up period (34 females, 29 males). None of the biomechanical variables were found to be gender-specific risk factors for incident PFPS ($P \geqslant .05$). Although not significant, risk factors tended to differ between males and females for transverse plane motion at the hip (males: rate ratio [RR] = 1.82; 95% confidence interval [CI]: 0.78, 4.28; $P = .17$ and females: RR = 0.88; 95% CI: 0.36, 2.14; $P = .77$) and knee (males: RR = 0.76; 95% CI: 0.31, 1.88; $P = .56$ and females: RR = 1.78; 95% CI: 0.77, 4.10; $P = .17$).

DISCUSSION: This preliminary analysis revealed that biomechanical risk factors for PFPS may not differ between males and females; however, based on the observable differences in rate ratios, increased hip internal rotation in males and increased knee internal rotation in females seem to influence the risk of incident PFPS. Additional research needs to be performed to confirm the lack of gender-specific risk factors for PFPS in a larger cohort. Future research should also include additional variables, such as psychosocial factors, to determine if these factors may influence the increased risk of PFPS in females.

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MIRROR GAIT RETRAINING FOR THE TREATMENT OF PATELLOFEMORAL PAIN SYNDROME IN FEMALE RUNNERS

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INTRODUCTION: Gait retraining, using real-time kinematic feedback, has been shown to reduce abnormal hip mechanics and pain in female runners with patellofemoral pain syndrome (PFPS).[1] Subjects have been shown to maintain these changes through 1 month postretraining (1MO). However, it is unknown if these changes persist longer. In addition, kinematic gait retraining requires a motion-capture system and does not readily transfer to clinical settings. We sought to determine the effectiveness of a simple gait retraining technique, using a full-length mirror, in female runners with PFPS. We hypothesized that (*a*) subjects would reduce pain and improve function post–gait retraining (POST), (*b*) subjects would reduce their abnormal mechanics during running, (*c*) this new movement skill would transfer to the untrained task of a single-leg squat (SLS), and (*d*) all changes would be maintained through 3 months postretraining (3MO).

METHODS: 8 subjects with PFPS with duration ⩾3 months have been recruited to date. All were running ⩾10 km/wk and had abnormal hip mechanics, defined as peak hip adduction (HADD) ⩾1 SD above a normative database (20.0°). A baseline instrumented analysis was conducted during overground running (3.35 m/s) and during SLS. Data were processed using Visual 3D. Running and SLS variables were indexed to peak values and to 45° of knee flexion, respectively. Pain and the Lower Extremity Functional Scale (LEFS)[2] scores were collected. Subjects underwent 8 sessions of mirror and verbal feedback on their lower extremity alignment during treadmill running. Subjects were asked to reduce their HADD. During the last 4 sessions, mirror and verbal feedback were progressively removed. Instrumented gait and SLS analyses were repeated at POST, 1MO, and 3MO gait retraining. Data were analyzed descriptively.

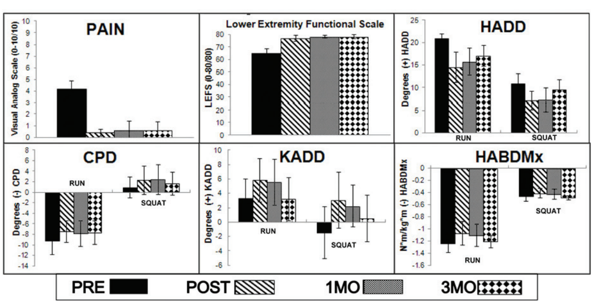

FIGURE. Mean ± SD pain, function (LEFS), running, and SLS mechanics at PRE, POST, 1MO, and 3MO.

RESULTS: At POST, subjects reduced their pain and LEFS scores (**FIGURE**). These levels were maintained through 3MO. For both running and the untrained task of SLS, HADD and CPD were both reduced, while increasing KADD at POST. Due to their improved alignment, a reduction in HABDMx was also noted. These changes were maintained at 1MO. At 3MO, mechanics began to drift toward baseline in the absence of continued feedback.

DISCUSSION: Through 1MO, the improvements due to mirror gait retraining in pain, function, and mechanics are of the same magnitude as those reported after a similar protocol utilizing real-time gait retraining.[1] In both studies, the reduction in abnormal lower extremity mechanics most likely decreased the subjects' Q-angle, resulting in a reduction in lateral tracking of the patella.[3] The transfer of improved hip mechanics to the unpracticed single-leg squat maneuver is an indication of the acquisition of a new motor skill. At 3MO, there was some notable drifting back toward baseline values for both running and SLS mechanics. It is unclear if mechanics would return to baseline levels if subjects were followed for >3MO. Longer follow-ups are needed. Interestingly, pain and function remained improved at the 3MO follow-up, despite the drift in mechanics.

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THE EFFECT OF A SERF STRAP ON PAIN AND KNEE VALGUS ANGLE DURING UNILATERAL SQUAT AND STEP LANDING IN PATELLOFEMORAL PATIENTS

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INTRODUCTION: A valgus position of the knee on functional loading tasks has been reported to be associated with a number of different knee injuries.[1,2] Females have been found to present with greater knee valgus during loading tasks than males,[3] and this is believed at least in part to explain the higher incidence of patellofemoral pain in females.[4] The presence of a valgus knee position has been associated with muscle weakness in the hip abductors and external rotators.[5] Considerable attention has been given to how best to train these muscles, with some success.[5] These neuromuscular changes take time, with training programs lasting between 4 and 6 weeks. External supports have been used to aid performance in a number of lower-limb pathologies, and it would appear logical that a strap would be developed to augment control at the hip, improving lower extremity kinematics. The Stability through External Rotation of the Femur (SERF) strap (DonJoy Orthopaedics Inc, Vista, CA) was developed with the aim of assisting lower-limb kinematics. The purpose of this study is to assess the influence of the SERF strap on lower-limb kinematics and pain.

METHODS: Twelve females with patellofemoral pain (mean ± SD age, 24 ± 3.2 years) participated in the study. Subjects performed a single-leg squat and unilateral step landing task on their symptomatic leg. They performed 3 test trials both with and without the SERF strap for each task. Step landing involved stepping off a 30-cm-high bench and landing onto a mark 30 cm from the bench. Two-dimensional (2-D) frontal projection angle of knee valgus alignment was measured from a digital video image. The angle subtended between the line formed between markers at the anterior superior iliac spine and middle of the tibiofemoral joint and that formed from markers on the middle of the tibiofemoral joint to the middle of the ankle mortise was recorded as the valgus angle of the knee.

While carrying out each task, perceived pain was recorded using a verbal pain scale.[6] The effect of the brace was analyzed using paired *t* tests.

RESULTS: Mean knee valgus angle during single-leg squat was 16.8° ± 5.4°. The application of the SERF brace significantly reduced the knee valgus ($P = .023$): mean decrease in knee valgus was 8.9° ± 2.3°. Pain reported during single-leg squat was reduced significantly ($P = .001$) with the application of the brace. Mean knee valgus angle during single-leg step landing was 13.9° ± 6.8°. The application of the SERF brace significantly reduced the knee valgus ($P = .034$): mean decrease in knee valgus was 6.9° ± 4.1°. Pain reported during single-leg step landing was reduced significantly ($P = .04$) with the application of the brace.

DISCUSSION: Application of the SERF brace would appear to improve knee valgus angle and reduce pain in females with patellofemoral pain, during unilateral functional loading tasks. But caution must be applied when interpreting these results. The mean change in knee valgus angle brought about using the brace during both tests did not exceed the smallest detectable difference (SDD) value previously reported from our laboratory[7] for these tests. The SDD statistic is useful in enabling a clinician to be able to distinguish real changes from meaningless fluctuation; it represents reliability in context of measurement error, with SDD being the minimal change required to be 90% confident that the difference between individual premeasures and postmeasures is due to real change.[8] This would indicate that the differences between the prebracing and postbracing values, in knee valgus, are more likely to be due to measurement error (or random chance) than an actual effect of the brace itself.

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EFFECTS OF ISOLATED HIP ABDUCTOR AND EXTERNAL ROTATOR MUSCLE STRENGTHENING ON PAIN, HEALTH STATUS, AND HIP STRENGTH IN FEMALES WITH PATELLOFEMORAL PAIN

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INTRODUCTION: Patellofemoral pain (PFP) is a common condition for which individuals seek medical care. Although a definitive cause remains elusive, increased hip adduction and internal rotation are common in persons with PFP during functional tasks and are known to adversely influence patella dynamics.[1] Because impaired hip muscle performance has been consistently observed in persons with PFP,[2] hip muscle strengthening has been advocated as an intervention, yet little empirical evidence exists to support this approach. To date, the combination of hip and quadriceps strengthening has been shown to be more effective in reducing symptoms of PFP than quadriceps strengthening alone.[3] However, the effect of isolated hip muscle strengthening in persons with PFP has not been examined. The purpose of the current study was to examine the effectiveness of hip abductor and external rotator strengthening on pain, health status, and hip strength in females with PFP.

METHODS: Twenty-eight females with bilateral PFP were sequentially assigned to an exercise ($n = 14$) or control group ($n = 14$). The exercise group completed bilateral hip abductor and external rotator strengthening 3 times per week for 8 weeks. Pain (VAS: 0-10 cm), health status (WOMAC), and hip strength (handheld dynamometer) were assessed at baseline and at 8 weeks. Pain and health status also were evaluated at 6 months in the patients assigned to the exercise group. Independent *t* tests were used to examine between-group differences at baseline. Separate 2-by-2 ANOVAs with repeated measures were used to determine the effects of the intervention on each outcome variable. Post hoc testing was completed using paired *t* tests to determine whether baseline variables changed with time within group.

RESULTS: Age, height, weight, pain, health status, and strength were not different between groups at baseline ($P>.05$). Significant group-by-time interactions were observed for each variable of interest. Post hoc testing revealed that pain, health status, and strength improved in the exercise group after the 8-week intervention but did not improve in the control group (**TABLES 1** and **2**). At 6-month follow-up, pain and health status in the exercise group remained significantly improved when compared to baseline ($P<.05$) (**TABLE 1**).

DISCUSSION: A program of isolated hip abductor and external rotator strengthening was effective in improving pain and health status in females with PFP when compared to a no-exercise control group. Importantly, the improvements in symptoms in the exercise group were maintained at 6-month follow-up. These findings support the use of hip strengthening as an intervention for persons with PFP.

ACKNOWLEDGEMENTS: This study was presented, in part, at the ACSM Annual Meeting, Denver, CO, June 1, 2011.

TABLE 1 Results of Self-Report Measures Over Time in the Exercise and Control Groups

	Baseline	Postintervention 8 wk	6-mo Follow-up
Exercise			
VAS, cm	7.9 ± 1.7	1.4 ± 1.9*	1.7 ± 2.7*
WOMAC	54.0 ± 18.1	10.7 ± 16.1*	10.8 ± 17.7*
Control			
VAS, cm	6.6 ± 2.0	6.7 ± 2.4	NT
WOMAC	55.9 ± 15.7	59.9 ± 12.6	NT

Abbreviations: NT, not tested; VAS, visual analog scale; WOMAC, Western Ontario and McMaster Universities Arthritis Index.
**Significant at P<.001 from baseline.*

TABLE 2 Results of Strength Assessments in Response to Intervention [N/bwt(N) × 100]

	Baseline	Postintervention 8 wk
Exercise		
Right abduction	11.6 ± 2.3	15.3 ± 2.5*
Left abduction	11.2 ± 2.7	15.9 ± 3.1*
Right external rotation	8.6 ± 2.3	11.8 ± 2.2*
Left external rotation	7.0 ± 1.8	10.9 ± 2.6*
Control		
Right abduction	12.3 ± 2.9	11.2 ± 2.5
Left abduction	12.5 ± 3.7	11.4 ± 3.1*
Right external rotation	8.9 ± 2.1	8.3 ± 2.3
Left external rotation	7.5 ± 1.6	7.3 ± 1.9

Abbreviation: bwt, body weight.
**Significant at P<.05 from baseline.*

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CHANGES IN PATELLOFEMORAL PAIN SEVERITY ACROSS THE FEMALE MENSTRUAL CYCLE

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INTRODUCTION: Patellofemoral pain (PFP) is a common, chronic condition among young, active individuals. A higher incidence in females,[1,2] combined with a tendency toward chronicity,[3] means that PFP can represent a significant burden in females, and impact on participation in a physically active lifestyle. Previous studies have demonstrated that pain sensitivity changes across the female menstrual cycle.[4] In particular, in females with pain conditions such as chronic pain and temporomandibular joint pain, greater pain severity is reported during menstruation.[5,6] Interestingly, no studies have investigated the relationship between menstrual cycle and PFP, despite its impact on females. As such, the primary aim of this study was to determine whether the severity of PFP changes over the various phases of the female menstrual cycle. A secondary aim was to evaluate the influence of the oral contraceptive pill (OCP) on PFP severity over the menstrual cycle.

METHODS: Females with PFP (insidious-onset anterior knee pain greater than 6 weeks in duration, aggravated by activities that load the patellofemoral joint [squatting, stairs]) were recruited from a 12-month randomized clinical trial (RCT).[7] Volunteers were excluded if they had no menstrual cycle (eg, implanon, continuous OCP use, hysterectomy), or an irregular cycle (<21 days or >35 days). Participants completed a baseline questionnaire regarding their menstrual and OCP history. For the duration of the 12-month RCT, participants were asked to record daily data regarding OCP use, whether they were menstruating, and PFP severity (10-cm visual analog scale [VAS]). One normal cycle (28-35 days) selected from each participant was divided into 4 phases[8]: (1) menstrual (7 days from the beginning of menstrual bleeding), (2) preovulatory (from the end of the menstrual phase to the beginning of the postovulatory phase), (3) postovulatory (7 days starting from 14 days before the next menstrual bleeding), and (4) premenstrual (7 days prior to onset of menstrual bleeding). One-way repeated-measures analysis of variance was conducted to compare average pain VAS scores for each phase, with OCP type (none, monophasic, triphasic) included as a between-subjects factor. Significance was set at .05.

RESULTS: Of 100 females enrolled in the RCT, 45 were suitable for inclusion and provided sufficient data for 1 complete cycle. The mean ± SD age at time of study entry was 29 ± 6 years (range, 18-39), and age of menarche was 13.3 ± 1.2 years, indicating that the group had stable menstrual cycles. Eighteen participants were on the OCP during the study (12/18 monophasic, 3/18 triphasic, 3/18 undefined), and had been taking it for 6.9 ± 5.8 years (age started, 19.5 ± 3.3 years). There was a significant main effect for menstrual cycle phase (P = .039), but no significant interaction effect between phase and OCP use or type (P = .41). Pairwise comparisons indicated that pain VAS was significantly higher during the menstrual phase than during the postovulatory phase (mean difference, 0.65; 95% confidence interval [CI]: 0.08, 1.22) and the premenstrual phase (mean, 0.88; 95% CI: 0.26, 1.5).

