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Breaking the Law of Valgus: the surprising and unexplained prevalence of medial patellofemoral cartilage damage

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Abstract

Objectives—To compare the prevalence of medial and lateral patellofemoral (PF) cartilage damage in three large osteoarthritis (OA) studies and determine the relationship of this damage to varus, neutral, and valgus knee alignment.

Methods—In the Boston OA of the Knee (BOKS), Framingham OA (FOA), and Multicenter OA (MOST) studies, MRIs were read for cartilage morphology at the medial and lateral patella and trochlea femoris using Whole-Organ MRI Scores (WORMS). WORMS scores 2 (any cartilage defect), 3 (areas of partial thickness loss), 4 (diffuse partial thickness loss), and 5 (extensive full thickness loss) were all variously considered as thresholds to identify damage that may indicate OA. Full-limb radiographs were measured for mechanical alignment, and varus ($< -2^\circ$), neutral (-2° to 2°), and valgus ($> 2^\circ$) knees were identified.

Results—The prevalence of medial PF cartilage damage exceeded that of lateral damage in all three OA studies and according to nearly every threshold. Only among severely involved knees (WORMS 4 or 5) did the prevalence of lateral PF cartilage damage approximate that of medial damage. The high prevalence of medial PF damage persisted in all strata of knee alignment. Even among knees with valgus malalignment, the prevalence of lateral PF cartilage damage equaled or surpassed that of medial PF damage only when the threshold was specific to severely involved knees.

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COMPETING INTERESTS

No competing interests are relevant to this article.

Conclusions—Medial PF cartilage damage is at least as prevalent among older adults as lateral PF cartilage damage.

Keywords

Osteoarthritis; Knee; Patellofemoral Joint; Prevalence; Articular Cartilage

INTRODUCTION

In their classic textbook on disorders of the patellofemoral (PF) joint, Ficat and Hungerford introduced a widely used concept in understanding the location and cause of disease in the PF compartment of the knee. They called it the “Law of Valgus.”[1] With reference to the valgus quadriceps angle (the Q angle), which acts like a bowstring to pull the patella laterally during knee motion, the Law of Valgus dictates that the predominant frontal plane force acting on the patella during activity is directed laterally (Figure 1). Accordingly, Ficat and Hungerford argued that PF pain syndrome was invariably a problem of excessive lateral pull and increased loading of the lateral PF compartment. It followed that later life PF osteoarthritis (OA) was also principally a disease of excessive loading and cartilage damage in the lateral PF compartment. In the years since Ficat and Hungerford, others[2-5] have repeated the assertion that PF OA is a consequence of excessive lateral loading and that the only circumstances under which medial PF OA should arise with any frequency are those in which frontal plane varus knee malalignment (such as may occur with pre-existing medial tibiofemoral OA) has supplanted the typical valgus Q angle with an atypical varus Q angle.

Despite widespread acceptance of the Law of Valgus and its supposed implications for PF disease, there have not been large-scale studies to corroborate the assertion that medial PF OA is less prevalent than lateral PF OA. Instead, only one small Japanese study[6] has corroborated these predictions. In a study of 44 knees with PF OA on skyline radiograph, only 7 had medial PF disease. The remaining 36 knees all had lateral PF OA, as evidenced by greater lateral than medial PF joint space narrowing on x-ray. Among the 7 knees judged to have medial PF disease, all but one also had concurrent medial tibiofemoral OA with varus knee malalignment. Thus, the findings of this small radiographic study were interpreted as being supportive of the traditional assertion that medial PF OA is a relatively uncommon condition and that, when present, it is almost invariably a consequence of primary varus knee malalignment.

Our own clinical experience contradicts these conclusions. In a variety of practice settings, we have found the clinical signs and symptoms of medial PF OA to be common, with many patients reporting medial peripatellar pain during activity and medial retropatellar tenderness during examination. Our experience is paralleled by reports from cadaver studies that fibrillation and other signs of cartilage damage are frequently identified at the medial patellar facet.[7, 8] These findings suggest that medial PF disease may be common.

