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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.[2] Females are reported to be at higher risk for the development of PFP than their male counterparts.[3] 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 have 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 focused on the contribution 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 were 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 titled “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 to 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, all of 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 upon 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.

References

1. Blond L, Hansen L. Patellofemoral pain syndrome in athletes: A 5.7 year retrospective follow-up of 250 athletes. Acta Orthop Belg. 1998;64:393–400. [PubMed: 9922542]

2. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: The joint undertaking to monitor and prevent ACL injury (JUMP-ACL cohort. Am J Sports Med. 2009;37:2108–2116. [PubMed: 19797162]
3. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. Gender differences in the incidence and prevalence of patellofemoral pain syndrome. Scand J Med Sci Sports. 2010;20:725–730. [PubMed: 19765240]
4. Stathopulu E, Baildam E. Anterior knee pain: A long term follow-up. Rheumatology. 2003;42:380–382. [PubMed: 12595641]
5. Taunton JE et al. A retrospective case-control analysis of running injuries. Br J Sports Med. 2002:36:95–101. [PubMed: 11916889]
6. Wood L, Muller S, Peat G. The epidemiology of patellofemoral disorders in adulthood: A review of routine general practice morbidity recording. Prim Health Care Res Dev. 2011;12:157–164. [PubMed: 21457600]

Consensus Statement: Local Factors

Consensus Statement: Distal Factors

Consensus Statement: Proximal Factors

Consensus Statement: Interventions

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,[8,27,56] 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/or maltracking (pathomechanics) results 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.[18] 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 as an expert opinion (Level of evidence = V) 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[19] and a combination of innervated tissues may be involved concurrently.[7,23,31] These structures include subchondral bone, infrapatellar fat pad, quadriceps tendon, patellar tendon, synovium, the medial and lateral retinaculum,[24,45] 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.[1,14,40]

4. Proprioception appears to play an important role in the dynamic stability of the patellofemoral and tibiofemoral joints[12] and a decrease in proprioception has been noted for patients with post-traumatic 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 experience in the previous year (r=0.55).[17] The latter study excluded patients with pain who demonstrated no bone metabolic activity, thus the strength of the correlation is suspect. Lastly, 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 0.05, 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,[3] where 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).[25] 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.[17,37] 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 towards 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 inter-subject 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.[5,32,48,61] One potential source for this inter-subject 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.[15]

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.[10,28,42] 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.[26] 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° flexion there is no required active quadriceps force and this force requirement increases as

the knee extends.[26] 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,[16,20] indicating that sub-millimeter accuracies are necessary to keep the errors in estimating patellofemoral contact kinematics within acceptable limits. Few techniques that can non-invasively quantify in vivo patellofemoral kinematics/alignment have reported accuracies to such a level.[4,6,22]

19. Based on modeling studies, patellofemoral joint and cartilage stress is significantly greater in individuals with PFP compared to controls.[21]

Bracing and Taping

20. Patellar taping has been shown to reduce pain[60] and alter patellofemoral kinematics.[15]

21. Taping has been shown to improve knee joint proprioception in individuals experiencing PFP who were rated as having poor proprioception.[12] 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[15] 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 un-taped state.[15]

Alterations in the Quadriceps

24. Quadriceps weakness and atrophy[30] and vastus medialis obliquus (VMO) atrophy[39] has been associated with idiopathic PFP, but evidence to the contrary has also been reported.[11]

25. While impaired VMO function (as assessed by EMG signal magnitude and timing) has been implicated in PFP,[13,59,63] this finding has not been consistent across all studies.[9,41]

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 earlier anatomical study.[2]

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 in an attempt 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 experience by these individuals and those 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 affect 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. As *in-vivo* measures of patellofemoral cartilage stress are unavailable, there is a need to develop neuro-musculoskeletal 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.[10,54]

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 sequelea of PFP.

13. The inter-relationships between proximal, distal, and local factors needs to be better understood.

14. Taping and bracing have been shown to improve proprioception at the knee.[12] Future work is needed to determine if this improvement in proprioception can be directly correlated with improved knee function or a reduction in PFP.