DISCUSSION: This is the first study to investigate fluctuations in PFP severity over the female menstrual cycle. Findings suggest that, irrespective of OCP use, females with PFP tend to experience more knee pain during the menstrual phase of their cycle, which is consistent with previous findings in other chronic pain conditions. Importantly, mean differences were greater than the standard error of measurement for pain VAS in PFP (0.6 cm).[9] These differences may be explained by decreases in tissue laxity[10,11] and neuromuscular control[12] that have been observed during menstruation in healthy females. Clinicians should consider potential fluctuations in PFP severity over the menstrual cycle, and may need to advise affected women to use pain-relieving interventions (eg, taping, analgesics) and to exercise caution with aggravating activities during menstruation. These findings also highlight the need for clinicians to consider nonmechanical contributors to PFP in female patients at times of heightened pain sensitivity.

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PATELLOFEMORAL PAIN SYNDROME VERSUS NONSPECIFIC KNEE COMPLAINTS IN GENERAL PRACTICE: DIFFERENCES IN PATIENT CHARACTERISTICS, MANAGEMENT STRATEGY, AND OUTCOME AFTER 1 AND 6 YEARS

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INTRODUCTION: In general practice, the diagnosis of painful knee disorders is mainly based on the combination of symptoms and clinical findings and in general demands no further radiological assessment.[1,2] Because clinical findings for patellofemoral pain syndrome (PFPS) and other nontraumatic, nonspecific knee complaints (NSKC) may vary, it is not known which patient characteristics and clinical findings in general practice are associated with the diagnosis of PFPS or with nonspecific knee complaints. Additionally, it is unknown to what extent there is an overlap between the diagnosis of the GP and the often-suggested characteristics of PFPS, that is, peripatellar pain, grinding of the patella, pain on knee flexion, stair climbing, cycling, and running.[3-6] Hence, the aims of this study were: (1) to describe the differences in baseline characteristics of patients who are diagnosed with PFPS compared to NSKC, (2) to describe a set of variables often suggested being indicative for PFPS in relation to the diagnoses of the GP, (3) to describe the difference in outcome between patients with PFPS compared to NSKC, and (4) to describe the differences in types of interventions applied between the patient groups.

METHODS: A prospective, observational cohort study with a follow-up of 6 years was carried out. Patients aged 12 years or above consulting their GP for a new episode of knee complaints were invited to participate in the study. For the present study, only patients with ICPC code L15 (nonspecific knee complaints) and L97.1 (patellofemoral pain syndrome) were included. Those with other ICPC codes were excluded. We refer to this diagnosis of the GP as PFPSgp (patellofemoral pain syndrome, L97.1) and NSKCgp (nonspecific knee complaints, L15). Patients with PFPS diagnosed by the GP (PFPSgp) are compared to nonspecific knee complaints (NSKCgp) and patients fulfilling the clinical criteria for PFPS (PFPScrit) are compared to those not fulfilling these criteria (NSKCcrit). The patient's characteristics, the initial management strategy, and the outcome of PFPS after 1 and 6 years are compared with NSKC in adjusted multivariable analyses.

RESULTS: At baseline, patients in the PFPS group (n = 71) showed a longer duration of complaints (32.4% versus 9.2%; P<.001), had a higher proportion of bilateral complaints (46.5% versus 24.1%; P = .01), and showed more pain at the patellar edge (57.7% versus 41.4%; P = .046) and less pain on knee extension (25.4% versus 29.9%; P = .009) than patients in the NSKC group (n = 87). By combining a set of variables suggested to be indicative for PFPS, only 61% overlap of diagnosis was seen. An active advice by the GP was more often applied by patients diagnosed with PFPS (OR = 2.90; 95% CI: 1.28, 6.55) compared to patients with NSKC. At follow-up, diagnosed PFPS patients showed significantly less recovery (44% and 60%) compared to NSKC patients (66% and 84%) after 1 and 6 years, respectively (OR = 0.41; 95% CI: 0.20, 0.86 at 1 year and OR = 0.24; 95% CI: 0.08, 0.68 at 6 years).

DISCUSSION: The present study confirms the difficulty in diagnosing PFPS in primary care. By excluding anterior knee pain due to intra-articular pathology, plica syndrome, Sinding-Larsen-Johansson disease, Osgood-Schlatter disease, bursitis, tendinopathy, neuromas, and other rarely occurring pathologies, it is suggested that remaining patients with a clinical presentation of anterior knee pain could be diagnosed with PFPS. The present study shows only 60% overlap in patients diagnosed with PFPS by the GP and patients fulfilling the generally accepted clinical criteria for PFPS.[3-6] This implies that, given the almost identical outcomes between PFPSgp and PFPScrit, the diagnosis of the GP is probably related to the initial policy of the GP.

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PATELLOFEMORAL PAIN DURATION IS THE MOST CONSISTENT PREDICTOR OF 12-MONTH PROGNOSIS

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INTRODUCTION: Patellofemoral pain (PFP) has a tendency to become chronic in a proportion of those affected. Prospective longitudinal studies have demonstrated this in active populations such as adolescent females[1] and military personnel.[2] This has obvious long-term implications for participation in daily occupational and physical activities. The current literature provides preliminary data as to baseline clinical factors that are indicative of PFP prognosis. A recent study found that longer duration of PFP most consistently predicted poor outcome over 12 months, with worse scores on pain and function measures also consistent prognostic factors.[3] The aim of the current study was to investigate whether clinical baseline factors such as duration and symptom severity were predictive of poor short- and long-term outcome in a larger international cohort of individuals with PFP.

METHODS: A prospective cohort study utilized data from 2 randomized clinical trials that investigated the effectiveness of exercise (the Netherlands),[4] and multimodal physiotherapy and foot orthoses (Australia).[5] Three hundred ten participants aged 14 to 40 years with insidious-onset PFP of at least 6 weeks' duration were followed up at 3 and 12 months using consistent outcome measures. These were pain severity (worst and during activity) measured on 100-mm visual analog and 11-point numerical rating scales, Kujala Patellofemoral Score (KPS),[6] Functional Index Questionnaire (FIQ),[7] and global perceived recovery (5- or 7-point Likert scale, dichotomized to "marked improvement" or "not improved"). Fourteen possible prognostic factors, measured at baseline, were investigated. Participant characteristics included age, gender, body mass index (BMI), and work type. PFP variables were symptom duration; bilaterality; treatment preference; and baseline scores of usual pain, worst pain, KPS, and FIQ. Study characteristics were recruitment method and allocated treatment. Univariate logistic (global perceived recovery) and linear (pain, KPS, FIQ) regression investigated the association between each potential prognostic variable and outcome at 3 and 12 months. Significant variables (P<.10) were entered in multivariate backward stepwise regression analyses (adjusted for treatment; P<.10). The strength of the predictive ability of identified prognostic factors ($P\leq$.05) in each multivariate model was evaluated with unstandardized regression coefficients (β) for continuous outcomes and odds ratios (ORs) for dichotomous outcomes.

RESULTS: Baseline characteristics indicated that the Dutch cohort (n = 131) were slightly younger, had a lower BMI, participated in more sport, and a higher percentage were not employed. Dutch participants also reported a longer duration of symptoms and worse KPS scores, and significantly more were recruited through health professionals than the Australian cohort. Longer symptom duration was significantly associated with poor outcome at 3 months (pain: 6-12 months, β = 12.3; 95% CI: 3.6, 21.1; >12 months, β = 11.4; 95% CI: 4.0, 18.8; KPS: 6-12 months, β = –5.4; 95% CI: –9.8, –0.9; >12 months, β = –4.4; 95% CI: –8.2, 0.7; recovery: 6-12 months, OR = 0.4; 95% CI: 0.1, 0.9; >12 months, OR = 0.4; 95% CI: 0.2, 0.8) and 12 months (pain: 2-6 months, β = 22.9; 95% CI: 13.3, 32.6; 6-12 months, β = 21.9; 95% CI: 11.3, 32.4; >12 months, β = 24.9; 95% CI: 15.5, 34.4; KPS: 2-6 months, β = –8.7; 95% CI: –13.6, –3.9; 6-12 months, β = –10; 95% CI: –15.3, –4.7; >12 months, β = –11.3; 95% CI: –16.1, –6.6; FIQ: 2-6 months, β = –1.6; 95% CI: –2.6, –0.5; >12 months, β = –1.6; 95% CI: –2.5, –0.7; recovery: 2-6 months, OR = 0.3; 95% CI: 0.1, 0.6; 6-12 months, OR = 0.2; 95% CI: 0.1, 0.6; >12 months, OR = 0.2; 95% CI: 0.1, 0.4). Higher usual pain severity and lower KPS scores at baseline were also significantly associated with poor outcome on multiple measures at 3 and 12 months (P<.05).

DISCUSSION: Findings suggest that long PFP duration is the most consistent predictor of poor short- and long-term outcome on measures of pain, function, and perceived recovery. Furthermore, those with worse symptoms on measures of pain severity and KPS also tend to have a poorer 12-month prognosis. These findings are consistent with previous reports in a smaller cohort,[3] and highlight the importance of preventing chronicity and severity of PFP. Healthcare practitioners should utilize interventions with known efficacy in reducing PFP, such as multimodal physiotherapy,[8] and promote education regarding the natural history and importance of early intervention for PFP to maximize prognosis.

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A CLINICAL STUDY OF THE BIOMECHANICS OF STEP DESCENT USING 3 TREATMENT MODALITIES FOR PATELLOFEMORAL PAIN

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INTRODUCTION: Previous studies have demonstrated that patellofemoral bracing and taping have significant effects on the coronal and torsional mechanics of the knee in both healthy subjects[1] and in patients with patellofemoral pain syndrome (PFPS)[2] and lead to a more controlled eccentric step descent. It is likely that some of the observed effects are due to enhanced proprioception mediated through increased cutaneous stimulation. To investigate this further, we recruited PFPS patients for 2 studies with identical methodologies, the combined results of which are presented here. Study A investigated the effect of bracing and taping; study B investigated the effect of Tubigrip and taping. To our knowledge, this is the first study to investigate the effect of a simple elasticated tubular bandage on the 3-D mechanics of the knee during a controlled eccentric step-down task in a group of patients with PFPS.

METHODS: Twenty-nine subjects with a diagnosis of PFPS (13 men, 16 women) with a mean age of 31 years were recruited (Modified Functional Index Questionnaire score, 24; visual analog scale for usual pain in the past week, 31 mm). Inclusion criteria were presence of traumatic or idiopathic peripatellar pain and pain provoked by deep squatting, kneeling, ascending, or descending stairs, alone or in combination. The exclusion criterion was any history of knee surgery. A step descent was used to assess the control of the knee as the body was lowered as slowly as possible from the step. The step descent was conducted under 4 randomized conditions over the 2 studies: (*a*) no intervention, (*b*) Tru-Pull Advanced sleeve knee brace (DJO, LLC, Vista, CA), (*c*) neutral patella taping, and (*d*) elasticated tubular bandage (Tubigrip; Mölnlycke Health Care, Norcross, GA). For the application of the taping technique, the subjects were sitting with a relaxed, extended knee. One strip of tape of a length equal to 50% of the circumference of the knee was applied without tension across the center of the patella. The tape was not pulled in either the medial or lateral direction. A length of Tubigrip equal to the circumference of the knee was applied. Kinetic data were collected at 200 Hz using 2 AMTI force platforms. A 20-cm step was built on top of 1 of the plates, which was set to 0 prior to data collection. The other plate was embedded in the floor. Kinematic data were collected using a 10-camera Oqus motion-analysis system (Qualisys Medical AB, Gothenburg, Sweden) at 100 Hz. The segments of the lower limbs were modeled in 6 degrees of freedom.[3] The knee joint kinematics were calculated relative to the shank coordinate system. The kinematic data were then quantified from toe-off of the contralateral limb to contact of the contralateral limb. The ranges of knee joint angle in the sagittal, coronal, and transverse planes were found.

TABLE Post Hoc Pairwise Comparisons for the Significant Knee Kinematics in the Coronal and Transverse Planes

	Mean Difference	*P* Value
No intervention: brace		
Coronal ROM	4.50	.005
Transverse ROM	3.15	.046
No intervention: tape		
Coronal ROM	0.83	.001
Transverse ROM	1.19	.052
No intervention: Tubigrip		
Coronal ROM	2.03	.005
Transverse ROM	0.73	.120
Tape: Tubigrip		
Coronal ROM	1.20	.155
Transverse ROM	−0.46	.604
Tape: brace		
Coronal ROM	3.70	.001
Transverse ROM	1.95	.052

Abbreviation: ROM, range of motion.

RESULTS: An ANOVA identified significant changes in the knee range of motion. Post hoc analyses are presented in the **TABLE**. No significant differences were identified in the sagittal plane.

DISCUSSION: Changes were identified in the coronal and transverse plane kinematics of the knee joint between no intervention and bracing, taping, and Tubigrip. Taping and Tubigrip resulted in similar changes in the joint mechanics of the knee in the coronal plane only, whereas the brace produced changes in both the coronal and transverse planes. The results of taping and Tubigrip are interesting, as there was a measurable reduction in coronal plane ROM following these interventions, which do not apply significant mechanical forces to the knee. These data lend support to the argument that these interventions produce subtle yet important improvements in patient control and function and that the observed effects are probably linked to enhanced proprioception. The brace, compared to the other interventions, resulted in significant reductions in ROM in both the coronal and transverse planes. This suggests the brace does have a mechanical effect, which is additional to any proprioceptive effects. The effect of the brace was greater than that of both tape and Tubigrip, but all 3 treatments appear to result in a step descent, which was more controlled compared to no intervention in this group of patients with PFPS.

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THE INFLUENCE OF 2 DIFFERENT BRACES ON PATELLAR ALIGNMENT

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INTRODUCTION: It is generally accepted that during knee extension the patella moves to medial, whereas an unstable patella is characterized by a reduced medial movement.[1] This is connected with an increased risk of a patella dislocation[2] and the development of patellofemoral pain syndrome (PFPS). Because small variations in the patellar alignment could lead to PFPS,[3] the purpose of a treatment is to realign the patella.[4] Therefore, the aim of this study was to investigate the effect of 2 different braces on the alignment of the patella, first in vitro on cadaveric legs and second in vivo on patients with an unstable patella.

METHODS: Two different braces were chosen: Patella Pro (PP) (Otto Bock HealthCare GmbH, Duderstadt, Germany) and a common elastic brace (BA) (GenuTrain P3; Bauerfeind AG, Zeulenroda, Germany). First, to analyze the basic mechanisms of the braces, we accomplished a study with cadaveric legs. Six fresh-frozen cadaveric legs (3 subjects, age 66-72 years) were thawed for 24 hours at room temperature. The legs were not dissected. With the aid of a fixture, each leg underwent 10 flexion/extension cycles through a range of 45° to 0°. During a flexion/extension cycle, the thigh muscles were strained using a tightened strap, which was armed with nails (inserted in the muscles) and fixed on the jig. The legs were tested in a nonbraced condition followed by 2 conditions with braces. Bone pins were screwed into the tibia, femur, and patella, which were armed with an array of 3 retroreflective markers. Anatomical landmarks were pointed using a bar 20 cm long attached with 3 retroreflective markers and related to their segmental bone pin. Kinematic data were obtained (Vicon Motion Systems Ltd, Oxford, UK; 100 Hz). The lateral patellar displacement (LPD) in relation to the femur mediolateral axis was calculated. The mean displacement of 10 extensions was obtained close to full extension (0°). In the second part of the study, 7 patients (mean ± SD age, 34.5 ± 7.6 years) with 7 knee joints with PFPS and unstable patella and 4 healthy knee joints were tested. The subjects performed 1 active squat per condition, standing on both legs with fixed lower legs. Video data were obtained by using a fluoroscope (OEC Fluorostar 7900; GE Healthcare, Waukesha, WI; 30 Hz). The LPD was determined close to full extension (0°) using anatomical landmarks digitized in Vicon Motus for each condition. To find differences in LPD between BA and PP conditions, we used a nonparametric Wilcoxon signed rank test for repeated measures with a significance level of $P<.05$.

