Evidence-based framework for a pathomechanical model of patellofemoral pain: 2017 patellofemoral pain consensus statement from the 4th International Patellofemoral Pain Research Retreat, Manchester, UK: part 3

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INTRODUCTION
The aetiology of patellofemoral pain (PFP) is a complex interplay among various anatomical, biomechanical, psychological, social and behavioural influences. Numerous factors associated with PFP have been reported in the literature, but the interaction between these proposed risk factors and the clinical entity of PFP remains unclear (figure 1).

The goal of this consensus document is to place known associated factors within the context of a pathomechanical model of PFP. An underlying assumption of the proposed pathomechanical model is that PFP is associated with abnormal loading of the patellofemoral joint (elevated joint stress). In this model, abnormal loading could affect the various patellofemoral structures that can contribute to nociception (ie, subchondral bone, infrapatellar fat pad, retinaculum and ligamentous structures); however, the specific tissue sources related to PFP are not known.

The experience of PFP is not just nociception. Persons with persistent PFP exhibit abnormal nociceptive processing (ie, widespread mechanical hyperalgesia, impaired pain modulation),2–5 altered somatosensory processing (implying neuropathic pain),6 impaired sensorimotor function (ie, proprioception and balance)7–10 and certain psychological factors (ie, catastrophising and kinesiophobia).11 The amount and quality of research in the non-‘patho-mechanical’ pathways to PFP are evolving, and will be included in future consensus statements emanating from the International Patellofemoral Pain Research Retreats.

Figure 1 Schematic overview of potential pathways to elevated patellofemoral joint (PFJ) stress, a proposed contributor to patellofemoral pain.
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At the 4th International Patellofemoral Pain Research Retreat, Dr Christopher Powers presented a draft framework of the pathomechanical model, which was based on prior consensus statements from the three previous Patellofemoral Pain Research Retreats. At the meeting, all attendees (clinician-researchers and research scientists) participated in a comprehensive discussion of the draft model, and agreed on the overall framework (Figure 1). Following the retreat, the authors conducted a thorough review of pertinent literature related to the specific pathways specified within the pathomechanical model.

The current document does not include risk factors for patellofemoral osteoarthritis. It also should be noted that the associated factors and statements in the current document pertain only to PFP, and not patellar instability. Although it is likely that many of the factors associate with PFP also play a role in patellar instability (particularly anatomical and biomechanical factors), the research that has formed the basis of this document primarily has focused on persons with pain.

The current consensus document should be viewed as a ‘living document’ and will be reviewed and edited as necessary at future retreats based on new research findings. In the interim, it is anticipated that this document will provide the basis from which to frame research questions related to PFP and provide clinicians with a contemporary synthesis of current evidence.


Statement 1. Persons with PFP exhibit elevated patellofemoral joint, cartilage and bone stress during functional tasks; however, this finding is not consistent across studies. Currently, no prospective studies have linked elevated mechanical stress to the development of PFP.

Persons with PFP exhibit greater patellofemoral joint (PFJ) stress during walking, as well as patellar cartilage stress and bone strain during squatting (45° knee flexion). However, studies that evaluated PFJ and patellar cartilage stress during tasks with relatively higher knee flexion angles (ie, stair ambulation, running and squatting to 60°) have not reported differences between persons with and without PFP. Similarly, braced-induced reductions in PFJ stress during walking resulted in an immediate decrease in PFP symptoms, but the same stress–pain relationship was not reported during stair ambulation.

The exact mechanism by which elevated joint loading may contribute to PFP is not clear. Repetitive overloading of the PFJ may increase patellar subchondral bone metabolic activity and/or elevate patellar bone water content. Elevated water content could increase the intraosseous pressure within the patella, thus stimulating pressure-sensitive mechanical nociceptors. In support of this premise, it has been reported that runners with PFP exhibit elevated bone water content, and that running-induced PFP fluctuates with changes in patellar water content.