References

1. AMerskey HBN. Part III Pain Terms. Classification of Chronic Pain. Seatle, WA: IASP Press; 1994:209–214.
2. Amis AA. Current concepts on anatomy and biomechanics of patellar stability. Sports Med Arthrosc. 2007;15:48–56. [PubMed: 17505317]
3. Amis AA, Oguz C, Bull AM, Senavongse W, Dejour D. The effect of trochleoplasty on patellar stability and kinematics: a biomechanical study in vitro. J Bone Joint Surg Br. 2008;90:864–869. [PubMed: 18591593]
4. Behnam AJ, Herzka DA, Sheehan FT. Assessing the accuracy and precision of musculoskeletal motion tracking using cine-PC MRI on a 3.0T platform. J Biomech. 2011;44:193–197. [PubMed: 20863502]
5. Belvedere C, Leardini A, Ensini A, Bianchi L, Catani F, Giannini S. Three-dimensional patellar motion at the natural knee during passive flexion/extension. An in vitro study. J Orthop Res. 2009;27:1426–1431. [PubMed: 19444875]
6. Bey MJ, Kline SK, Tashman S, Zauel R. Accuracy of biplane x-ray imaging combined with model-based tracking for measuring in-vivo patellofemoral joint motion. J Orthop Surg Res. 2008;3:38. [PubMed: 18771582]
7. Biedert RM, Sanchis-Alfonso V. Sources of anterior knee pain. Clin Sports Med. 2002;21:335–347. [PubMed: 12365231]
8. Boling M, Padua D, Marshall S, Guskiewicz K, Pyne S, Beutler A. Gender differences in the incidence and prevalence of patellofemoral pain syndrome. Scand J Med Sci Sports. 2009;20:725–730.
9. Brindle TJ, Mattacola C, McCrory J. Electromyographic changes in the gluteus medius during stair ascent and descent in subjects with anterior knee pain. Knee Surg Sports Traumatol Arthrosc. 2003;11:244–251. [PubMed: 12695878]
10. Brossmann J, Muhle C, Schroder C, et al. Patellar tracking patterns during active and passive knee extension: evaluation with motion-triggered cine MR imaging. Radiology. 1993;187:205–212. [PubMed: 8451415]
11. Callaghan MJ, Oldham JA. Quadriceps atrophy: to what extent does it exist in patellofemoral pain syndrome? Br J Sports Med. 2004;38:295–299. [PubMed: 15155431]
12. Callaghan MJ, Selfe J, McHenry A, Oldham JA. Effects of patellar taping on knee joint proprioception in patients with patellofemoral pain syndrome. Man Ther. 2008;13:192–199. [PubMed: 17296323]
13. Cowan SM, Hodges PW, Bennell KL, Crossley KM. Altered vastii recruitment when people with patellofemoral pain syndrome complete a postural task. Arch Phys Med Rehabil. 2002;83:989–995. [PubMed: 12098160]
14. Crossley K, Bennell K, Green S, Cowan S, McConnell J. Physical therapy for patellofemoral pain: a randomized, double-blinded, placebo-controlled trial. Am J Sports Med. 2002;30:857–865. [PubMed: 12435653]
15. Derasari A, Brindle TJ, Alter KE, Sheehan FT. McConnell taping shifts the patella inferiorly in patients with patellofemoral pain: a dynamic magnetic resonance imaging study. Phys Ther. 2010;90:411–419. [PubMed: 20110340]
16. Draper CE, Besier TF, Gold GE, et al. Is cartilage thickness different in young subjects with and without patellofemoral pain? Osteoarthritis Cartilage. 2006;14:931–937. [PubMed: 16647278]
17. Draper CE, Fredericson M, Gold GE, et al. Patients with patellofemoral pain exhibit elevated bone metabolic activity at the patellofemoral joint. J Orthop Res. 2011;29:312–317. [PubMed: 20949442]