RESULTS: The cadaveric study showed in the nonbraced condition a patellar movement to medial (–11.3 mm to –7.5 mm) over the full flexion/extension cycle. The patella was more laterally close to the maximal knee extension (–7.5 mm). For the PP brace, the average LPD close to maximal knee extension was more medial by 0.86 ± 0.90 mm. For the BA condition, the average LPD close to maximal knee extension was more lateral by –0.73 ± 1.41 mm. There was a significant difference in lateral displacement between PP and BA. The study in vivo showed in the nonbraced condition the patella was more laterally close to the maximal knee extension (–4.8 ± 4.9 mm). In relation to the nonbraced condition, we found no significant displacement of the patella for the BA condition in relation to the nonbraced condition. In contrast, there was a significant displacement of the patella toward medial (–2.9 ± 6.8 mm) in the PP condition in relation to the nonbraced and the BA condition.

DISCUSSION: The aim of a brace is to center the patella in the trochlear groove. We found a more medial LPD after bracing with the PP brace and, in contrast to this, a more lateral LPD after bracing with the BA brace. With the help of the presented studies, the mechanism of the PP brace was confirmed and the mechanism of the BA brace was not confirmed. Likewise, 1 study found no significant differences in LPD between a nonbraced condition and the Bauerfeind GenuTrain P3 brace.[4] Compared to studies that investigated the effect of bracing on PFPS,[5] our findings suggest that the use of the BA brace might not be effective in reducing PFPS and the design of the PP brace provides prerequisites to reduce PFPS.

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INTRINSIC RISK FACTORS AND THE EFFECTS OF PROPHYLACTIC BRACING ON THE DEVELOPMENT OF PATELLOFEMORAL PAIN IN MALE SUBJECTS

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INTRODUCTION: General consensus exists concerning the multifactorial nature of patellofemoral pain that leads to similar clinical features.[2] Moreover, broad ranges of pain levels and discomfort are included. Patellofemoral bracing has frequently been used as part of a rehabilitation protocol. Two previous studies demonstrated the positive preventative effects of these devices on the development of PFP.[1,5] The present study aims to identify which intrinsic risk factors can predict PFP. For this, nonbony risk factors are explored and the influence of patellofemoral bracing as a preventative method is investigated.

METHODS: One hundred sixty-nine male officer trainees (17-27 years old) of the first year at the Belgian Royal Military Academy volunteered for the study. The authors focused on the military recruits prior to their 6-week basic military training (BMT). The military physical fitness test, onset timing of the vastus medialis obliquus (VMO) compared to the vastus lateralis (VL), active joint reposition test (AJRT), isokinetic torque of the knee flexors and extensors, leg muscle flexibility, and a short questionnaire were assessed prior to the start of the BMT. The OnTrack (OrthoRx, Inc, San Diego, CA) dynamic patellofemoral brace system was used in this study. This brace consists of knee patches with Velcro (Velcro USA Inc, Manchester, NH) and a neoprene sleeve. The design of the brace is based on the correction of the position of the patella as described by McConnell.[3] A military physician diagnosed all anterior knee pain syndromes during the BMT.

RESULTS: Thirty-one subjects were withdrawn from the study because of different reasons. Forty-three volunteers were assigned to the braced group. Ninety-five recruits served as controls. The mean ± SD age of the 138 subjects in this study group was 19.7 ± 1.82 years and is homogeneous concerning BMI (22.1 ± 2.51 kg/m^2), height (180.4 ± 6.14 cm), and weight (71.9 ± 8.79 kg). After BMT, fewer recruits in the brace group appeared to develop PFP compared to the recruits in the control group ($P = .042$). Out of the 43 recruits in the brace group, 7 (16.3%) developed PFP during the BMT. In the control group (n = 95), 32 recruits (33.7%) developed PFP. Several parameters were significantly ($P<.05$) different between the PFP and healthy subjects at baseline (before the BMT). These significant factors were used to determine prediction models of the development of PFP using stepwise backward logistic regression procedures. The probability of obtaining PFP during the BMT can be expressed as: $P(X) = 1/(1 + e^{-z})$, with $z = 12.053 - (2.146 \times \text{"Q2"}) + (0.129 \times \text{"VMO-VL"}) - (0.079 \times \text{MPFT}) - (0.040 \times \text{PTExt}) - (1.439 \times \text{JPS}) + (2.297 \times \text{Brace})$.

DISCUSSION: The high incidence of PFP (28.3%) in this cohort is notable and has already been reported previously.[4,5] The incidence of PFP in prone individuals could be downsized using patellofemoral bracing if intrinsic risk factors are identified. A "poor" JPS, a delayed timing of the onset of the VMO-VL, a lower knee extensor peak torque, a weak score on the fitness test, and the expectation of sustaining an injury are the variables that are significantly different between healthy subjects and individuals who will develop PFP. The use of the logistic regression model could identify individuals who could derive benefit from patellofemoral bracing during training sessions such as the BMT.

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SHOE INSERTS PRODUCE IMMEDIATE PAIN RELIEF IN INDIVIDUALS WITH PATELLOFEMORAL JOINT OSTEOARTHRITIS

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INTRODUCTION: Patellofemoral joint (PFJ) osteoarthritis (OA) is a distinct clinical entity that results in considerable pain and morbidity in a large proportion of the population.[1] Similarities in symptoms, biomechanics, and observed muscle dysfunction between PFJ OA and patellofemoral pain (PFP) in younger adults suggest that PFJ OA may be a component of the natural history of PFP, although prospective studies to establish this are lacking.[2] As such, efficacious interventions for PFP may also relieve pain associated with PFJ OA. Findings of a recent randomized clinical trial (RCT) suggest that foot orthoses are an effective intervention for PFP.[3] However, the efficacy of this simple, noninvasive intervention has not been investigated in a PFJ OA cohort. This study sought to determine the immediate effects of foot orthoses and flat shoe inserts on pain associated with PFJ OA during performance of functional tasks.

METHODS: Individuals with PFJ OA (osteophytes on skyline radiographs, anterior knee pain during activities that load the PFJ, eg, steps, squatting) were recruited for a within-subjects, repeated-measures, randomized crossover trial. Baseline data included demographics (age, height, weight), severity of symptoms (pain at rest and during activity on 100-mm visual analog scales, Knee Injury and Osteoarthritis Outcome Score [KOOS][4]), and tibial and calcaneal alignment. Participants performed level walking and step-downs under 3 test conditions: (1) running sandals (Strap Runner; Nike, Inc, Beaverton, OR), (2) sandals with prefabricated foot orthoses (Vasyli International, San Rafael, CA), and (3) sandals with flat EVA inserts. After each task and condition, participants rated their knee pain severity on an 11-point numerical rating scale (0 is no pain, 10 is worst pain imaginable). After completion of each test condition, participants rated the sandals alone, foot orthoses, and flat inserts regarding their comfort and support on 100-mm visual analog scales. Repeated-measures analysis of variance and post hoc tests of simple effects examined differences in pain between the 3 conditions for each functional task. Pearson correlations investigated relationships between changes in pain that occurred with orthoses and flat inserts and demographic, symptom, alignment, and comfort measures. Significance was set at .05.

RESULTS: The 23 participants (12 males; mean ± SD age, 52.9 ± 8.9 years; range, 40-75) tended to have varus tibial alignment (−10.5° to 2°; mean, 2.8° ± 3.1°), but were more variable in calcaneal alignment (range, −5° to 5°; mean, 0.4° ± 2.22°). Baseline measures indicated mild to moderate symptom severity (pain at rest, 27.6 ± 26; pain with walking, 24.1 ± 21.1; pain going down stairs, 51.9 ± 24.9; KOOS: pain, 59.3 ± 14.2; symptoms, 44.8 ± 13.7; activities of daily living, 68.1 ± 14.8; sport/recreation, 31 ± 16.2; quality of life, 39.3 ± 13.6). Significant main effects were observed for walking (Wilks λ = 0.737, P = .041; partial η^2 = 0.263) and the step-down task (λ = 0.608, P = .005, η^2 = 0.392). Pairwise comparisons revealed that, compared to the sandal alone, significant reductions in pain occurred during the step-down task for both orthoses (mean difference, −1.28; 95% confidence interval [CI]: −2.13, −0.43) and flat inserts (mean, −1.35; 95% CI: −2.15, −0.55), although the 2 inserts were not significantly different (mean, 0.07; 95% CI: −0.7, 0.82). During walking, significant reductions in pain also occurred for both insert conditions compared to sandals alone (orthoses, −0.78; 95% CI: −1.46, −0.11; flat inserts, −0.83; 95% CI: −1.46, −0.2); however, there were no significant differences between the 2 (mean, 0.04; 95% CI: −0.46, 0.54). Greater body weight was associated with less improvement on stairs with orthoses (Pearson correlation coefficient = −0.59; P = .003) and flat inserts (r = −0.52; P = .011). Older age was associated with greater improvement with orthoses during walking (r = 0.51; P = .018), and female gender with improvement with flat inserts on stairs (r = 0.423; P = .044). There was no significant relationship between effect and height, tibial and calcaneal angle, pain severity, KOOS subscale scores, or comfort or support (P>.05).

DISCUSSION: Study outcomes indicate that shoe inserts, be they prefabricated foot orthoses or flat EVA inserts, can produce immediate and significant reductions in perceived pain during activities that typically aggravate symptoms associated with PFJ OA, irrespective of baseline symptoms and lower-limb posture. Importantly, the magnitude of the change in pain with the step-down task approaches what is deemed to represent a clinically meaningful effect.[5] This indicates that shoe inserts are likely to be an effective intervention for PFJ OA, and warrant further investigation in RCTs to determine longer-term effects. Findings that those with lower body weight experience greater improvements in pain during a task that maximally loads the PFJ suggest that greater success may be achieved by targeting particular individuals, or combining intervention with a weight management program.

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GREATER TREATMENT EFFICACY OF ORTHOSES COMPARED TO A WAIT-AND-SEE APPROACH IN PEOPLE WITH ANTERIOR KNEE PAIN AND MORE MOBILE MIDFOOT

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INTRODUCTION: In-shoe foot orthoses are often used in conjunction with or instead of other conservative treatments for people with anterior knee pain (AKP). While evidence supporting their use is increasing,[1-3] no study has considered natural history as a comparator. As such, no study has evaluated the amount of improvement attributable to an orthosis beyond that of natural history. The majority of studies reporting orthoses to be efficacious have used excessive pronation as part of their inclusion criteria. A randomized clinical trial (RCT) reported orthosis success regardless of foot type.[2] A post hoc examination of this investigation identified midfoot mediolateral mobility as 1 of 4 predictors of increased orthosis success.[4] In contrast, a recent case-control study found the Foot Posture Index, which consists of a battery of measures, had no predictive ability.[1] This suggests that single measures of foot mobility have more value in predicting orthosis success than a battery test. What is not known is whether other foot measures can be used to identify individuals who are likely to improve with the use of orthoses. The primary aim of this RCT was to assess the short-term clinical efficacy of orthoses over natural history in AKP. Second, whether measures of foot posture and mobility are able to predict if an individual will succeed with orthosis therapy.

METHODS: Forty participants with clinically diagnosed AKP were randomly allocated to an intervention or control group. Foot posture and derived mobility measures were obtained using a foot assessment platform, which has been shown to be a valid and reliable measurement tool.[5] The intervention group was assigned a prefabricated contoured orthosis they had ranked the highest from a selection of 3 (soft, medium, and hard contoured) (Vasyli International, San Rafael, CA) after jogging in each. Patient-perceived improvement was measured using a 6-point global improvement scale at the 6-week follow-up. The categories "completely recovered" and "much improved" were considered a success. "Improved," "no change," "worse," and "much worse" were considered nonsuccess. Secondary outcome measures were measured at baseline and follow-up. These were the Kujala Patellofemoral Score (KPS), the Patient Specific Functional Scale (PSFS, a measure of patient-perceived function) and usual and worst pain severity. Dichotomized global improvement was analyzed using the Fisher exact test. Secondary outcome measures were analyzed using ANCOVA with baseline measures and participant characteristics as covariates and group as a fixed factor. Results are expressed as mean (95% CI) and standardized mean difference (SMD). A classification tree was used to determine the sequence of binary decisions for relating a successful treatment to the various foot postures and mobility measures.[6]

RESULTS AND DISCUSSION: Thirty-nine participants completed the study. There were no differences between groups at baseline ($P>.73$). At 6 weeks, the orthosis group success rate, expressed entirely by "much improved," was 47.37% (9/19), compared with 1/20 in the control group ($\chi^2 = 7.086$; $P = .008$). A significant, moderate effect (SMD, 0.71; $P = .002$) in favor of the orthosis group was also found on the PSFS. No other differences were found between groups. This suggests that while participants' function improved, their pain did not. The classification tree identified the important predictor of successful treatment was the presence of orthoses. Following this, a midfoot width difference from weight bearing to non–weight bearing over 11.25 mm most consistently predicted orthosis success. Within the orthosis group, 77.8% of individuals with midfoot mobility >11.35 mm reported their symptoms had much improved. This builds on previous research[4] by suggesting this is the most important foot measure to consider.

SUMMARY: Comfortable in-shoe foot orthoses improve AKP at 6 weeks that are beyond natural history, mainly in the function domain and more so for those with greater midfoot width mobility.

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EFFECTS OF LANDING PATTERN MODIFICATION IN RUNNERS WITH PATELLOFEMORAL PAIN: A CASE SERIES WITH 3 MONTHS OF FOLLOW-UP

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INTRODUCTION: Seventy-five percent of runners strike the ground with a rearfoot strike (RFS) pattern.[1] This landing pattern results in a very distinct vertical impact peak (VIP) that is associated with high rates of loading. These impacts have shown to be related to stress fractures in runners in the past.[2] More recently, RFS runners with a history of PFPS also exhibited increased impacts. It has been demonstrated that runners with a midfoot (MFS) or forefoot (FFS) strike pattern[3] have significantly lower VIP (15.7%) and rates of loading (12.7%-12.9%). Therefore, the purpose of this preliminary study was to examine whether gait retraining aimed at altering footstrike pattern could reduce impacts, as well as symptoms, in runners with PFPS.