Statement 1.1. Elevated patellofemoral joint stress can result from diminished contact area in persons with PFP, but this is variable and likely dependent on knee flexion angle.
Elevated patellofemoral joint stress during walking in persons with PFP is the result of diminished contact area. However, diminished contact area in this population appears to be dependent on the knee flexion angle evaluated. For example, some studies found a difference in available contact area during relatively higher knee flexion angles between persons with and without PFP, while others did not. The premise that lower contact area in persons with PFP may be knee flexion-dependent is supported by research reporting diminished contact area in this population at 20°, but not 40° of knee flexion. Contact area differences between persons with and without PFP are minimised once the patella moves deeper within the femoral trochlea.

Statement 1.1a. Patellar malalignment and/or maltracking in persons with PFP can contribute to diminished contact area, but only in a subset of persons with PFP.
Patellar malalignment and/or maltracking in persons with PFP can result in diminished contact area. A systematic review of 40 studies reported that lateral patellar displacement in persons with PFP is more pronounced in knee extension, regardless of weightbearing.
Although persons with PFP exhibit patellar malalignment, and/or altered patellar kinematics, this finding is not consistent across all studies. This suggests that altered patellar alignment or tracking may not be a universal finding in this population. In addition, conflicting findings may be attributed to differences in the methods used to quantify patellar tracking (ie, dynamic MRI, CT and others), the conditions under which patellar alignment/tracking was quantified (static vs dynamic; weightbearing vs non-weightbearing), and the specific measurements and frames of reference used (two-dimensional (2D) vs three-dimensional).
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Statement 1.1a.1. Impaired quadriceps function is a common finding in persons with PFP, but its role in patellar malalignment and/or maltracking is unclear.

Generalised quadriceps weakness and/or atrophy is evident in idiopathic PFP6–11; however, isolated atrophy of the vastus medialis (VM) has not been consistently reported.1,2 Similarly, altered magnitude and/or timing of VM activation (determined using electromyography) relative to the vastus lateralis (VL) is not consistently implicated in PFP6–11 Nonetheless, quadriceps weakness,1,2 as well as delayed onset of VM relative to VL,13,14 is associated with the development of PFP.

Cadaveric studies demonstrate that simulated muscle force imbalance of the VM relative to the VL results in lateral patellar displacement and tilt,15,16 and increased contact pressures on the lateral patellar facet.17 However, diminished force generation of the VM following a motor branch block in vivo explained some, but not all, of the expected changes in lateral patellar displacement and tilt.18 Reduced activation and/or delayed onset timing of the VM relative to the VL is associated with increased lateral patellar tilt and displacement.19,20 The inverse association between lateral patellar displacement and VM activation2 suggests that increased motor unit activity may be a response to meet the increased demand of providing medial patellar stability. While activation of the synergistic medial and lateral components of the vasti may be altered in a subgroup/proportion of persons with PFP, the implication of this altered activation in relationship to patellar kinematics is unclear.1


Statement 1.1a.3. There is evidence that persons with PFP exhibit impairments related to soft tissue restraints, and that these impairments may contribute to patellar malalignment and/or maltracking.

Persons with PFP have a tighter1 and thicker2 iliotibial band compared with pain-free individuals. Based on in vivo and
Cadaveric studies, iliotibial band tension has a substantial effect on patellar alignment and lateral patellar translation. 

Currently, it is not known whether iliotibial band tightness and thickening is an adaptation to or a cause of lateral tilt/translation of the patella.

Ligamentous injury or laxity (particularly the medial patellofemoral ligament) can contribute to altered patellar tracking. Although ligamentous laxity has been proposed to be a risk factor for patellar instability, increased passive mobility of the patella has not been reported in persons with PFP. However, generalised ligamentous laxity is associated with PFP development.


Statement 1.1a.4. Patellar alignment and patellofemoral joint kinematics are influenced by the bony geometry of the distal femur, patellar height, but not the static quadriiceps angle (Q-angle).

The sulcus angle and inclination of the lateral anterior femoral condyle are important determinants of patellar malalignment and maltracking. However, the inclination of the lateral anterior femoral condyle is a better predictor of mediolateral tracking of the patella than the sulcus angle.

The height of the patella within the trochlear groove is an important contributor to malalignment and maltracking of the patella. For example, lateral patellar tilt and displacement is more prevalent in persons with patellar alta than persons with normal patellar height. In addition, patellar height is the best structural predictor of lateral patellar tilt at 0° of knee flexion. Persons with patellar alta exhibit lower contact area for a given knee flexion angle and higher patellofemoral stress during fast walking.