18. Dye SF. The pathophysiology of patellofemoral pain: a tissue homeostasis perspective. Clin Orthop Relat Res. 2005:100–110.
19. Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. Am J Sports Med. 1998;26:773–777. [PubMed: 9850777]
20. Eckstein F, Reiser M, Englmeier KH, Putz R. In vivo morphometry and functional analysis of human articular cartilage with quantitative magnetic resonance imaging--from image to data, from data to theory. Anat Embryol (Berl). 2001;203:147–173. [PubMed: 11303902]
21. Farrokhi S, Keyak JH, Powers CM. Individuals with patellofemoral pain exhibit greater patellofemoral joint stress: a finite element analysis study. Osteoarthritis Cartilage. 2011;19:287–294. [PubMed: 21172445]
22. Fellows RA, Hill NA, Gill HS, et al. Magnetic resonance imaging for in vivo assessment of three-dimensional patellar tracking. J Biomech. 2005;38:1643–1652. [PubMed: 15958222]
23. Fulkerson JP. The etiology of patellofemoral pain in young, active patients: a prospective study. Clin Orthop Relat Res. 1983;179:129–133.
24. Fulkerson JP, Tennant R, Jaivin JS, Grunnet M. Histologic evidence of retinacular nerve injury associated with patellofemoral malalignment. Clin Orthop Relat Res. 1985;197:196–205.
25. Harbaugh CM, Wilson NA, Sheehan FT. Correlating femoral shape with patellar kinematics in patients with patellofemoral pain. J Orthop Res. 2010;28:865–872. [PubMed: 20108348]
26. Hungerford DS, Barry M. Biomechanics of the patellofemoral joint. Clin Orthop Relat Res. 1979;144:9–15.
27. Iwamoto J, Takeda T, Sato Y, Matsumoto H. Retrospective case evaluation of gender differences in sports injuries in a Japanese sports medicine clinic. Gend Med. 2008;5:405–414. [PubMed: 19108813]
28. Jan MH, Lin DH, Lin CH, Lin YF, Cheng CK. The effects of quadriceps contraction on different patellofemoral alignment subtypes: an axial computed tomography study. J Orthop Sports Phys Ther. 2009;39:264–269. [PubMed: 19346623]
29. Jerosch J, Prymka M. Knee joint proprioception in patients with posttraumatic recurrent patella dislocation. Knee Surg Sports Traumatol Arthrosc. 1996;4:14–18. [PubMed: 8819058]
30. Kaya D, Citaker S, Kerimoglu U, et al. Women with patellofemoral pain syndrome have quadriceps femoris volume and strength deficiency. Knee Surg Sports Traumatol Arthrosc. 2011;19:242–247. [PubMed: 20953760]
31. LLopis E, Padron M. Anterior knee pain. Eur J Radiol. 2007;62:27–43. [PubMed: 17350782]
32. MacIntyre NJ, Hill NA, Fellows RA, Ellis RE, Wilson DR. Patellofemoral joint kinematics in individuals with and without patellofemoral pain syndrome. J Bone Joint Surg Am. 2006;88:2596–2605. [PubMed: 17142409]
33. Mason JJ, Leszko F, Johnson T, Komistek RD. Patellofemoral joint forces. J Biomech. 2008;41:2337–2348. [PubMed: 18644310]
34. Matthews LS, Sonstegard DA, Henke JA. Load bearing characteristics of the patello-femoral joint. Acta Orthop Scand. 1977;48:511–516. [PubMed: 596148]
35. McWalter EJ, Hunter DJ, Wilson DR. The effect of load magnitude on three-dimensional patellar kinematics in vivo. J Biomech. 2010;43:1890–1897. [PubMed: 20413124]
36. Monk AP, Doll HA, Gibbons CLMH, et al. The patho-anatomy of patellofemoral subluxation. Journal of Bone and Joint Surgery-British Volume. 2011;93B:1341–1347.
37. Naslund JE, Odenbring S, Naslund UB, Lundeberg T. Diffusely increased bone scintigraphic uptake in patellofemoral pain syndrome. Br J Sports Med. 2005;39:162–165. [PubMed: 15728696]
38. Panagiotopoulos E, Strzelczyk P, Herrmann M, Scuderi G. Cadaveric study on static medial patellar stabilizers: the dynamizing role of the vastus medialis obliquus on medial patellofemoral ligament. Knee Surg Sports Traumatol Arthrosc. 2006;14:7–12. [PubMed: 16001289]
39. Pattyn E, Verdonk P, Steyaert A, et al. Vastus medialis obliquus atrophy: does it exist in patellofemoral pain syndrome? Am J Sports Med. 2011;39:1450–1455. [PubMed: 21487120]