METHODS: Three female RFS runners (age range, 26-32 years) with unilateral PFPS were recruited from a local running club. They had been running between 3 and 5 years and were currently running between 20 and 30 mi per week. Subjects ran on an instrumented treadmill at 10 km/h. The ground reaction forces associated with the symptomatic leg were sampled at 200 Hz for 10 seconds. The VIP and the vertical average and instantaneous load rates (VALR and VILR) were averaged across

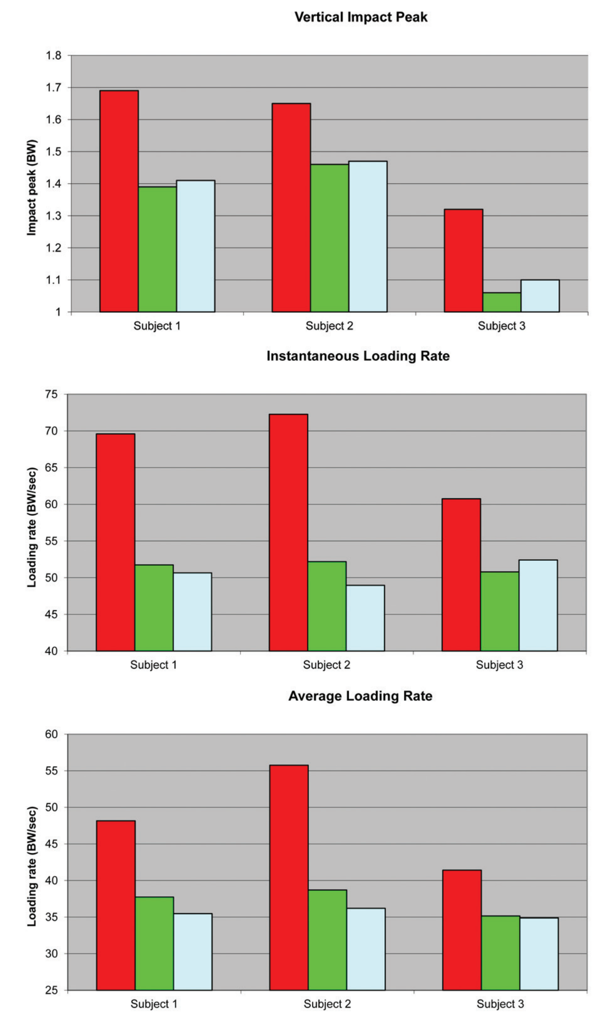

FIGURE. Impact loading results (red = pre; green = post; blue = 3-mo follow-up).

12 contacts in the 10-second period. Additionally, severity of symptoms and the level of functional disturbance were assessed using Kujala[4] and Laprade[5] scales. Participants also reported their best time to complete a 10-km run during the previous month. The participants received 8 sessions of landing-pattern modification training over 2 weeks. A force transducer connected to an audible buzzer was placed under the calcaneus of the symptomatic leg. A warning sound was generated when a runner landed on their heel. Runners were instructed to eliminate the buzzer noise by shortening their stride length and avoiding an RFS landing. The feedback was systematically removed as described by Crowell and Davis.[6] Force data, along with outcome measures, were assessed at the end of the training and again at the 3-month follow-up. Runners were encouraged to utilize their new pattern once the training was complete.

RESULTS: All 3 runners were able to eliminate their heelstrike in 90% of their footfalls. Participants demonstrated impact peak loading reductions following the training program, and they were maintained at the 3-month follow-up (**FIGURE**). Additionally, the Kujala scores were improved by 7 to 18 points across subjects. The Laprade pain score was also improved by 10.4 to 19.5 points. Running performance for 10 km was enhanced in 1 of the participants (**TABLE**).

DISCUSSION: This case series demonstrated a proof of concept that RFS runners with PFPS could be trained to modify their footstrike pattern

TABLE Functional Outcome Measures

Measure	Subject 1	Subject 2	Subject 3
RFS or MFS landings, %			
Pre	0	0	0
Post	93	100	100
3 mo	100	100	93
Kujala scale, % (100 is best)			
Pre	87	72	85
Post	94	85	96
3 mo	94	90	95
Laprade scale, % (100 is best)			
Pre	70.3	59.0	77.3
Post	89.8	69.4	90.9
3 mo	91.2	74.1	92.2
Self-reported best 10-km time in recent mo, min			
Pre	62	67	61
Post	62	66	60
3 mo	62	62	61

Abbreviations: MFS, midfoot strike; RFS, rearfoot strike.

to reduce impact loading. More importantly, these reductions were associated with reductions in pain and improvement in function. It is encouraging that vertical impact loading was reduced in all 3 subjects after the program. These loading reductions (VALR, 15.1%-35.1%; VILR, 3.7%-32.3%) were even greater than those reductions (12%-16%) reported by Altman and Davis.[3] While all 3 participants demonstrated reductions in patellofemoral pain after training, only 1 subject reported improvement in her 10-km run time. Interestingly, this subject presented with the greatest initial level of patellofemoral pain and the slowest 10-km time. Therefore, her success might be due to the fact that she had the greatest room for improvement among the 3 subjects. If impact loading is related to PFPS, mitigating these impacts should lead to a reduced rate of recurrence. While these results show promise for this novel intervention, further study on a larger cohort of PFPS is warranted.

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POSTER PRESENTATIONS

DOES THE CHANGE IN Q-ANGLE MAGNITUDE IN UNILATERAL STANCE DIFFER WHEN COMPARING ASYMPTOMATIC INDIVIDUALS TO THOSE WITH PATELLOFEMORAL PAIN?

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INTRODUCTION: Q-angle is frequently cited as a possible predictor of knee pathology and lower-limb injury.[1] Abnormally high Q-angles in excess of 15° for males and 20° for females are regarded as an anatomical risk factor in the etiology of overuse injuries of the knee, such as patellofemoral joint pain.[1,2] In spite of this, there is currently an inability among health professionals to conclude what should be considered as a "normal" angle.[3] Subsequent to this, questions have arisen as to the validity of linking excessive Q-angles with the occurrence of knee pathologies and other lower-limb injuries, resulting in doubts concerning the diagnostic value of the Q-angle.[4] These findings could be somewhat explained by the methods used for the measurement of Q-angle.[3] One of the reasons for this might be because these studies are assessing Q-angle in bilateral stance. It would appear more logical to assess the effect of unilateral stance on Q-angle, which may give functional validity to this measure. The objective of the study was to determine if Q-angle changes in magnitude from bilateral stance when compared to unilateral stance and then how this change relates to the presence of patellofemoral joint pain.
METHODS: Participants were 60 asymptomatic females (mean ± SD age, 21.9 ± 4.1 years) and 12 females with patellofemoral pain (mean ± SD age, 24 ± 3.2 years). Both groups had their Q-angles measured in bilateral and unilateral stance from images taken using digital photography and then calculated from the ImageJ computer program. Statistical analysis of all data was carried out using SPSS Version 13.0. Paired-samples *t* tests were conducted to compare stance positions and linear regressions were used to formulate predictive equations for Q-angles.
RESULTS: Statistical analysis (paired *t* tests) revealed significant differences between bilateral position and unilateral stance position Q-angles ($P<.005$) for asymptomatic subjects. Unilateral stance causes a significant reduction in Q-angle. The linear regression equations generated from the asymptomatic subjects showed predictive equations and positive correlations for unilateral and bilateral stance Q-angles (r = 0.81-0.89; $P<.001$). The equations generated were used to predict unilateral Q-angle from bilateral Q-angle measurements in 12 patients with patellofemoral joint pain. The actual unilateral Q-angle measurement of the symptomatic knee was significantly greater than that predicted for each individual (P = .01), while the asymptomatic knee showed no significant difference (P = .16) between predicted and actual Q-angle.
DISCUSSION: This study showed a strong positive relationship between bilateral and unilateral stance Q-angles in asymptomatic female subjects, which could be represented in a positive linear regression equation. The linear regression equation was then used to predict the effect on the Q-angle of moving from a bilateral to a unilateral stance. It has been found previously that patients with patellofemoral joint pain, on loading the limb in unilateral stance in activities such as walking and stair descent, have increased knee valgus angle.[5] The current study supports those findings, indicating that when taking up unilateral stance patients with patellofemoral joint pain demonstrate a significantly greater than expected increase in Q-angle, which could increase loading on the patellofemoral joint.

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FRONTAL AND TRANSVERSE PLANE HIP AND KNEE KINETICS AND KINEMATICS DURING RUNNING IN INDIVIDUALS WITH PFPS

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INTRODUCTION: Weakness of the proximal hip musculature has been found in individuals with PFPS[1] and is hypothesized to lead to increased frontal and transverse plane motion of the hip and knee. Alterations in frontal and transverse plane kinematics of the hip and knee have been found

	PFPS	Control	*P* Value
Knee adduction angle	1.4 ± 3.5	2.8 ± 3.3	.241
Knee internal rotation angle	3.2 ± 5.6	3.3 ± 6.1	.947
Hip adduction angle	12.4 ± 4.1	11.7 ± 5.8	.668
Hip internal rotation angle	3.8 ± 4.9	4.5 ± 5.7	.710
Knee abduction moment	−0.96 ± 0.41	−0.88 ± 0.46	.588
Knee external rotation moment	−0.14 ± 0.08	−0.11 ± 0.08	.293
Hip abduction moment	−1.9 ± 0.29	−1.7 ± 0.47	.189
Hip external rotation moment	−0.06 ± 0.04	−0.10 ± 0.12	.166

Abbreviation: PFPS, patellofemoral pain syndrome.

in individuals with PFPS, though findings have been inconsistent. Kinematic changes have been found in walking, running, and jump-landing. However, few studies have examined the hip and knee kinetics in PFPS patients during similar tasks. Knee abduction moment has been prospectively related to developing PFPS[2] and also related to developing patellofemoral joint osteoarthritis.[3] Furthermore, differences in hip and knee kinetics during walking have also been noted between those with and without PFPS[4] and peak knee abduction moment decreases following a hip strengthening intervention in both those with[5] and without[6] PFPS. Therefore, the purpose of this project was to determine if there are differences in frontal and transverse plane hip and knee joint moments and angles during running between individuals with and without PFPS.

METHODS: As part of a larger RCT study, 25 men and women with PFPS (mean ± SD age, 28.4 ± 5.7 years; mass, 70.2 ± 13.9 kg; height, 1.73 ± 0.08 m) participated in the study. The participants met inclusion criteria that are common for PFPS research (pain 3/10 for a minimum of 4 weeks; pain during physical activity, prolonged sitting, jumping, squatting). The control group consisted of 17 men and women (mean ± SD age, 29.4 ± 7.9 years; mass, 68.8 ± 11.7 kg; height, 1.7 ± 0.09 m) who were free from any lower extremity injury and had no history of PFPS or knee surgery. Both the PFPS and control participants were active a minimum of 30 minutes at least 3 times per week. Baseline testing occurred prior to the initiation of any rehabilitation exercises. For the PFPS participants, the most painful knee was tested, and this was matched for the control participants. Three-dimensional kinematic data were collected at 200 Hz and ground reaction force data were collected at 1000 Hz. Participants ran at a consistent speed (4.0-4.5 m/s) wearing standard footwear, and after several practice trials, 5 trials were recorded. Internal joint moments were calculated using an inverse dynamics approach. Knee joint moments were reported in the leg reference frame. Peak joint angle and moment data were extracted from the stance phase and analyzed using repeated-measures ANOVA ($P<.05$). The dependent variables analyzed were hip and knee adduction and internal rotation angles, and hip and knee abduction and external rotation moments. The independent variable was group (PFPS or control).

RESULTS: There were no significant differences between PFPS and control participants in any of the variables analyzed.

DISCUSSION: While differences in knee abduction moment have been seen during walking,[4] no differences were seen during running. Knee abduction moments in this study were higher than those reported by Paoloni et al.[4] Findings of this study are also contrary to those of Stefanyshyn et al,[2] who did report a significantly higher knee abduction impulse in individuals with PFPS. No kinematic differences were seen and contribute to the contradictory nature of this literature. However, knee abduction moment should continue to be examined, as it does appear to be modifiable with strengthening.[5,6] Future research should continue to examine frontal plane knee mechanics, as well as patellofemoral joint mechanics to further understand the etiology of PFPS.

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GLUTEAL MUSCLE ACTIVITY AND PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW

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INTRODUCTION: Abnormal neuromuscular control is frequently considered as a possible etiological factor for patellofemoral pain syndrome (PFPS) in the literature. Traditionally, research has focused on muscle function of the vastii, showing a trend toward delayed onset of vastus medialis obliquus relative to vastus lateralis.[1] More recent research and theoretical analyses[2] have expanded the neuromuscular control focus to include proximal muscle dysfunction. For example, it is theorized that impaired gluteal muscle function may result in increased hip joint adduction and internal rotation during activities such as running, squatting, and stair negotiation. This excessive hip motion is proposed to increase lateral PFJ stress, associated with PFPS development. Supporting this theory, a recent systematic review[3] found that individuals with PFPS exhibit reduced gluteus medius (GMed) and gluteus maximus (GMax) muscle strength. A growing body of work has measured electromyography (EMG) of the gluteal muscles during a range of functional tasks, often reporting differences in onset times, amplitude levels, and/or activity durations between symptomatic and control subjects. A systematic literature review to synthesize these EMG findings and provide guidance for clinical practice and further research was therefore undertaken.

METHODS: MEDLINE, EMBASE, CINAHL, and Web of Knowledge databases were searched from inception until April 2011 for studies evaluating gluteal muscle EMG in individuals with PFPS prospectively or retrospectively. Studies including participants with other knee conditions such as patellar tendinopathy or osteoarthritis, where individuals with PFPS could not be separately analyzed, were excluded. Reference lists and citing articles of included papers were screened for additional publications. All potential publications were assessed by 2 independent reviewers for inclusion and quality using the Downs and Black Quality Index.[4] Means and standard deviations of each variable were extracted or sought from original authors to allow effect size calculations. Sample sizes used, participant demographics, and population sources were also extracted.

RESULTS: Six case-control studies and 1 interventional study that reported baseline EMG for PFPS and control groups were included for final review. No prospective studies were identified. A large amount of heterogeneity in methodological design, inclusion/exclusion criteria, and results was identified. The majority of studies contained low participant numbers and/or absence of a sample size calculation. All 7 studies evaluated EMG activity of GMed, while only 2 studies evaluated GMax. Effect size calculations from studies evaluating GMed EMG indicated that delayed onset and shorter duration of muscle activity exist in some individuals with PFPS during running and stair negotiation. However, delayed onset was not consistent across all studies. Limited research on GMax EMG indicates greater amplitude in some individuals with PFPS during running and stair descent.

DISCUSSION: Findings included in this systematic review were subject to a large level of heterogeneity. This may be the result of heterogeneity in methodological design, inadequate power of some studies, and/or differences in inclusion/exclusion criteria. Additionally, it may also reflect the multifactorial nature of PFPS. The ability to distinguish between cause and effect is impaired by the absence of prospective research evaluating the influence of gluteal muscle activity on PFPS development. Delayed onset and shorter duration of GMed EMG during running and stair negotiation found in 3 of the case-control studies may explain the mechanism behind greater hip adduction and internal rotation reported in some previous PFPS case-control studies evaluating lower-limb kinematics.[5] Greater amplitude of GMax EMG during running and stair descent may indicate an attempt by individuals with PFPS to recruit weakened musculature to reduce/prevent excessive hip motion and subsequent PFJ stress. Evaluation of the effectiveness of intervention strategies (eg, hip muscle retraining) to encourage earlier and longer duration of GMed EMG activity and reduce GMax EMG amplitude is warranted. Future research into the association of gluteal muscle activity with PFPS should recruit larger sample sizes and use more consistent inclusion/exclusion criteria for the recruitment of individuals with PFPS. Additionally, prospective research evaluating the link between gluteal muscle activity and PFPS development is clearly needed.