A systematic review of prospective studies indicates the Q-angle as a static measure is not a risk factor for PFP. Based on MRI measurements, the static Q-angle does not represent the quadriiceps line-of-action, and this measurement should not be used to infer patellofemoral kinematics.


Statement 1.2. Patellofemoral (PF) joint reaction forces in persons with PFP differ from those in pain-free individuals.

Persons with PFP exhibit lower peak resultant PF joint reaction forces compared with healthy controls during walking, running and stair ambulation. However, persons with PFP have a higher lateral component of the PF joint reaction force than do pain-free individuals. The lower resultant PF joint reaction forces in persons with PFP may represent a compensatory strategy to minimise patellofemoral joint loading during functional tasks.


Statement 1.2a. Persons with PFP exhibit differences in tibiofemoral kinematics in all three planes of motion compared with pain-free individuals, but not consistently.

Altered tibiofemoral joint kinematics in the sagittal, frontal and transverse planes can influence the magnitude and direction of the resultant PF joint reaction force vector. In the sagittal plane, greater knee flexion would be expected to increase the posterior (compression) component of the PF joint reaction force vector. However, persons with PFP exhibit lower knee flexion during walking, stair ambulation and running. Although reduced knee flexion in persons with PFP may represent a compensatory strategy to minimise patellofemoral joint loading during functional walking and stair ambulation.

tasks (statement 1.2), the finding of reduced knee flexion during ambulatory tasks is not consistent across all studies. Inter-
estingly, it has been reported that lower knee flexion during a jump-
landing task is a risk factor for the development of PFP. 

Increased frontal plane motion of the knee (ie, abduction or valgus) can increase the laterally directed component of the patellofemoral joint reaction force (PFJRF) vector. Although persons with PFP exhibit increased knee abduction during gait, and single tasks such as squatting, stepping and hop landing, higher knee abduction has not been reported during stair descent or running. Furthermore, 2D measures of knee valgus can predict the development of PFP. Knee abduction also is correlated with self-reported pain in men and women with PFP. 

Transverse plane tibiofemoral rotation also can affect the later-
ally directed component of the PFJRF vector, with the influence dependent on relative segmental motion. For example, internal rotation of the femur relative to the tibia can increase the laterally directed forces acting on the patella, while tibia internal rotation relative to the femur would result in a reduction of the laterally directed forces acting on the patella. While women with PFP exhibit higher degrees of knee external rotation during single leg squats, jumps and running, greater degrees of knee internal rotation have been reported in persons with PFP during stair descent. It should be noted that the finding of abnormal tibiofemoral rotation during stair descent is not consistent across studies.

Small degrees of simulated knee valgus (5°) do not affect patellofemoral contact mechanics; however, a 10° change in the frontal plane alignment of the extensor mechanism increases patellofemoral joint pressures by 45%. Interestingly, the elevated contact pressures observed in this study occurred without a change in contact area, suggesting that the finding of elevated patellofemoral stress was likely a function of increase in the laterally directed PFJR.5


Statement 1.2a.2. Persons with PFP exhibit altered hip kinematics; however, findings are not consistent among all studies.

Given that the hip joint shares a common segment with the tibiofemoral joint (ie, femur), abnormal hip kinematics could contribute to the altered tibiofemoral kinematics described above (statement 1.2a).1 The motions of hip adduction and knee abduction are significantly correlated, resulting in medial displacement of the knee.2 With respect to tibiofemoral rotation, internal rotation of the femur relative to the tibia (ie, hip internal rotation) could result in relative tibiofemoral external rotation and vice versa. Hip internal rotation was also correlated with knee abduction during single limb squats; however, the opposite was evident during running, where greater hip external rotation was associated with greater knee abduction angles.4

Persons with PFP exhibit excessive hip adduction during a wide range of functional tasks including running, stepping and landing from a jump. The results of two systematic reviews have concluded that there is a moderate association between hip adduction and PFP.3,6 Furthermore, excessive hip adduction predicts self-reported PF and function during a stepdown task.7 Additionally, a prospective study reported that hip adduction was significantly greater in runners who later went on to develop PFP.8

A systematic review also identified a moderate association between hip internal rotation during running and PFP.9 Furthermore, hip internal rotation can predict self-reported pain and function during a stepdown task,7 and the development of PFP in Naval Academy cadets.11 Interestingly, lower than normal hip internal rotation has been observed during gait in persons with PFP,12,13 which may represent a compensatory strategy to avoid pain.