40. Piva SR, Fitzgerald GK, Wisniewski S, Delitto A. Predictors of pain and function outcome after rehabilitation in patients with patellofemoral pain syndrome. J Rehabil Med. 2009;41:604–612. [PubMed: 19565153]
41. Powers CM, Landel R, Perry J. Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain. Phys Ther. 1996;76:946–955; discussion 956–967. [PubMed: 8790273]
42. Powers CM, Ward SR, Fredericson M, Guillet M, Shellock FG. Patellofemoral kinematics during weight-bearing and non-weight-bearing knee extension in persons with lateral subluxation of the patella: a preliminary study. J Orthop Sports Phys Ther. 2003;33:677–685. [PubMed: 14669963]
43. Reilly DT, Martens M. Experimental analysis of the quadriceps muscle force and patello-femoral joint reaction force for various activities. Acta Orthop Scand. 1972;43:126–137. [PubMed: 5079747]
44. Sanchis-Alfonso V, Rosello-Sastre E, Monteagudo-Castro C, Esquerdo J. Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A preliminary study. Am J Sports Med. 1998;26:703–709. [PubMed: 9784819]
45. Sanchis-Alfonso V, Rosello-Sastre E, Revert F. Neural growth factor expression in the lateral retinaculum in painful patellofemoral malalignment. Acta Orthop Scand. 2001;72:146–149. [PubMed: 11372945]
46. Sanchis-Alfonso V, Rosello-Sastre E, Revert F, Garcia A. Histologic retinacular changes associated with ischemia in painful patellofemoral malalignment. Orthopedics. 2005;28:593–599. [PubMed: 16138473]
47. Schutzer SF, Ramsby GR, Fulkerson JP. Computed tomographic classification of patellofemoral pain patients. Orthop Clin North Am. 1986;17:235–248. [PubMed: 3714207]
48. Seisler AR, Sheehan FT. Normative three-dimensional patellofemoral and tibiofemoral kinematics: a dynamic, in vivo study. IEEE Trans Biomed Eng. 2007;54:1333–1341. [PubMed: 17605365]
49. Selfe J, Harper L, Pedersen I, Breen-Turner J, Waring J, Stevens D. Cold legs: a potential indicator of negative outcome in the rehabilitation of patients with patellofemoral pain syndrome. Knee. 2003;10:139–143. [PubMed: 12787996]
50. Selfe J, Richards J, Thewlis D. Common movement tasks in Clinical Assessment. In: Richards J, ed. Biomechanics in Clinic and Research: Elsevier Science; 2008.
51. Selfe J, Thewlis D, Hill S, Whitaker J, Sutton C, Richards J. A clinical study of the biomechanics of step descent using different treatment modalities for patellofemoral pain. Gait Posture. 2011;34:92–96. [PubMed: 21570291]
52. Sheehan FT, Bhushan BS, Behnam AJ, Alter KE. Alterations in in vivo knee joint kinematics following a femoral nerve branch block of the vastus medialis: Implications for patellofemoral pain syndrome. Clin Biomech. In press. doi:10.1016/j.clinbiomech.2011.12.012
53. Sheehan FT, Derasari A, Fine KM, Brindle TJ, Alter KE. Q-angle and J-sign: indicative of maltracking subgroups in patellofemoral pain. Clin Orthop Relat Res. 2009;468:266–275. [PubMed: 19430854]
54. Shibanuma N, Sheehan FT, Stanhope SJ. Limb positioning is critical for defining patellofemoral alignment and femoral shape. Clin Orthop Relat Res. 2005;434:198–206.
55. Souza RB, Draper CE, Fredericson M, Powers CM. Femur rotation and patellofemoral joint kinematics: A weight-bearing MRI analysis. J Orthop Sports Phys Ther. 2010;40:277–285. [PubMed: 20436239]
56. Taunton JE, Ryan MB, Clement DB, McKenzie DC, Lloyd-Smith DR, Zumbo BD. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med. 2002;36:95–101. [PubMed: 11916889]
57. Thijs Y, Vingerhoets G, Pattyn E, Rombaut L, Witvrouw E. Does bracing influence brain activity during knee movement: an fMRI study. Knee Surg Sports Traumatol Arthrosc. 2010;18:1145–1149. [PubMed: 20033675]
58. Utting MR, Davies G, Newman JH. Is anterior knee pain a predisposing factor to patellofemoral osteoarthritis? Knee. 2005;12:362–365. [PubMed: 16146626]