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PATIENTS WITH PATELLOFEMORAL PAIN SYNDROME (PFPS) HAVE WEAKNESS OF QUADRICEPS AND HIP EXTERNAL ROTATORS AND REDUCED HAMSTRING AND PSOAS FLEXIBILITY

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INTRODUCTION: PFPS is a common knee problem. The cause and specific treatment are debatable.[1] Muscle weakness and tightness are considered major causes.[2,3] We set out to investigate which specific muscles are affected.

METHODS: A series of flexibility and isometric strength tests (Humac Norm Isokinetic Dynamometer) used by physiotherapists were performed in 20 patients with PFPS and 20 matched asymptomatic subjects and repeated after a week. Three tests were excluded (unreliable and not valid). The flexibility tests were Ober and Thomas tests (psoas and quadriceps components), and tests for calf and hamstring flexibility.

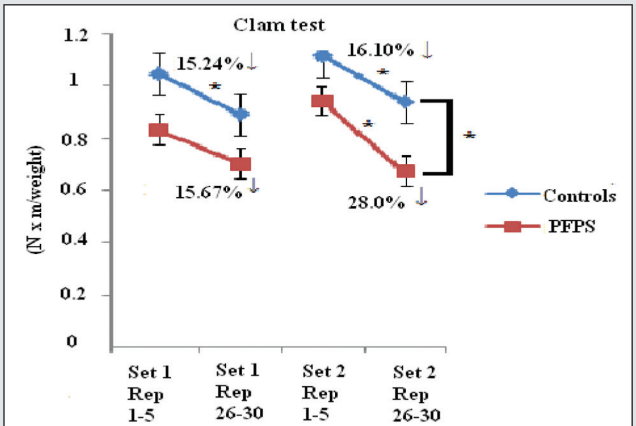

FIGURE. Analysis of first and last 5 repetitions of clam test performance. *Significant difference.

The muscles tested for strength were quadriceps, hip external rotators, hip abductors, and hip abductors with external rotators (clam position). Then a fatigue protocol (2 sets of 30 eccentric reps) of hip abduction and external rotation was carried out in the clam position. Subjects then performed either 3 sets of 20 three-stair descents or 3 sets of 10 one-leg squats. The isometric strength tests were then repeated. One week later, the protocol was repeated. The first series of tests was discarded because of a learning effect. Independent *t* tests, paired *t* tests, and a mixed-model ANOVA were applied to the second series.

RESULTS: The Thomas test (psoas) (18.1%, P = .001) and hamstring flexibility test (18.8%, P = .032) were reduced in the PFPS group compared to controls, with no difference in the Ober, calf, and Thomas (quads) flexibility tests. Strength reduction was found for quadriceps (19.2%, P = .006), external rotators (28.2%, P = .004), hip abductors/external rotators (from clam test position) (42.6%, P = .001) in PFPS subjects, while there was no difference in hip abductor in extension strength. The clam fatigue protocol showed more rapid and more severe fatigability in the PFPS group with a 28% drop in eccentric torque (P<.001) (**FIGURE**). After the 2 functional tasks, the PFPS group showed reduced isometric strength in hip abductors (7.2%, P = .037) and quadriceps (11.3%, P = .048), whereas only quadriceps strength (11.7%, P = .002) was reduced in controls.

DISCUSSION: PFPS patients have significant weakness of quadriceps and external hip rotators. Weakness of hip abduction only became apparent on fatigue testing. Flexibility of psoas and hamstrings was lower. This suggests physiotherapy should be aimed at strengthening quadriceps and external rotators, increasing flexibility of psoas and hamstrings, and that the clam test is useful to demonstrate external rotator weakness and as a therapeutic exercise. The Ober test did not differentiate between the 2 groups.

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THE INFLUENCE OF PATELLOFEMORAL PAIN ON HIP MUSCLE ACTIVATION DURING THERAPEUTIC EXERCISE: A PILOT STUDY USING FINE-WIRE ELECTRODES

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INTRODUCTION: Weakness of the gluteal muscles has been linked to excessive hip internal rotation and adduction, which contribute to patellofemoral pain (PFP).[1,2] When designing a rehabilitation program for persons with PFP, a common goal is to select exercises that activate the gluteal muscles while limiting activation of the tensor fascia lata (TFL). This is important because the TFL is an internal rotator of the hip that has the potential to exert a lateral force on the patella. To date, it is not known which exercises preferentially activate the gluteal muscles while limiting TFL activity, or how the relative activation of these muscles compares between persons with and without PFP. The purpose of this study was to compare the electromyographic (EMG) activity of the gluteal muscles and the TFL between persons with and without PFP while performing various rehabilitation exercises. To accomplish this goal, we utilized a novel method to assess relative EMG activity of the gluteal muscles and TFL (the Gluteal-TFL Activation Index, or GTA). The purpose of the index is to identify exercises that emphasize gluteal activation while limiting TFL activation.

METHODS: Six pain-free persons and 6 with a diagnosis of PFP, between

TABLE GTA Index

Exercise	Control	PFP
UniBRIDGE	76	14
Abduction	49	26
HIKE	23	18

Abbreviations: HIKE, hip hike; PFP, patellofemoral pain; UniBRIDGE, unilateral bridging.

the ages of 18 and 50 years, have participated in the study thus far. Fine-wire electrodes were inserted into the TFL, gluteus medius (GMED), and superior gluteus maximus (SUP-GMAX). Subjects performed maximum voluntary isometric contractions (MVICs) for each muscle. Raw EMG signals were sampled at 1560 Hz with a bandwidth of 35 to 750 Hz. Subjects performed sidelying abduction (ABD), hip hike (HIKE), and unilateral bridging (UniBRIDGE) exercises, with a metronome pacing the movements. The mean root-mean-square (RMS) of the EMG signal in each exercise was normalized to MVIC for each muscle. Simple-type contrast tests (alpha level of .05) were used to compare each gluteal muscle to the TFL for each exercise. The GTA Index was calculated for each exercise in each group to illustrate the comparison between subject groups for how well the exercises activated the gluteal muscles while keeping TFL activity to a minimum. The index was derived from the relative normalized activation ratios of the GMED/TFL and SUP-GMAX/TFL. Each of these ratios was multiplied by its respective gluteal muscle normalized activity, then the totals for each muscle were added together and divided by 2: $\{[(GMED\ /\ TFL) \times GMED] + [(SUP\text{-}GMAX\ /\ TFL) \times SUP\text{-}GMAX]\}\ /\ 2$. The higher the index value, the better the exercise is at emphasizing gluteal activity while minimizing TFL activity.

RESULTS: For UniBRIDGE, the contrast tests showed SUP-GMAX was significantly less than TFL in the PFP group but both GMED and SUP-GMAX were higher than TFL in the pain-free group (resulting in a lower GTA Index in PFP versus control group). For ABD, the contrast tests showed no significant differences among muscles in the PFP group (ie, gluteals were not higher than TFL), but GMED was higher than TFL in the pain-free group, leading to a lower GTA Index in PFP versus pain-free group. For HIKE, SUP-GMAX was less than TFL in both groups. The GTA Index was lower in the PFP group for all exercises, as shown in the **TABLE**.

DISCUSSION: The results of this study to date must be interpreted with caution due to the small sample size; however, data collection is ongoing. Lower TFL EMG and/or higher gluteal muscle EMG yield higher (more desirable) GTA Index values. Based on the contrast test and GTA Index results to date, persons with PFP were unable to activate the gluteals better than TFL across exercises. In particular, TFL EMG was higher and gluteal EMG, especially SUP-GMAX, was lower in the PFP group, yielding a lower (ie, less desirable) GTA Index. This may warrant exercise prescription emphasizing SUP-GMAX activation with training to minimize recruitment of the TFL in persons with PFP. In addition, the pain-free group findings demonstrated that UniBRIDGE was best and HIKE was worst for activating the gluteals while minimizing TFL activity. Based on the findings to date, it appears that persons with PFP may overactivate the TFL and underactivate SUP-GMAX compared to control subjects.

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PATELLAR TRACKING ASSESSMENT IN NAVIGATED TOTAL KNEE ARTHROPLASTY

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INTRODUCTION: The literature reports how total knee arthroplasty (TKA) alters not only normal tibiofemoral joint (TFJ) kinematics[1] but also normal patellofemoral joint (PFJ) kinematics, with consequent frequent PFJ disorders and TKA failure.[2] More precisely, patellar tracking in case of TKA with patellar resurfacing is further affected by patellar bone preparation and relevant component positioning. Within computer-aided techniques, surgical navigation systems (SNS) have been developed for TKA to optimize femoral/tibial component implantation and to track TFJ kinematics before and after relevant trial/final component implantation.[1] The traditional technique used to perform patellar resurfacing, even in navigated TKA, is based only on visual inspection of the patellar articular aspect by clamping a cutting jig and on a simple caliper reading to check for patellar thickness before and after bone cut, thus, without any computer assistance. Although a navigated patellar resurfacing based on patient-specific morphology seems fundamental, this has been completely disregarded until now. To date, its efficacy has only been assessed in vitro.[3] The authors of the present study have developed a new methodology for measuring the effects of every surgical action on PFJ kinematics in navigated TKA. The aim of this study is to report early experiences of in vivo measurements by means of this new methodology in patients undergoing patellar resurfacing in a TKA procedure. A custom SNS was used in parallel to the clinical system to monitor the impact of the surgical procedure on PFJ kinematics; video-fluoroscopy analysis was performed at the follow-up to assess TFJ and PFJ kinematics, the latter according to a novel technique.

METHODS: Ten patients affected by primary gonarthrosis were implanted with a fixed-bearing posterior-stabilized prosthesis (NRG; Stryker Orthopaedics, Mahwah, NJ) with patellar resurfacing. All TKAs were performed using a standard SNS (Stryker Leibinger GmbH & Co KG, Freiburg, Germany). The novel procedure to monitor patellar tracking throughout the procedure using a second custom SNS (PSNS) was approved by the local ethical committee; the patients gave informed consent prior to the surgery. The procedures for standard navigation were performed to calculate preoperative TFJ deformities and kinematics.[1] The PSNS was used for relevant patellar reference-frame definition and PFJ kinematics assessment.[2] After standard procedures for femoral/tibial component implantation, the patellar cut level and effect of patellar resurfacing on PFJ kinematics were assessed with the support of the PSNS. Pre/post-TKA radiographs were used to check for the patellar thickness and final lower-limb alignment. The patients were analyzed also at 6-month follow-up by standard video-fluoroscopic analysis for 3 motor tasks. The latter includes a novel technique for tracking the patella as well by means of fiducial tantalum beads inserted into the patellar component before the implantation. Clinical assessment was performed using the International Knee Society (IKS) score preoperatively and at follow-up.

RESULTS: The in vivo patellar tracking technique was performed successfully in all cases without complication, resulting in 30-minute-longer TKA. Intraoperative passive PFJ kinematics after implantation correlates well with postoperative follow-up active kinematics throughout a wide range of relevant motor tasks. The latter replicated intraoperative findings, even if reasonable discrepancies were observed, mainly due to the differences between passive and active conditions. However, depending on the case, PFJ kinematics was, or was not, within the reference values recently reported by the authors of the present study.[4] The need to navigate patellar preparation in the future was also clearly corroborated by observed discrepancies in thickness of up to 5 mm between PSNS-and caliper-based measurements.

DISCUSSION: These findings, obtained for the first time by navigated and fluoroscopic techniques, support relevance, feasibility, and efficacy of patellar tracking in in vivo–navigated TKA. The encouraging in vivo results may lay ground for the design in TKA of a standard navigation system that also includes patellar-based measurement for a more comprehensive assessment of the original whole-knee anatomy and kinematics. This may be of good value for patellar component positioning in case of resurfacing. It may also be helpful in nonresurfacing, because a femoral preparation/implantation that also takes into account patellar tracking may better restore the native PFJ kinematics, if this is normal.

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EMG DECOMPOSITION OF VASTUS MEDIALIS AND VASTUS LATERALIS IN NORMAL SUBJECTS AND PATELLOFEMORAL PATIENTS: A NEW WAY OF ASSESSING THE BALANCE OF MUSCLE FUNCTION?

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INTRODUCTION: Much has been published on the electromyographic (EMG) differences between vastus medialis (VM) and vastus lateralis (VL); this work has mainly focused on the timing differences in the onset of muscle activation using surface EMG.[1] However, little has been reported on the frequency of the EMG signals, which can indicate differences in motor unit firing patterns.

METHODS: This study used surface EMG signal decomposition technology to explore the properties of numerous simultaneously active motor units. EMG decomposition was conducted on VM and VL during a weight-bearing closed-kinetic-chain isometric "squat" task in pathology-free individuals and case studies of patients with patellofemoral pain. EMG signals were collected with 2 five-pin surface array sensors, each providing 4 channels of data, which were placed over the belly of VM and VL. Each channel was then sampled at 20 KHz using a modified 16-channel Bagnoli EMG system (Delsys Inc, Boston, MA). The signals were then decomposed into the constituent action potentials. The mean firing rate for each motor unit action potential train, the number of peaks per second (PPS), was then calculated.

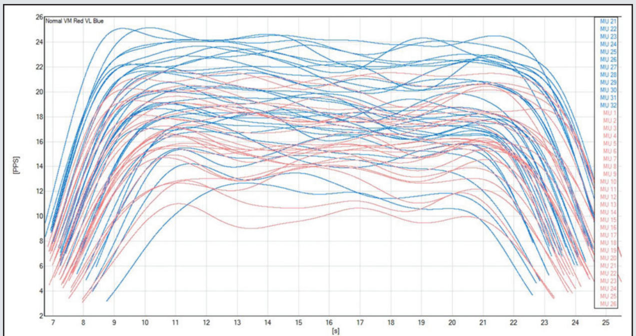

FIGURE 1. Normal subject VM in red and VL in blue, showing overlap in VM and VL firing rates, peak per second (PPS).

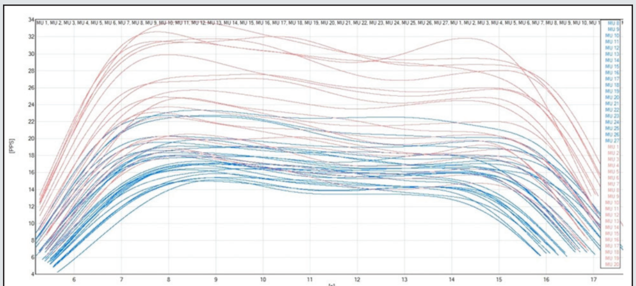

FIGURE 2. Patellofemoral pain syndrome patient VM in red and VL in blue, showing differences in VM and VL firing rates, peak per second (PPS).