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subsequently developed PFP. This discrepancy might reflect the imprecise relationship between hip strength and hip and knee kinematics.6–11 It is possible that hip weakness develops as a consequence of PFP.

Two bony abnormalities may influence altered hip kinematics in the context of PFP: (1) excessive femoral anteversion and (2) increased femoral neck inclination (i.e., coxa valga).7 Although excessive femoral anteversion can contribute to excessive hip rotation during gait,12 excessive femoral anteversion is not apparent in persons with PFP.13,14 In addition, coxa valga can contribute to diminished the lever arm for the gluteus medius.14 Although women with PFP exhibit greater degrees of coxa valga compared with healthy controls,2 the reported difference was quite small (4.4°) and likely of little clinical relevance.


Statement 1.2a.3. Excessive rotation of the tibia accompanies subtalar joint pronation, and may be a contributor to patellofemoral dysfunction. While a subset of persons with PFP may exhibit altered foot kinematics and plantar pressures, measures of dynamic foot function are not consistently associated with PFP.

Excessive subtalar joint pronation may result in greater tibia and femur rotation through coupling mechanisms.12–15 A systematic review of 24 case-control studies reported that persons with PFP exhibit tendencies towards excessive and delayed rearfoot eversion during walking and running.1 However, dynamic foot function did not emerge as a risk factor for the development of PFP.6,5 The discrepancy between the cross-sectional and prospective studies may relate to the quantification of pronation (i.e., mid-foot vs rearfoot motion), and the variable coupling actions of the mid-foot, rearfoot, tibia and femur among individuals.6 To date, only one study found a correlation between rearfoot eversion and lower extremity kinematics in persons with PFP (hip adduction)16; however, the reported relationship was moderate (r=0.48).

Altered plantar pressures may be found in persons with PFP; however, findings among studies are variable.6–11 A prospective study reported that persons who developed PFP had significantly greater lateral pressure at the rearfoot during walking, suggestive of a less pronated foot.12

Statement 1.2a.3a. Persons with PFP exhibit physical impairments at the foot and ankle that are consistent with excessive foot pronation, but these findings are not universal.

Persons with PFP exhibit rearfoot and forefoot varus, navicular drop, and calf tightness; however, this is not a universal finding across all studies. Similarly, static measures of foot posture (ie, foot arch height index, foot posture index) are not associated with PFP across all studies. While some authors report differences in foot posture in persons with PFP, others do not. Although there is some indication that foot posture may be associated with certain measures of dynamic foot function, there is limited evidence that a pronated foot posture is associated with PFP development. In contrast, navicular drop is associated with PFP development.

Foot and ankle mobility (ie, limited ankle dorsiflexion, increased mid-foot mobility) is associated with 2D measures of knee valgus and diminished quality of movement during a lateral stepdown task.


Statement 1.2b. Persons with PFP exhibit differences in tibiofemoral kinetics in all three planes of motion compared with pain-free individuals; however, there are inconsistencies among studies.

Altered tibiofemoral joint kinetics in the sagittal plane can influence the magnitude of the PFJRF. Consistent with the finding of lower PFJRFs in this population, persons with PFP exhibit lower knee extensor moments during walking, stair climbing and running. Other studies report no differences in knee extensor moments between persons with PFP and healthy controls during walking, stair climbing and running. Persons with PFP exhibit higher muscle co-contraction at the knee (quadriiceps-hamstrings), which may lower the net joint moments at the knee.

With respect to knee kinetics in the frontal plane, persons with PFP have elevated knee abductor moments during walking and stair ambulation, as well as higher knee abduction impulses during running. In the transverse plane, persons with PFP exhibit increased knee external rotator moments during loading response, when compared with age-matched and sex-matched controls.

Increased knee abduction moments during a drop jump task can predict PFP development in young female athletes. Furthermore, the knee abduction impulse can predict PFP in runners. The kinetic tendencies displayed by persons with PFP may be accompanied by abnormal kinematics described in statement 1.2a.