59. Voight ML, Wieder DL. Comparative reflex response times of vastus medialis obliquus and vastus lateralis in normal subjects and subjects with extensor mechanism dysfunction. An electromyographic study. Am J Sports Med. 1991;19:131–137. [PubMed: 2039064]
60. Warden SJ, Hinman RS, Watson MA Jr, Avin KG, Bialocerkowski AE, Crossley KM. Patellar taping and bracing for the treatment of chronic knee pain: a systematic review and meta-analysis. Arthritis Rheum. 2008;59:73–83. [PubMed: 18163413]
61. Wilson NA, Press JM, Koh JL, Hendrix RW, Zhang LQ. In vivo noninvasive evaluation of abnormal patellar tracking during squatting in patients with patellofemoral pain. J Bone Joint Surg Am. 2009;91:558–566. [PubMed: 19255215]
62. Wilson NA, Sheehan FT. Dynamic in vivo 3-dimensional moment arms of the individual quadriceps components. J Biomech. 2009;42:1891–1897. [PubMed: 19515373]
63. Witvrouw E, Sneyers C, Lysens R, Victor J, Bellemans J. Reflex response times of vastus medialis oblique and vastus lateralis in normal subjects and in subjects with patellofemoral pain syndrome. J Orthop Sports Phys Ther. 1996;24:160–165. [PubMed: 8866275]
64. Wunschel M, Leichtle U, Obloh C, Wulker N, Muller O. The effect of different quadriceps loading patterns on tibiofemoral joint kinematics and patellofemoral contact pressure during simulated partial weight-bearing knee flexion. Knee Surg Sports Traumatol Arthrosc. 2011;19:1099–1106. [PubMed: 21222108]
65. Yamaguchi GT, Zajac FE. A planar model of the knee joint to characterize the knee extensor mechanism. Journal of Biomechanics. 1989;22:1–10. [PubMed: 2914967]

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.[2]

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.[2] However, it is unclear how these kinematic variations relate to patellofemoral joint loading and subsequent pathology, and whether they represent aetiological 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.[4] 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.[5] 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 re-supinate 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 roll over pattern of plantar pressure was a risk factor for PFP development in predominantly male military recruit,[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).[1,6,14,15,17,20,23,24] However, the mechanism(s) by which orthotics reduce PFP remain(s) unclear.

9. Foot orthoses have been shown to improve functional performance immediately[7] and in the medium term (12 weeks)[6] in persons with PFP, and appear to have similar immediate effects in patellofemoral joint osteoarthritis.[13]

10. Those with PFP tend to report soft foot orthoses to be 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] and lower baseline pain severity,[8,26] as well as less ankle dorsiflexion and wearing of less supportive shoes.[8] 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,[8] 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 non-rearfoot 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 1 of the greatest challenges. Inconsistent definitions of segments within multi-segment 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 may 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 on-going 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 multi-segment kinematic foot models that consider the forefoot and midfoot, and methods of measuring patellar motion. Technologies such as standing MRI would allow

more accurate visualisation 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 provide evidence for whether foot interventions (eg, foot orthoses, intrinsic exercises) are worthwhile to prevent PFP in those at risk. More studies utilising 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) need to be further evaluated, ideally in randomized clinical trials with a natural history control.

References

1. Amell TK, Stothart JP, Kumar S. The effectiveness of functional foot orthoses as a treatment for patellofemoral stress syndrome: The client's perspective. Physiotherapy Canada. 2000;162:153–157.
2. Barton CJ et al. Kinematic gait characteristics associated with patellofemoral pain syndrome: a systematic review. Gait & Posture. 2009;30:405–416. [PubMed: 19651515]
3. Barton CJ et al. Foot and ankle characteristics in patellofemoral pain syndrome: a case control and reliability study. J Orthop Sports Phys Ther. 2010;40:286–296. [PubMed: 20436240]
4. Barton CJ et al. The relationship between rearfoot, tibial and femoral kinematics in individuals with patellofemoral pain syndrome. Proceedings of the 2nd Patellofemoral Pain Research Retreat: Proximal, Local, and Distal Factors. Ghent, Belgium, 2011.
5. Barton CJ et al. Relationships between the Foot Posture Index and foot kinematics during gait in individuals with and without patellofemoral pain syndrome. Journal of Foot and Ankle Res. 2011;4:10. [PubMed: 21401957]