RESULTS: The results from the normal subjects support previous findings of VM and VL EMG decomposition with approximately equal firing rates of VM and VL (**FIGURE 1**). However, the results in the patellofemoral patients show clear differences between the firing rates in VM and VL, with VM having significantly greater firing rates than any previously published data from normal subjects[2] (**FIGURE 2**).

DISCUSSION: The ability to conduct surface EMG signal decomposition is a recent technological development. The elevated firing rate measured in the VM in this study could be explained in a number of ways. For example, it could be an indicator of localized muscle fatigue in the VM or it could indicate a change in recruitment pattern of the motoneuron pool; either of these explanations could contribute to patellar maltracking. Although exploratory at this time, these differences in motor unit recruitment patterns between healthy subjects and patients with patellofemoral pain syndrome could represent an important future outcome measure of knee control when treating patellofemoral pain. Certainly this is an area worthy of further study.

ACKNOWLEDGEMENTS: Delsys Inc for the loan of the EMG decomposition equipment.

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DEVELOPMENT OF A DOCUMENTATION TOOL TO DEFINE AND QUANTIFY REHABILITATION

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INTRODUCTION: Rehabilitation is a complex intervention consisting of multiple treatment modalities that aim to help individuals maximize functional performance. Despite numerous studies evaluating the clinical effectiveness of rehabilitation, it is difficult to develop specific guidelines because of research design limitations. This includes inadequate control or delivery of multiple treatment modalities so that the treatment effects of individual interventions are unknown.[1,2] To improve rehabilitation of patellofemoral pain syndrome (PFPS), higher-quality studies are needed, but this requires a better definition of rehabilitation content and quantification of treatment. The aim of this investigation was to develop a tool for documentation and quantification of rehabilitation content for

TABLE 1 Papers Included

Rehabilitation Concept	Number of Papers (PFPS Papers)
Electrotherapy	3 (1)
Exercise therapy	30 (2)
Manual therapy	2 (1)
Mixed modality	6 (3)
Tape	1 (0)

Abbreviation: PFPS, patellofemoral pain syndrome.

TABLE 2 Modalities Frequently Used in PFPS Rehabilitation

Modality	Frequency of Use	Modality	Frequency of Use
Gluteal strengthening	67%	Rehab advice	42%
Stretches	62%	Pacing	38%
PFJT mobilizations	60%	Taping	38%
Exercise advice	60%	Squats	38%
Quadriceps strengthening	56%	Ultrasound	29%
Function/sport-specific exercise	51%	Proprioception	27%
		Core stability	27%
Neuromuscular control training	51%	Soft tissue release	20%
Vastus medialis obliquus strengthening	44%		

Abbreviations: PFJT, patellofemoral joint; PFPS, patellofemoral pain syndrome.

knees (TRAK), using a taxonomy of rehabilitation concepts and interventions to comprehensively describe standard care.

METHODS AND RESULTS: A mixed-methods approach was used. This was a 5-stage inductive process combining scientific evidence with expert clinical opinion.[3] Stage 1: reviewing the literature systematically. This evaluated the clinical effectiveness of rehabilitation modalities for the management of knee conditions. From an initial keyword search, 158 articles were identified; 42 were of sufficient quality and met the inclusion criteria. Five rehabilitation concepts were identified and are listed in **TABLE 1**, along with the number of articles for that concept. This was used to inform the structure of TRAK and the open question style used in stage 2. Stage 2: UK-wide questionnaire to physiotherapists specializing in knee rehabilitation. Three hundred physiotherapy departments across the UK were contacted. Physiotherapists specializing in knee rehabilitation were asked to complete the online survey. Physiotherapists were asked about their treatment of 3 knee conditions, including PFPS. Based on these results, 9 rehabilitation concepts were identified: cardiovascular exercise, coordination exercise, flexibility, functional exercise, strengthening, electrotherapy, taping, manual therapy, and advice and information. Individual rehabilitation modalities were listed under each of the concepts. Only interventions reported as being used more than 10% of the time were included. The most frequently used modalities in PFPS rehabilitation are listed in **TABLE 2**. A time scale with 5-minute increments was added for each concept to quantify treatment.[3] Stage 3: clinician and manager feedback. Open questions identified the need for free-text space. Stage 4: piloting TRAK in Cardiff and Vale University Health Board. Six physiotherapists of varying experience piloted TRAK; 55 treatment sessions were documented. Content analysis based on frequency of using modalities identified that some intervention terms could be combined. Advice and information, functional exercise, and strengthening were the rehabilitation concepts that clinicians spent the most time on. Stage 5: clinician feedback based on piloting. Space to document clinical diagnosis and future treatment plan was requested. TRAK can be viewed at: http://www.cardiff.ac.uk/sohcs/contactsandpeople/a-h/button-kate-dr-overview_new.html.

DISCUSSION: TRAK is a tool that has been developed to document and quantify the content of rehabilitation for knee conditions, including PFPS. It is a taxonomy of treatment concepts and modalities that ensures the standardization of terminology and could be developed into an electronic patient record. It can be used in clinical practice to define "standard" care and achieve a better understanding of rehabilitation content, so that successful treatment strategies can be identified. This information can be used to develop new interventions and design higher-quality randomized control trials. Physiotherapists use a range of modalities to treat PFPS. The frequency of use of certain modalities is not inline with the clinical effectiveness literature. Therefore, a better understanding of standard care for PFPS is required. The delivery of PFPS treatment may need to be revised if further piloting confirms that providing advice and information is the concept that physiotherapists spend most time on.

ACKNOWLEDGEMENTS: Research Capacity Building Collaboration Wales.

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WHAT DOES PREDICT THE FUNCTIONAL OUTCOME AFTER REHABILITATION IN PATIENTS WITH PATELLOFEMORAL PAIN SYNDROME?

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INTRODUCTION: Due to the multifactorial origin of patellofemoral pain syndrome (PFPS), many rehabilitation programs with various emphases have been proposed to treat this mainly chronic disorder. The effect of therapy is often with varying success.[1] Moreover, it is unclear why some

TABLE The Relationship Between the Predictive Variables and the Outcome Variable (Kujala Score) (n = 36)

	Correlation Coefficient (*r*)	*P* Value
Age	−0.09	NS
Gender	−0.13	NS
Body mass index	−0.15	NS
Duration of symptoms	−0.21	NS
Frequency of pain	−0.48	NS
Intensity of worst pain	−0.38	NS
Clicking sensations	−0.29	NS
Pain during walking	−0.44	NS
Pain ascending stairs	−0.52	NS
Pain descending stairs	−0.47	NS
Pain during squatting/kneeling	−0.31	NS
Pain during prolonged sitting	−0.36	NS
Pain in daily life	−0.58	.03
Pain during isokinetic test	−0.44	NS
Kujala score (pretreatment)	0.51	NS
Step test ascending pain	−0.16	NS
Step test descending pain	0.09	NS
Single-legged hop test	0.33	NS
Triple hop test	0.43	NS
Average peak torque concentric, 60°/s	0.20	NS
Average peak torque concentric, 240°/s	0.18	NS
Average peak torque eccentric, 60°/s	0.18	NS
Average peak torque eccentric, 240°/s	0.19	NS
Cross-sectional area VMO	0.17	NS
Cross-sectional area total quadriceps	0.42	NS

Abbreviations: NS, nonsignificant; VMO, vastus medialis obliquus.

patients benefit from a specific treatment while others don't. Therefore, more insight is required into the factors that could predict the outcome. The objective of this study was to identify predisposing factors for the functional short-term outcome after rehabilitation for PFPS.

METHODS: Thirty-six patients with PFPS (16 male and 20 female; mean ± SD age, 23.8 ± 6.7 years) completed a 7-week rehabilitation period. The treatment included neuromuscular coordination, stabilization and strengthening exercises with emphasis on the vastus medialis obliquus (VMO), stretching, and home exercises. Prior to the start of the rehabilitation program, the patients were evaluated on the following measurements: pain during rest and activities, step test, single-legged hop test, triple hop test, concentric and eccentric knee extensor strength at 60°/s and 240°/s, and the anatomical cross-sectional area (CSA) of the quadriceps muscle and in particular of the VMO measured by magnetic resonance imaging. Four baseline factors (age, gender, body mass index, duration of symptoms) were also investigated for their prognostic ability on outcome assessed at 7 weeks. The success of the treatment was evaluated by the subjective and functional Kujala anterior knee pain scale.[2]

RESULTS: The **TABLE** shows the correlation coefficients between the various parameters evaluated prior to treatment (predictors) and the posttreatment outcome obtained by the Kujala score. The linear regression analysis identified a statistically significant correlation between the Kujala score at 7 weeks posttreatment (functional outcome) and the pain in daily life ($P = .03$). The less pain the patients experienced in daily life before treatment, the better the functional outcome was after 7 weeks of therapy ($r = -0.58$). Inclusion of any other variables did not significantly improve the prediction obtained by linear regression ($P>.05$).

DISCUSSION: Only the pain in daily life could be determined as a predisposing variable to the functional outcome after a rehabilitation program in patients with PFPS. This is in agreement with previous research that demonstrated that higher baseline pain severity was associated with poor outcome at follow-up.[2] Pain may restrict muscle strengthening as well as functional improvement. The results suggest that a quick pain relief should be specifically targeted during the treatment of patients with PFPS along with the advice to avoid pain-provoking activities. Nevertheless, there may be other factors of a physical or psychological nature that are stronger predictors of outcome for PFPS.

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FUNCTIONAL MEASURES FOR PATELLOFEMORAL PAIN: WHICH ONE IS MOST RESPONSIVE?

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INTRODUCTION: Patellofemoral pain (PFP) is a common, chronic condition aggravated by activities that load the patellofemoral joint, such as squatting, stair walking, and running. As such, it can significantly impair function, impacting participation in daily activities and exercise. Conservative interventions such as patellar taping and vasti retraining aim to minimize pain and disability.[1] To determine the efficacy of such interventions, clinicians and researchers require reliable, valid, and responsive tools to measure change. While previous studies have reported the responsiveness of measures of pain and disability,[2] evidence regarding measures of function alone is lacking in this patient population. Considering the abundance of functional measures described in the PFP literature, it is important to establish which are the most responsive to guide future research and clinical practice. The aim of this study was to investigate the responsiveness of patient-reported outcomes (PROs) and physical tests of function in individuals with PFP over a 12-month period.

METHODS: One hundred seventy-nine individuals with an insidious onset of PFP (>6 weeks) were entered into a 12-month randomized clinical trial (RCT).[3] A blinded assessor took functional measures at baseline, 6 weeks, 3 months, and 12 months. PROs included the Functional Index Questionnaire (FIQ),[4] Lower Extremity Functional Scale (LEFS),[5] and Patient-Specific Functional Scale (PSFS).[6] Physical tests were the number of pain-free step-ups and step-downs from a 25-cm step, and the number of pain-free squats.[7] Responsiveness statistics were calculated for each follow-up time (PASW Statistics 18.0). Effect sizes were calculated as the mean change score (mean post – mean baseline), divided by the baseline standard deviation (SD), and standardized response means (SRM) by dividing the mean change score by the SD of the change score.[8] Intraclass correlation coefficients (ICCs) were calculated using test-retest data from 10 participants. Standard error of measurement (SEM) was calculated as the SD of follow-up scores, multiplied by the square root of 1 – ICC, and the minimal detectable change (MDC_{95}) as the product of 1.96, the SEM, and the square root of 2.8. MDC was expressed as

TABLE Measures of Responsiveness for Functional Measures in PFP

	FIQ	LEFS	PSFS	Step-up	Step-down	Squat
6 wk						
ES	1.19	0.82	2.66	1.42	1.05	0.92
SRM	0.95	1.00	1.55	0.91	0.67	0.61
MDC_{95} (%)	4.35 (27)	7.50 (9)	0.90 (9)	1.79 (7)	4.71 (19)	1.63 (7)
3 mo						
ES	1.46	1.08	3.22	1.66	1.40	1.41
SRM	1.17	1.32	1.76	0.98	0.78	0.79
MDC_{95} (%)	4.37 (27)	7.69 (10)	0.95 (10)	1.86 (7)	5.43 (22)	2.56 (10)
12 mo						
ES	1.80	1.33	3.68	2.44	2.23	1.97
SRM	1.30	1.41	1.77	1.46	1.16	1.01
MDC_{95} (%)	4.65 (29)	6.87 (9)	1.02 (10)	1.77 (7)	5.48 (22)	3.19 (13)

Abbreviations: ES, effect size; FIQ, Functional Index Questionnaire; LEFS, Lower Extremity Functional Scale; MDC, minimal detectable change; PFP, patellofemoral pain; PSFS, Patient-Specific Functional Scale; SRM, standardized response mean.

a percentage of the maximum total score to facilitate comparisons between measures.

RESULTS: Of the PROs, the PSFS demonstrated the largest effect sizes and SRMs over 12 months (**TABLE**), indicating greater responsiveness than the FIQ and LEFS. The MDC_{95} for the FIQ was substantially greater than the LEFS and PSFS when expressed as a percentage of the total possible score (30% versus 10%), suggesting greater error associated with the FIQ. Of the physical tests, effect sizes and SRMs were greatest for the step-up test across 12 months. The step-up test also consistently had the lowest MDC_{95} over 12 months (7%), with the step-down test demonstrating the largest amount of error (approximately 20%).

DISCUSSION: Findings suggest that, of the 6 measures investigated, the PSFS is most responsive to change over a 12-month period in those with PFP. When considered alongside the FIQ and LEFS, this may be due to the patient-specific nature of the functional tasks represented in the PSFS, which are likely to reflect those commonly aggravating in PFP. In comparison, the LEFS was intended for general lower-limb conditions, and as such contains activities that are often not pain provocative for PFP, such as putting on shoes and socks. Although the FIQ contains more activities applicable to PFP, it has been shown by a number of studies to have substantial associated error. Despite reports that walking down steps is more aggravating in PFP, findings demonstrate smaller effect sizes and higher error associated with the step-down test. Although the squat test was found to have a similar MDC_{95} to the step-up test, it was not as responsive over 12 months. Researchers and clinicians who choose to utilize a single measure of function for PFP should select the PSFS, and include the step-up test if a physical measure of function is desired.

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EARLY INTERVENTION FOR ADOLESCENTS WITH PATELLOFEMORAL PAIN SYNDROME

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INTRODUCTION: Knee pain is highly prevalent among adolescents, and a large proportion of knee pain can be attributed to patellofemoral pain syndrome (PFPS).[1] While treatment for PFPS may be successful for the short term, long-term results are less promising. In a recent review[2] covering the long-term prognosis for patients diagnosed with PFPS, it was reported that only one-third of those diagnosed with PFPS and treated conservatively will be pain free and about one-quarter will stop participating in sports because of knee pain. Predictors of long-term outcome (>52 weeks) indicate that a long symptom duration[3] and higher age[4] are associated with a poorer outcome after treatment. These prognostic factors suggest that an early initiation of treatment could lead to a better long-term prognosis. The purpose of this study is to examine the short- and long-term effectiveness of multimodal physiotherapy compared to standard wait-and-see treatment applied at a very early state of disease among adolescents.