Statement 1.2b.1. Persons with PFP exhibit differences in ground reaction forces compared with pain-free individuals, but study results are inconsistent.

When compared with healthy controls, persons with PFP have lower ground reaction forces and loading rates during free and fast walking,\(^1\) higher than normal loading rates during stair ambulation,\(^2\) and no differences in vertical force loading rate during running.\(^3\) Although higher ground reaction and loading rates during stair ambulation were associated with pain and functional status,\(^4\) the influence of vertical force loading rate on patellofemoral joint loading has not been established.


Statement 1.2b.2. Persons with PFP exhibit differences in trunk kinematics compared with pain-free individuals, but findings are not consistent.

Sagittal plane trunk posture has the potential to influence the knee extensor moment and therefore the PFJRF. For example, running with a more upright trunk posture is associated with higher knee extensor moments, PFJRFs and higher PFJ stress when compared with running with a more flexed trunk.\(^1\) Running-induced fatigue can result in a compensatory forward trunk lean, which was hypothesised to minimise PFJ loading and pain.\(^2\) In contrast to running, however, persons with PFP do not exhibit altered sagittal plane trunk kinematics during stair descent.\(^3\)

Persons with PFP also exhibit altered trunk kinematics in the frontal plane. Specifically, persons with PFP exhibit an ipsilateral trunk lean during single limb tasks (ie, single leg squatting, jumping/landing).\(^4\) An ipsilateral trunk lean would shift the centre of mass of the body towards the stance limb, thereby increasing the potential for a knee abductor moment.\(^1\)


Statement 1.2b.2a. Impaired hip and trunk muscle performance may contribute to altered trunk kinematics in persons with PFP; however, there is inconsistency among studies.

Abnormal motions/postures of the trunk in the frontal and sagittal planes may be compensatory strategies related to diminished hip strength.\(^1\) Runners with hip extensor weakness tend to adopt a more upright trunk posture to reduce the demand on the hip extensors, while runners with greater hip extensor strength run with a more forward trunk.\(^2\) In addition, ipsilateral trunk lean in persons with PFP has been reported to be a compensation for stance limb hip abductor weakness.\(^3\)

Persons with PFP have been reported to exhibit reduced trunk strength\(^4\); however, this impairment has not been shown to be associated with faulty trunk kinematics. Nonetheless trunk strength has been shown to be correlated with knee abduction during weightbearing,\(^5\) as well as with self-reported function in persons with PFP.\(^6\)


Statement 1.2c. Persons with PFP exhibit tightness of the quadriceps and hamstring muscle groups. However, prospective studies linking muscle tightness to the development of PFP are inconsistent.

Shorter hamstring length was associated with greater PFJRFs and stress during squatting in healthy persons.\(^1\) Although persons with PFP have shorter hamstring muscles compared...
with asymptomatic controls,\textsuperscript{2,4} hamstring tightness was not a predictor of PFP development in a prospective study.\textsuperscript{3}

Tightness of the quadriceps muscles can increase PFJRFs. Several studies report that persons with PFP exhibit tightness of the quadriceps muscles compared with those without PFP,\textsuperscript{3,4,6,7} and a shortened quadriceps muscle has been shown to be a predictor of PFP development.\textsuperscript{7}


\textbf{Statement 1.3. Reduced patellar cartilage thickness can contribute to elevated patellofemoral joint loading.}

Reduced patellar cartilage thickness has been reported to be associated with higher patellar bone strain\textsuperscript{1} and diminished deformational behaviour of patellar cartilage.\textsuperscript{2} Reduced cartilage thickness also has been shown to result in elevated cartilage stress for a given load.\textsuperscript{3} As such, decreased patellar cartilage thickness may contribute to a vicious cycle of patellofemoral joint pathology (reduced cartilage thickness \rightarrow elevated cartilage stress \rightarrow further reduction in cartilage thickness \rightarrow patellofemoral osteoarthritis (PFJOA)).\textsuperscript{2}

It has been reported that persons with elevated patellofemoral joint loading exhibit decreased thickness and diminished deformational behaviour of patellar cartilage when compared with age-matched and activity-matched controls.\textsuperscript{5,7} However, the finding of decreased cartilage thickness in this population is not consistent across studies.\textsuperscript{6}


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