6. Barton CJ, Menz HB, Crossley KM. Effects of prefabricated foot orthoses on pain and function in individuals with patellofemoral pain syndrome: a cohort study. Phys Ther in Sport. 2011;12:70–75. [PubMed: 21496768]
7. Barton CJ, Menz HB, Crossley KM. The immediate effects of foot orthoses on functional performance in individuals with patellofemoral pain syndrome. Br J Sports Med. 2011;45:193–197. [PubMed: 20647297]
8. Barton CJ, Menz HB, Crossley KM. Clinical predictors of foot orthoses efficacy in individuals with patellofemoral pain. Med Sci Sports Exerc. 2011;43:1603–1610. [PubMed: 21311359]
9. Boling MC et al. A preliminary analysis of gender specific risk factors for patellofemoral pain syndrome. Proceedings of the 2nd Patellofemoral Pain Research Retreat: Proximal, Local and Distal Factors. Ghent, Belgium, 2011
10. Boling MC et al. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: the Joint Undertaking to Monitor and Prevent ACL Injury (JUMP-ACL) cohort. Am J Sports Med. 2009;37:2108–2116. [PubMed: 19797162]
11. Cheung R, Davis IS. Landing pattern modification to improve patellofemoral pain in runners: a case series. J Orthop Sports Phys Ther. 2011;41:914–919. [PubMed: 22031595]
12. Collins NJ et al. Efficacy of non-surgical interventions for anterior knee pain: systematic review and meta-analysis of randomised trials. Sports Med. 2012;42:31–49. [PubMed: 22149696]
13. Collins NJ et al. Shoe inserts produce immediate pain relief in individuals with patellofemoral joint osteoarthritis. Proceedings of the 2nd Patellofemoral Pain Research Retreat: Proximal, Local and Distal Factors. Ghent, Belgium, 2011
14. Collins NJ et al. Foot orthoses and physiotherapy in the treatment of patellofemoral pain syndrome: randomised clinical trial. BMJ. 2008;337:a1735. [PubMed: 18952682]
15. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. Phys Ther. 1993;73:62–68. [PubMed: 8421719]
16. Finestone A et al. A prospective study of the effect of foot orthoses composition and fabrication on comfort and the incidence of overuse injuries. Foot Ankle Int. 2004;25:462–466. [PubMed: 15319103]
17. Johnston LB, Gross MT. Effects of foot orthoses on quality of life for individuals with patellofemoral pain syndrome. J Orthop Sports Phys Ther. 2004;34:440–448. [PubMed: 15373007]
18. Leitch J et al. Lower-limb and foot kinematics in distance runners with patellofemoral pain syndrome. Proceedings of the 2nd Patellofemoral Pain Research Retreat: Proximal, Local and Distal Factors. Ghent, Belgium, 2011
19. McPoil TG et al. Variations in foot posture and mobility between individuals with patellofemoral pain and those in a control group. Journal of the Am Pod Med Assoc. 2011;101:289–296.
20. Mills K et al. A randomised control trial of short term efficacy of in-shoe foot orthoses compared with a wait and see policy for anterior knee pain and the role of foot mobility. Br J Sports Med. 2012;46:247–252. [PubMed: 21930514]
21. Mills K, Blanch P, Vicenzino B. Comfort and midfoot mobility rather than orthosis hardness or contouring influence their immediate effects on lower limb function in patients with anterior knee pain. Clin Biomech. 2012;27:202–208.
22. Nigg BM, Nurse MA, Stefanyshyn DJ. Shoe inserts and orthotics for sport and physical activities. Med Sci Sports Exerc. 1999;31:421–428. [PubMed: 10188747]
23. Pitman D, Jack D. A clinical investigation to determine the effectiveness of biomechanical foot orthoses as initial treatment for patellofemoral pain syndrome. J Prosthet and Orthot. 2000;12:110–116.
24. Sutlive TG et al. Identification of individuals with patellofemoral pain whose symptoms improved after a combined program of foot orthosis use and modified activity: a preliminary investigation. Phys Ther. 2004;84:49–61. [PubMed: 14992676]
25. Thijs Y et al. A prospective study on gait-related intrinsic risk factors for patellofemoral pain. Clin J Sport Med. 2007;17:437–445. [PubMed: 17993785]

26. Vicenzino B et al. A clinical prediction rule for identifying patients with patellofemoral pain who are likely to benefit from foot orthoses: a preliminary determination. Br J Sports Med. 2010;44:862–866. [PubMed: 18819958]

27. Witvrouw E et al. Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. Am J Sports Med. 2000;28:480–489. [PubMed: 10921638]

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 inter-relationships among hip muscle performance (eg, strength, endurance, and neuromuscular activity), kinematics, and kinetics (collectively referred 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 or not 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.