METHODS: Online questionnaires will be forwarded to 1600 students aged 12 to 19 years in 2 local lower secondary schools and 2 local upper secondary schools. The questionnaires will contain questions regarding knee pain and general musculoskeletal pain, activity level, and quality of life measured by using EQ5D. Students who report knee pain will be called by telephone and offered a clinical examination by an experienced rheumatologist. The inclusion and exclusion criteria used will be identical to those used by Collins et al.[5] Subjects who are diagnosed with PFPS will be offered to participate in the study. One hundred four students diagnosed with PFPS will be cluster randomized into 2 groups based on which school they attend. One group will receive patient education given by a physiotherapist about the diagnosis and how to avoid painful activities. The intervention group will receive the same patient education as the control group combined with supervised multimodal physiotherapy consisting of patellofemoral joint mobilization, patellar taping, quadriceps muscle retraining, and instructions on home training for a period of 3 months. The multimodal physiotherapy intervention will be carried out at school premises right after the end of class. Compliance of home training will be monitored through weekly follow-up by short message system (SMS). Those who do not wish to participate and to be randomized into 1 of the 2 groups will be followed through an observational cohort. The observational cohort will be followed at the same time points and they will be asked which treatment they have received. Follow-up self-report questionnaires will be filled out by the patients at baseline and at 3, 6, 12, and 24 months after inclusion in the study. The primary outcome measure is perception of recovery after 12 months measured on a 7-point Likert scale ranging from "completely recovered" to "worse than ever."[6] Patients are categorized as recovered if they rate themselves as "fully recovered" or "strongly recovered." Patients rating themselves as "slightly recovered" to "worse than ever" are categorized as "not recovered." This threshold will be used to dichotomize perceived recovery into 2 categories: "recovered" and "not recovered."[6] Secondary outcomes include the change from baseline to each point of follow-up in the average score for 4 of the 5 KOOS subscales, covering pain, symptoms, difficulty in sports and recreational activities, and quality of life. Sample-size calculations are based on a 30% difference between the 2 categories "recovered" and "not recovered."[6] Sample-size calculations show that 42 subjects in each group are needed to detect a statistical difference ($\alpha = .05, \beta = .1$). With an estimated dropout rate of 25%, 52 students will be included in each group. Between-group comparison will be analyzed on an intention-to-treat basis. Comparison of the primary dichotomous outcome will be analyzed through logistic regression for repeated measurement.

DISCUSSION: This study has been designed after reviewing the literature on exercise therapy for PFPS. It was concluded that a possible way to address the poor long-term results was to apply intervention at a very early state of the disease. The research will address the effectiveness of supervised multimodal physiotherapy versus standard care in patients with PFPS.

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THE INFLUENCE OF PUBLISHED EVIDENCE ON PHYSIOTHERAPISTS' CLINICAL REASONING WHEN TREATING PATELLOFEMORAL PAIN SYNDROME: A MIXED-METHODS STUDY

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INTRODUCTION: Etiology of patellofemoral pain syndrome (PFPS) is considered multifactorial, with various intrinsic and extrinsic factors thought to contribute to development and chronicity. As a result, a vast array of conservative treatment options have been proposed and made available to physiotherapists to address these factors. Choosing the most appropriate course of treatment for an individual with PFPS requires sound clinical reasoning, incorporating the best available evidence and therapist clinical experience. This study aimed to evaluate the link between current evidence for the treatment of PFPS and the clinical reasoning processes used by experienced physiotherapists.

METHODS: Recent research publications on conservative interventions for the treatment of PFPS, including a high-quality systematic review[1] and the consensus statement produced following the first Patellofemoral Pain Retreat in 2009,[2] were summarized. Additionally, a purposive sample of 8 experienced physiotherapists (9-25 years) working in the United Kingdom from a variety of clinical and academic backgrounds were interviewed. The interview was semistructured, using a topic guide covering current evidence for conservative interventions when treating PFPS.

RESULTS: Based on the best available current evidence, provision of multimodal physiotherapy intervention was found to be the gold standard. Specifically, this should consist of vastii and gluteal strengthening, patellofemoral joint mobilization and taping, and lower-limb stretching. Additionally, patient-specific advice and education should be provided. Consideration should also be made to the inclusion of prefabricated foot orthosis prescription, acupuncture, weight reduction, and gait retraining. Thematic analysis showed knowledge and incorporation of this evidence across the participating physiotherapists to range from very weak to very strong. As a result, the influence of the evidence base and clinical reasoning varied. From the established evidence, the implementation of patient-specific advice and education by the treating physiotherapist was consistently considered to be the most important. Clinical reasoning behind the implementation of other treatment components was frequently stated as dependent on individual patient presentation. Common treatments considered by treating physiotherapists included vastii retraining, PFJ mobilizations and taping, and lower-limb stretching. Gluteal strengthening, foot orthosis prescription, and acupuncture were less frequently considered. Additionally, implementing strategies to reduce participants' weight was rare, and often avoided due to an implied sensitivity of the topic. A number of barriers inhibiting the use of evidence-based practice when treating PFPS were identified, including (1) limited knowledge of current evidence, (2) variable access to published research, (3) time available for professional development, and (4) limited external validity of research carried out to apply to patients seen clinically. A number of treatment interventions possessing anecdotal validity but lacking evidence were also highlighted. These included icing for short-term pain relief, activity modification and strategies to return to previous function, footwear advice, and addressing psychosocial factors.

DISCUSSION: Consistent with the research literature, physiotherapists consider PFPS a multifactorial condition, which requires varying treatment approaches depending on the individual. Above all other treatment options, physiotherapists place greatest emphasis on the importance of patient-specific advice and education. The variability across physiotherapists in the use of remaining evidence for the treatment of PFPS during the clinical reasoning process should be addressed to ensure optimal patient outcomes. This will require strategies to improve knowledge of current evidence, access to published research, and time available to physiotherapists to complete professional development. Additionally, due to the multifactorial nature of the condition, identification of subgroups likely to respond to various interventions may improve the external validity of future research. Based on anecdotal evidence, further research is recommended to establish the efficacy of icing, activity modification and strategies to return to previous function, patient education, footwear advice, and addressing psychosocial factors.

ACKNOWLEDGEMENTS: We would like to thank all the physiotherapists who took the time to be interviewed for this project. We also thank the Nuffield Foundation for funding the research.

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FACTORS ASSOCIATED WITH PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW

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INTRODUCTION: The most frequently diagnosed condition in adolescents and adults with knee complaints is patellofemoral pain syndrome (PFPS).[1] It is suggested that the etiology of PFPS is multifactorial.[1,2] However, there is no agreement with regard to which factors contribute to or are related to PFPS.[3] In the literature, the focus on the etiology of PFPS varies. These associations aim at the etiology of PFPS as well as the consequences of PFPS. However, there is a lack of overview of these factors. Therefore, the aim of this study is to systematically summarize the factors associated with PFPS.

METHODS: A search was conducted in PubMed, EMBASE, Web of Science (WoS), and the Cochrane Central Register. Prospective studies including ≥20 patients with PFPS and that examined at least 1 possible association with PFPS were included. Studies focusing on other named pathologies were excluded. An assessment list was created to evaluate the quality of the included studies. A meta-analysis was performed to establish associations that had a consistent definition, and whether results were reported for the same outcome measures. Significant differences were based on calculated mean differences (MDs) with matching 95% confidence intervals (CIs). For dichotomous data, odds ratio (OR) or relative risk (RR) with matching 95% CI were calculated or abstracted from the individual studies. If a meta-analysis was not possible due to clinical heterogeneity, data were analyzed descriptively.

RESULTS: Of the 3845 potentially relevant articles, 37 were included in this review. The 37 studies examined in total 380 variables and pooling was possible for 4 variables. The pooled data showed significantly less hip abduction strength (% body weight) and a significantly larger Q-angle (WMD, 1.33; 95% CI: 0.31, 2.35 and –3.41; 95% CI: –5.61, –1.21, respectively) in PFPS patients compared to controls. Hip extension rotation strength and foot arch height index were not associated with PFPS (WMD, –1.21; 95% CI: –2.43, 0.01 and 0.01; 95% CI: 0.00, 0.03, respectively). Although pooling was not possible, 2 studies found lower knee extension peak torque at 60°/s in PFPS patients compared to controls (MD, –21.40; 95% CI: –34.49, –8.31 and –56.50; 95% CI: –81.07, –31.93). Other variables that were significantly associated with PFPS were based on single studies.

DISCUSSION: This review examined the factors associated with PFPS. The 37 included studies evaluated 380 variables for PFPS. Pooling was possible for 4 variables and a significantly larger Q-angle[4-10] and significantly lower hip abduction strength[10,11] were found in the PFPS patients compared to controls. No difference was found for arch height index[5,12] and hip external rotation strength.[10,11] A meta-analysis for the other 376 evaluated variables was not feasible, because of the difference in outcome measures, methodological measurements, missing data, and statistical heterogeneity. A total of 27 studies evaluated more than 1 variable per 10 cases, which could lead to overfitting.[13] Two studies examined the association between knee extension strength and PFPS expressed by peak torque; although pooling was not possible due to statistical heterogeneity, both studies found less knee extension strength in PFPS patients compared to controls.[5,14] We conclude that a larger Q-angle, less hip abduction strength, and less knee extension strength expressed by peak torque were associated with PFPS. Because several factors associated with PFPS were described in single studies, they require further research.

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RISK FACTORS FOR PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) is the most frequently diagnosed condition in patients with knee complaints younger than 50 years. The cause of PFPS is reported to be multifactorial.[1] Because of the high incidence of PFPS, especially among athletes, prevention of PFPS is important. The first step toward prevention is identification of possible risk factors or factors associated with PFPS. Therefore, this review systematically outlines the risk factors for PFPS.

METHODS: A search was conducted in PubMed, EMBASE, Web of Science, and the Cochrane Central Register. Prospective studies including $\geqslant$20 patients with PFPS and that examined at least 1 possible risk factor for PFPS were included. Studies focusing on other named pathologies were excluded. An assessment list was created to evaluate the quality of the included studies. A meta-analysis was performed to establish risk factors that had a consistent definition, and whether results were reported for the same outcome measures. Significant differences were based on calculated mean differences (MDs) with matching 95% confidence intervals (CIs). For dichotomous data, odds ratio (OR) or relative risk (RR) with matching 95% CI were calculated or abstracted from the individual studies. If a meta-analysis was not possible due to clinical heterogeneity, data were analyzed descriptively.

RESULTS: Of the 3845 potentially relevant articles, 7 were included in this review. These studies examined a total of 135 variables and pooling was possible for 13 potential risk factors. The pooled data showed that relative extension peak torques by body weight at 60°/s and 240°/s (MD, –0.24; 95% CI: –0.39, –0.09 and –0.11; 95% CI: –0.17, –0.05, respectively), by body mass index at 60°/s and 240°/s (MD, –0.84; 95% CI: –1.23, –0.44 and –0.32; 95% CI: –0.52, –0.12, respectively), and in a concentric mode at 60°/s and 240°/s (MD, –17.54; 95% CI: –25.53, –9.54 and –7.72; 95% CI: –12.67, –2.77, respectively) were significantly lower in the PFPS group compared to controls and were therefore risk factors for PFPS. For the other risk factors that showed significant differences between PFPS patients and controls, pooling was not possible, because these factors were described in single studies.

DISCUSSION: This review examined the risk factors for PFPS. The 7 included studies evaluated 135 variables as potential risk factors for PFPS. This number of variables is noteworthy because only 243 PFPS patients were included in these 7 studies. None of the studies adhered to "the rule of 10."[2] Pooling was possible for 13 variables: height, weight, BMI, age, Q-angle, and peak torques (8 variables). The results of this systematic review show that female gender,[3] less quadriceps and gastrocnemius flexibility,[4] less knee extension strength, and a lower extension peak torque are risk factors for PFPS.[3,5,6] It is remarkable that the focus of the 7 studies is mainly on biomechanical and neuromuscular risk factors and rarely on structural (or static) risk factors. Moreover, structural anomalies and lower extremity malalignment are often examined as associative factors for PFPS in case-control studies.[7-9] We conclude that a lower knee extension strength expressed by peak torque seems to be a risk factor for PFPS in both men and women. Because several risk factors for PFPS were described in single studies, these risk factors, as well as those with conflicting evidence, need to be confirmed in future studies.

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COHORT STUDIES IN PATELLOFEMORAL PAIN SYNDROME: THE SEARCH FOR MODIFIABLE RISK FACTORS: A SYSTEMATIC REVIEW AND CRITIQUE

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INTRODUCTION: A better understanding of the etiology of patellofemoral pain syndrome (PFPS) may facilitate optimization of preventative and treatment strategies. Adequately designed cohort studies not only provide information on the strength of the association between putative risk factors and the condition under study but also establish temporality of the association. We undertook a systematic review of cohort studies of PFPS risk factors.

METHODS: We searched AMED, EMBASE, MEDLINE, CINAHL, and SPORTDiscus limited to English-language publications. Key words are shown below (**TABLE 1**). Abstracts were examined by 2 investigators independently with differences resolved by a third. Studies were considered eligible if they investigated risk factors for PFPS in adolescents and/or young adults (16-40 years of age) using a retrospective or prospective cohort design. For the purposes of this study, PFPS was defined as anterior knee pain or retropatellar pain in the absence of other specific pathology. Reference lists of identified studies and reviews were checked for additional studies. For each identified study, information on the design, risk of bias, and results was extracted onto a specially designed form by 2 investigators independently, with disagreements resolved by a third.

RESULTS: The search identified 2187 abstracts, from which 11 eligible studies were identified.[1-11] All studies were prospective and the oldest was published

TABLE 1 Key Words for Electronic Search Strategy

Arthralgia
Patella
Patellofemoral or patella-femoral
Anterior knee pain
Patellofemoral pain syndrome
((Patellofemoral or patella-femoral) adj (pain or syndrome or dysfunction))
((lateral compression or lateral facet or lateral pressure or odd facet) adj syndrome)
Chondromalacia patella
((Chondromal$ or chondropath$) adj (knee or patell$ or femoro$ or femoro-patell$ or retropatell$ or retro-patell$))

TABLE 2	Types of Intrinsic Risk Factors Investigated
Hip and knee kinetics and kinematics	
EMG activity of quadriceps muscle	
Hip and knee muscle strength	
Muscle tightness	
Lower-leg alignment	
Leg-length differences	
Q-angle	
Navicular drop	
Ankle movements	
Foot pronation	
Foot architecture	
Plantar pressures	

in 1983. Of the 11 studies, 8 were undertaken in military groups,[1-4,7-9,11] 2 in runners,[5,6] and 1 in students undertaking physical education classes.[10] The number of participants varied between 62 and 1319 subjects,[1,2] with follow-up periods ranging from 6 weeks to 3 years.[2,4,7,8] PFPS was defined in most studies (n = 10), but in these the definition of PFPS varied. In 6 studies, it was clearly stated that subjects were free of PFPS at study commencement. The methods by which incident cases of PFPS were ascertained varied, with some studies relying on self-report or self-referral to a clinician and others on regular clinical evaluation. In 7 studies, it was unclear whether ascertainment of PFPS had been assessed blind to exposure status. Most studies provided a clear rationale for the risk factors under investigation, but there was little discussion about how these fitted into the causal pathway for the development of PFPS or were modifiable. One study investigated the association between sports participation and PFPS,[9] and 1 investigated psychological factors.[10] However, intrinsic and biomechanical factors were most investigated. The diversity of factors is highlighted in **TABLE 2**. Similar factors were often determined using different techniques.