References

1. Aminaka N, Pietrosimone BG, Armstrong CW, Meszaros A, Gribble PA. Patellofemoral pain syndrome alters neuromuscular control and kinetics during stair ambulation. J Electromyogr Kinesiol. 2011;21:645–651. [PubMed: 21524921]
2. Bazett-Jones DM, Cobb SC, Joshi MN, Cashin SE, Earl JE. Normalizing hip muscle strength: establishing body-size-independent measurements. Arch Phys Med Rehabil. 2011;92:76–82. [PubMed: 21187208]
3. Bolgla LA, Boling MC. An update for the conservative management of patellofemoral pain syndrome: a systematic review of the literature from 2000 to 2010. Int J Sports Phys Ther. 2011;6:112–125. [PubMed: 21713229]
4. Bolgla LA, Malone TR, Umberger BR, Uhl TL. Comparison of hip and knee strength and neuromuscular activity in subjects with and without patellofemoral pain syndrome. Int J Sports Phys Ther. 2011;6:285–296. [PubMed: 22163090]
5. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: the Joint Undertaking to Monitor and Prevent ACL Injury (JUMP-ACL) cohort. Am J Sports Med. 2009;37:2108–2116. [PubMed: 19797162]

6. Brindle TJ, Mattacola CG, McCrory JL. Electromyographic changes in the gluteus medius during stair ascent and descent in subjects with anterior knee pain. Knee Surg Sports Traumatol Arthrosc. 2003;11:244–251. [PubMed: 12695878]
7. Cowan SM, Crossley KM, Bennell KL. Altered hip and trunk muscle function in individuals with patellofemoral pain syndrome. Br J Sports Med. 2009;43:584–588. [PubMed: 18838402]
8. Dierks TA, Manal KT, Hamill J, Davis IS. Proximal and distal influences on hip and knee kinematics in runners with patellofemoral pain during a prolonged run. J Orthop Sports Phys Ther. 2008;38:448–456. [PubMed: 18678957]
9. Earl JE, Hoch AZ. A proximal strengthening program improves pain, function, and biomechanics in women with patellofemoral pain syndrome. Am J Sports Med. 2011;39:154–163. [PubMed: 20929936]
10. Ferber R, Kendall KD, Farr L. Changes in knee biomechanics after a hip-abductor strengthening protocol for runners with patellofemoral pain syndrome. J Athl Train. 2011;46:142–149. [PubMed: 21391799]
11. Finnoff JT, Hall MM, Kyle K, Krause DA, Lai J, Smith J. Hip strength and knee pain in high school runners: a prospective study. PMR. 2011;3:792–801.
12. Lee TQ, Morris G, Csintalan RP. The influence of tibial and femoral rotation on patellofemoral contact area and pressure. J Orthop Sports Phys Ther. 2003;33:686–693. [PubMed: 14669964]
13. Noehren B, Davis I. The effect of real-time gait retraining on hip kinematics, pain and function in subjects with patellofemoral pain syndrome. Br J Sports Med. 2011;45:691–696. [PubMed: 20584755]
14. Powers CM, Ward SR, Fredericson M, Guillet M, Shellock FG. Patellofemoral kinematics during weight-bearing and non-weight-bearing knee extension in persons with lateral subluxation of the patella: A preliminary study. J Orthop Sports Phys Ther. 2003;33:677–685. [PubMed: 14669963]
15. Prins MR, van der Wurff P. Females with patellofemoral pain syndrome have weak hip muscles. a systematic review. Aust J Physiother. 2009;55:9–15. [PubMed: 19226237]
16. Salsich GB, Perman WH. Patellofemoral joint contact area is influenced by tibiofemoral rotation alignment in individuals who have patellofemoral pain. J Orthop Sports Phys Ther. 2007;37:521–528. [PubMed: 17939611]
17. Souza RB, Draper CE, Fredericson M, Powers CM. Femur rotation and patellofemoral joint kinematics: a weight-bearing magnetic resonance imaging analysis. J Orthop Sports Phys Ther. 2010;40:277–285. [PubMed: 20436239]
18. Souza RB, Powers CM. Differences in hip kinematics, muscle strength, and muscle activation between subjects with and without patellofemoral pain. J Orthop Sports Phys Ther. 2009;39:12–19. [PubMed: 19131677]
19. Souza RB, Powers CM. Predictors of hip internal rotation during running: an evaluation of hip strength and femoral structure in women with and without patellofemoral pain. Am J Sports Med. 2009;37:579–587. [PubMed: 19098153]
20. Thijs Y, Pattyn E, Van Tiggelen D, Rombaut L, Witvrouw E. Is hip muscle weakness a predisposing factor for patellofemoral pain in female novice runners? A prospective study. Am J Sports Med. 2011;39:1877–1882. [PubMed: 21632979]
21. Willson JD, Binder-Macleod S, Davis IS. Lower extremity jumping mechanics of female athletes with and without patellofemoral pain before and after exertion. Am J Sports Med. 2008;36:1587–1596. [PubMed: 18448577]
22. Willson JD, Davis IS. Lower extremity mechanics of females with and without patellofemoral pain across activities with progressively greater task demands. Clin Biomech. 2008;23:203–211.
23. Willson JD, Davis I. Lower extremity strength and mechanics during jumping in women with patellofemoral pain. J Sport Rehabil. 2009;18:75–89.
24. Willson JD, Kernozek TW, Arndt RL, Reznichek DA, Straker JS. Gluteal muscle activation during running in females with and without patellofemoral pain syndrome. Clin Biomech. 2011;26:735–740.
25. Willy RW, Davis IS. The effect of a hip-strengthening program on mechanics during running and during a single-leg squat. J Orthop Sports Phys Ther. 2011;41:625–632. [PubMed: 21765220]

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 non-randomized 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 of 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 sub-grouping of subjects so that the intervention is more targeted and may increase the power of the trial.
3. These sub-groups might include foot posture, patellar alignment, muscle weakness and flexibility as well as patient fear, psychology, and the menstrual cycle that 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 sub-groups exist and if they can be identified in clinical practice.

5. Finally, there are effective non-surgical 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 post intervention. Therefore, studies with at least 1 year follow up may give a different impression of treatment efficacy.

References

1. Callaghan Selfe J. Patellar taping for patellofemoral pain syndrome in adults. The Cochrane Library, Issue 4, 2012.
2. Collins Crossley K, Beller E, Darnell R, McPoil T, Vicenzino B. Foot orthoses and physiotherapy in the treatment of patellofemoral pain syndrome: randomised clinical trial. Br J Sports Med. 2008;337:a1735.
3. Earl Hoch AZ. A Proximal Strengthening Program Improves Pain, Function, and Biomechanics in Women With Patellofemoral Pain Syndrome. Am J Sport Med. 2011;39:154–163.
4. Fukuda Rossetto FM, Magalhaes E Bryk FF, Lucareli PR, de Almeida Aparecida CN. Short-term effects of hip abductors and lateral rotators strengthening in females with patellofemoral pain syndrome: a randomized controlled clinical trial. J Orthop Sports Phys Ther. 2010;40:736–742. [PubMed: 21041965]
5. Hunter Harvey W, Gross KD, Felson D, McCree P, Li L, Hirko K, Zhang B, Bennell K. A randomized trial of patellofemoral bracing for treatment of patellofemoral osteoarthritis. Osteoarthritis and Cartilage. 2011;19:792–800. [PubMed: 21232620]
6. Khayambashi K, Mohammadkhani Z, Ghaznavi K, Lyle MA, Powers CM. The effects of isolated hip abductor and external rotator muscle strengthening on pain, health status, and hip strength in females with patellofemoral pain. J Orthop Sports Phys Ther. 2012;42:22–29. [PubMed: 22027216]
7. Powers CM, Doubleday KL, Escudero C. Influence of patellofemoral bracing on pain, knee extensor torque, and gait function in females with patellofemoral pain. Physiother Theory and Pract. 2008;24:143–150
8. van Linschoten R, van Middelkoop M, Berger MY, Heintjes EM, Verhaar JAN, Willemsen SP, Koes BW, Bierma-Zeinstra SM. Supervised exercise therapy versus usual care for patellofemoral pain syndrome: an open label randomised controlled trial. BMJ. 2009;339:4074.
9. Van Tiggelen D, Witvrouw E, Roget P, Cambier D, Danneels L, Verdonk R. Effect of bracing on the prevention of anterior knee pain-a prospective randomized study. Knee Surg Sports Traumatol Arthrosc. 2004;12:434–439. [PubMed: 15064923]