DISCUSSION: There is still a need for well-designed prospective studies in general populations that focus on investigating associations between modifiable risk factors and the development of PFPS. This requires some further consideration of the causal pathway for PFPS.

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THE ADDITIONAL EFFECT OF ORTHOTIC DEVICES ON EXERCISE THERAPY FOR PATIENTS WITH PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW

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INTRODUCTION: Patellofemoral pain syndrome (PFPS) is a common diagnosis in young active adults leading to limitations in physical activities. Possible treatment options for PFPS are exercise therapy and orthotic devices. Until now, there has been no overview on the additional effect of orthotic devices over exercise therapy on pain and function. Therefore, the aim of this study was to determine the effectiveness, in measures of pain and function, of a physiotherapeutical intervention consisting of exercise therapy and orthotic devices or exercise therapy and placebo orthotics compared with exercise therapy only for patients with PFPS. Orthotic devices in this review include patellar bracing, patellar taping, and foot orthotics.

METHODS: A systematic literature search was conducted in MEDLINE, CINAHL, EMBASE, Cochrane, and PEDro. Randomized controlled trials (RCTs) and controlled clinical trials (CCTs) of patients diagnosed with PFPS that evaluated at least 1 clinically relevant primary outcome measure (eg, pain, functional status, recovery) were included. Treatment had to include exercise therapy combined with orthotics and the control intervention had to include an identical exercise program with or without a sham orthotics. Methodological assessment was done using the sources of risk of bias assessment as suggested by the Cochrane Library. Data were summarized using a best-evidence synthesis. Effect sizes (ES) were calculated by dividing the mean difference between O and C by the pooled SD of the baseline scores, when available.

RESULTS: From the 153 articles retrieved from the search, a total of 8 RCTs fulfilled the eligibility criteria.[1-8] Three of the 8 included studies had a low risk of bias. The results show that there is moderate evidence for no additive effectiveness of knee braces to exercise therapy on pain (ES varied from −0.14 to 0.04) and conflicting evidence on function (ES = −0.33). There is moderate evidence for no difference in effectiveness between knee braces and exercise therapy versus placebo knee braces and exercise therapy on pain and function (ES varied from −0.1 to 0.10). There is conflicting evidence for the additive effectiveness of tape and foot orthotics to exercise therapy on pain and function (ES varied from −0.22 to 2.89). Finally, limited evidence was found that tape and exercise therapy are more effective on pain and function compared to placebo tape and exercise therapy (ES varied from 1 to 3).

DISCUSSION: There is no additional effect of knee braces over exercise therapy regarding pain and function outcomes for patients with PFPS. The evidence for the additional effect of tape and foot orthotics on exercise therapy is conflicting when compared to exercise only. The combination of tape and exercise seems to be preferable when compared to placebo tape and exercise. This conclusion is based on a small number of high-risk-of-bias studies. There are inconsistencies in pain and function measures used. Furthermore, there was no heterogeneity of exercise protocols and type of braces that were used. In further research, this should be changed to make the studies comparable and to create a body of evidence on the possible additional effect of knee braces on exercise therapy for patients with PFPS.

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EXERCISE THERAPY FOR PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW: COCHRANE DATABASE OF SYSTEMATIC REVIEWS

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INTRODUCTION: Exercise therapy is frequently used in the treatment of

patellofemoral pain syndrome (PFPS) and is believed to be an effective means in reducing pain and restoring function of patients. Although PFPS is under scope of research, for the past 25 years the mechanism of exercise therapy on the condition and its effectiveness remain unclear. Its effectiveness was systematically reviewed in 2003, showing limited evidence with respect to pain reduction and conflicting evidence on improving function.[1] The review called for larger and methodologically more sound studies to draw conclusions upon the effectiveness of exercise therapy. Our review complements the above review to reassess the effectiveness of exercise therapy and also includes over 10 clinical trials that have been undertaken after the first review. The aim of the study is to evaluate the short- (3 months or less) and longer-term effects of exercise therapy, specifically aimed at reducing pain intensity and improving function and/or recovery in patients with PFPS.

METHODS: Design: systematic review. We searched the Cochrane Bone, Joint and Muscle Trauma Group Specialized Register (December 2009), the Cochrane Central Register of Controlled Trials (The Cochrane Library 2009-4), MEDLINE, EMBASE, and other databases to December 2009. Inclusion criteria: (1) RCTs comparing exercise therapy for PFPS (AKP) with placebo/no treatment/different conservative strategies, and (2) evaluation of at least 1 relevant outcome (pain, knee function, and recovery). Two reviewers independently selected studies and extracted data on study characteristics, risk of bias, and outcomes. When appropriate, data were pooled using the random-effect model. Mean differences (continuous data) and risk ratios (dichotomous data) with 95% confidence intervals (CIs) were calculated.

RESULTS: Twenty-three trials were included in the review, involving 1503 participants with age ranging from 14 to 40 years. At short term (–1.52; 95% CI: –2.29, –0.76) and long term (–1.30; 95% CI: –2.15, –0.46), exercise therapy shows to be effective on pain reduction compared to no intervention. At short term, exercise therapy is effective on improving knee function (5.69; 95% CI: 0.70, 10.67). At long term, these effects are not significant. The effects of exercise therapy are not clearly reflected on the outcome measures for recovery. At short term, exercise therapy is more effective on pain reduction than other conservative strategies such as brace, tape, or insoles (–9.26; 95% CI: –17.47, –1.05). No significant differences in effect were found when comparing exercise strategies for quadriceps muscle strengthening with other exercise strategies such as closed versus open kinetic chain, hip abductor, or abdominal muscle exercise.

DISCUSSION: In the update of the literature, we found 13 newly published RCTs meeting the inclusion criteria and they were added to this review. Seven studies published beyond the date of the first review were classified as low risk of bias (RoB ≥6), but 6 studies still suffered from methodological drawbacks (RoB <6), mainly because of improper allocation methods, no reporting of the study design, or dealing with intention-to-treat analysis. In general, the quality of the studies improved after the review in 2003.[1] However, in the update of the review, only 9 of the 23 studies included were classified as high quality (RoB >6). The review provides evidence that exercise therapy is beneficial for patients with PFPS when compared to no treatment (wait-and-see approach). Exercise therapy is effective in reducing knee pain at short and long term and improving knee function at short term following an exercise protocol that contains at least strengthening exercises of the knee muscles. The significant improvement of pain and function scores is clinically relevant, resulting in approximately 30% to 40% more pain reduction than for usual care, with a mean effect size of 0.55 for pain and 0.45 for function for short term. The clinical effectiveness is supported by a recent study that suggests that supervised exercise therapy is also cost-effective.[2]

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PATELLAR TAPING FOR PATELLOFEMORAL PAIN SYNDROME IN ADULTS: RESULTS FROM THE COCHRANE REVIEW

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INTRODUCTION: When patellofemoral pain syndrome (PFPS) is clinically diagnosed, physiotherapy is the mainstay of treatment and often includes patellar taping. The effect on pain relief after applying tape can be instantaneous, but there have been few analyses on taping as part of a short-, medium-, or long-term rehabilitation program. The objectives of this Cochrane review were to investigate the efficacy of patellar taping as part of an exercise rehabilitation regimen on pain and function in adults with PFPS.

METHODS: I searched the Cochrane Bone, Joint and Muscle Trauma Group Specialized Register, the Cochrane Central Register of Controlled Trials, MEDLINE (1950 to present), CINAHL (1982 to present), EMBASE (1980 to present), PEDro, SPORTDiscus (1830 to present), AMED (1985 to present), and reference lists of articles. I also searched for theses via the following databases available through the University of Manchester and University of Central Lancashire libraries: the Thesis Canada Protocol, the Australian Digital Thesis Program, and ProQuest. For conference proceedings, I searched the Chartered Society of Physiotherapy in-house library catalog. For ongoing trials, I also searched using the metaRegister of controlled trials at Current Controlled Trials. To identify unpublished trials, I contacted experts in the field. There were no language restrictions. In MEDLINE (OVID ONLINE), the search strategy was combined with the first 2 sections of the optimal MEDLINE search strategy for randomized controlled trials (Higgins 2005). Similar strategies (also using OVID ONLINE) were used for CINAHL, AMED, EMBASE, and SPORTDiscus.

RESULTS: Twelve trials were included, with a total of 772 participants. All used patellar taping as part of an exercise program for PFPS. Sample sizes were modest, with the largest trial having 179 subjects, but only 91 (2

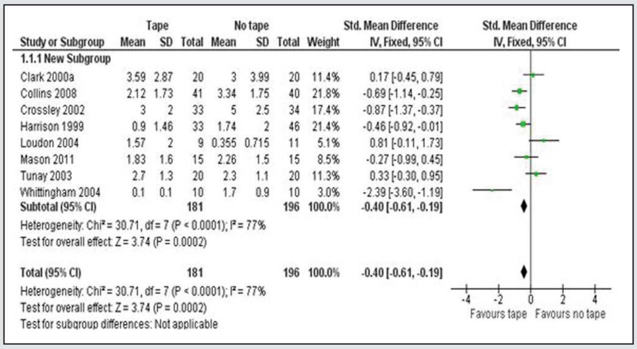

TABLE Key Words for MEDLINE Search Strategy

Arthralgia/ Patella/. ((patellofemoral or patello-femoral) adj (joint)). anterior knee pain.tw. Patellofemoral pain syndrome/. ((Patello-femoral or patellofemoral) adj (pain or syndrome or dysfunction)).tw. ((lateral compression or lateral facet or lateral pressure or odd facet) adj (syndrome)).tw.

Chondromalacia patellae/. ((chondromal$ or chondropath$) adj (knee or patell$ or femoropatell$ or femoro-patell$ or retropatell$ or retro-patell$)).tw (taping or tape$). tw. strap$.tw. (McConnell and (knee$ or patell$)).tw.. randomized controlled trial.pt. controlled clinical trial.pt.

Randomized Controlled Trials/. Random Allocation/. Double Blind Method/. Single Blind Method/. Animals/ not Humans/. clinical trial.pt. exp Clinical Trials/. (clinic$ adj trial$).tw. ((singl$ or doubl$ or trebl$ or tripl$) adj (mask$ or blind$)).tw. Placebos/. placebo$.tw. random$.tw. Research Design/

groups) being evaluated as part of this review. Meta-analysis was performed on the VAS outcome measure (**FIGURE**) but was problematic due to heterogeneity. The FIQ permitted meta-analysis with 3 trials and the anterior knee pain (Kujala) score with 2 trials. These 2 meta-analyses showed a benefit of taping with exercise over just exercise without tape. The risk of bias assessment revealed that the lack of blinding of the patient and of the therapist posed the greatest risk to the study protocol.

CONCLUSION: This Cochrane review has considered trials using taping as part of an exercise regime. There is evidence from the FIQ outcome at 4 weeks that using taping as part of an exercise program for PFPS is more beneficial than not using tape. This benefit is not seen in the Kujala score, nor the WOMAC score at any time points, nor the FIQ at 6 weeks. The VAS also shows benefit but suffers from heterogeneity.

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THE EFFICACY OF PATELLAR TAPING IN INDIVIDUALS WITH PATELLOFEMORAL PAIN SYNDROME: A SYSTEMATIC REVIEW

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INTRODUCTION: Patellar taping is frequently used during the treatment of patellofemoral pain syndrome (PFPS) as part of multimodal treatment. Pain reduction has been hypothesized to be achieved via changes to neuromuscular control (improved vastii function) and/or patellofemoral joint (PFJ) kinematics (reduced lateral PFJ compression). Warden et al[1] published a high-quality systematic literature review and meta-analysis on patellar taping and bracing for chronic knee pain, covering the literature until November 2006. The review concluded that medially directed patellar taping provided a clinically meaningful improvement in knee pain. However, it did not differentiate findings related to PFPS from other causes of anterior knee pain such as patellar tendinopathy or osteoarthritis. Additionally, the effects of patellar taping on neuromuscular control or PFJ kinematics were not evaluated. Due to these limitations and the high number of recent publications, an up-to-date systematic review and meta-analysis evaluating the effects of patellar taping on pain, neuromuscular control, and PFJ kinematics is warranted.

METHODS: MEDLINE, CINAHL, SPORTDiscus, Google Scholar, Web of Science, and EMBASE databases were searched from inception until February 2011 for randomized or quasi-randomized studies evaluating the effects of patellar taping on pain, neuromuscular control, and/or PFJ kinematics in individuals with PFPS. Studies that included participants in their sample with other knee conditions such as patellar tendinopathy or osteoarthritis were excluded. The reference lists and citing articles of included papers were screened for additional publications of interest. All potential publications were assessed by 2 independent reviewers for inclusion and quality using the Downs and Black Quality Index.[2] Means and standard deviations of each variable (pain, neuromuscular, or kinematic) were extracted from included publications or sought from original authors to allow effect-size calculations. Sample sizes used, participant demographics, and population sources were also extracted.

RESULTS: Ten studies of varying quality were included for final review. However, adequate data to complete effect-size calculations could not be collected from all studies. Two studies investigated medium- to longer-term effects of taping on pain (4-52 weeks), and 8 studies investigated the immediate effects of taping on pain, neuromuscular control, and/or PFJ kinematics. Effect-size calculations showed that perceived pain was significantly reduced at 4 weeks by medially directed taping in combination with exercise compared to placebo taping combined with exercise, and exercise alone. However, medially directed taping in combination with education did not improve outcomes at 12 or 52 weeks compared to education alone or combined education and exercise. In the immediate term, medially directed patellar taping was found to significantly reduce pain during a range of functional tasks compared to no tape and placebo tape. When evaluating neuromuscular control, patellar taping was found to significantly reduce vastus medialis obliquus (VMO) to vastus lateralis (VL) ratio, and produce earlier VMO onset timing. Only 1 study evaluated the effects of patellar taping on PFJ kinematics, reporting a significant inferior shift of the patella in relation to the femur.

DISCUSSION: Evidence suggests that patellar taping provides an effective means of pain relief in individuals with PFPS in the immediate term. Medium- and long-term effects on pain were more variable. Limited longer-term follow-up of patellar taping in individuals with PFPS indicates outcomes may be improved when it is used as an adjunct to exercise, but not education. However, further research is needed to confirm these findings because they stem from single trials. Additionally, further high-quality randomized trials with long-term follow-up evaluating the efficacy of patellar taping as an adjunct or alternative treatment to other evidence-based interventions such as multimodal physiotherapy, foot orthosis prescription, and acupuncture are needed. Although further research is needed, possible neuromuscular mechanisms behind patellar taping efficacy may be reduced levels and earlier onsets of VMO EMG activity. There is currently a paucity of research evaluating the effects of patellar taping on PFJ kinematics, which needs to be addressed considering its emphasis in the theoretical rationale for patellar taping.

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