Using standard radiographic methods, lateral and medial PF OA are distinguished by comparing the extent of lateral versus medial PF joint space narrowing (JSN). Implicit in this comparison is an assumption that the presence and extent of lateral PF JSN on x-ray corresponds to the presence and extent of actual lateral cartilage damage in the PF joint.[9]

With the advent of MRI and large-scale OA studies, it is possible to compare the prevalence of medial and lateral PF cartilage damage directly. Such an evaluation would serve to confirm or refute the assertion that lateral PF disease is more prevalent than medial PF disease. If this assertion is confirmed, confidence will be strengthened in continued efforts to develop and apply surgical and rehabilitative treatments that unload the lateral PF compartment by medially realigning the patella. If, on the other hand, assertions about the low prevalence of medial PF cartilage damage are refuted, many common surgical and rehabilitative interventions for PF OA may need to be reconsidered.

Our objective was to determine and compare the prevalence of medial and lateral PF cartilage damage using compartment-specific MRI measures of cartilage morphology in three large OA studies. In addition, we sought to better understand the prevalence of medial and lateral PF cartilage damage by determining their relationship to varus, neutral, and valgus knee alignment.

METHODS

We determined the prevalence of medial and lateral PF cartilage damage, and of isolated medial, isolated lateral, and mixed medial and lateral PF cartilage damage within the Boston Osteoarthritis of the Knee Study (BOKS), the Framingham Osteoarthritis Study (FOA), and the Multicenter Osteoarthritis Study (MOST). In each of these three OA studies, MRIs were read for cartilage morphology at the medial and lateral patella and at the medial and lateral trochlea femoris using semiquantitative Whole-Organ MRI Scores (WORMS).[10] In addition, full-limb anteroposterior (AP) radiographs were measured for mechanical axis knee alignment using an established protocol.[11] Valgus measures were recorded as positive values, while varus measures were recorded as negative values. From these measured values, we identified the presence of varus (<-2 degrees), neutral (-2 to 2 degrees), and valgus (> 2 degrees) knee alignment and determined the relationship of knee alignment to prevalent medial and lateral PF cartilage damage.

Boston Osteoarthritis of the Knee Study (BOKS)

The Boston Osteoarthritis of the Knee Study (BOKS) is a completed prospective cohort study investigating the natural history of symptomatic knee OA. Participants in BOKS had at least one knee that met the American College of Rheumatology (ACR) criteria for clinical diagnosis of primary knee OA. A validated survey instrument supplemented by questions about medication use was used to exclude patients with rheumatoid arthritis and other types of inflammatory arthritis.[12] The sources and recruitment of BOKS participants have been detailed elsewhere.[13] BOKS was based at the Department of Veterans Affairs Medical Center in Boston, Massachusetts, USA and approved by the Institutional Review Boards (IRBs) at the Veterans Administration Health Care System and Boston University Medical Center.

MRIs of the more symptomatic knee were acquired in BOKS at the baseline visit using a Signa 1.5 Tesla (T) system (General Electric Corp., Milwaukee, Wisconsin, USA) and a phased-array knee coil. A positioning device was used to ensure uniformity in positioning the knee in full extension with the foot perpendicular to the table. The imaging protocol

included sagittal, coronal, and axial spin-echo fat-suppressed proton density and T2-weighted images (repetition time (TR) 2200 ms; time to echo (TE) 20/80 ms; slice thickness 3 mm; 0.3 mm interslice gap; 256×192 matrix; 120 mm² field of view (FOV); echo train length (ETL) 1). To characterize PF cartilage damage and its location, we focused on the axial and sagittal views.

Standing full-limb radiographs were obtained at the first BOKS follow-up visit with the knee fully extended and according to the same established protocol that was utilized in the other two OA studies.[11] Mechanical axis knee alignment was measured on a continuous scale to the nearest 0.1 degree from the digitized images. The intra-class correlation coefficient (ICC) for inter-rater reliability in the measurement of knee alignment was 0.97.

Framingham Osteoarthritis Study (FOA)

Participants in the Framingham Osteoarthritis Study (FOA)[14-16] were from two subgroups: 1) members of the Framingham Heart Study Offspring cohort, a longitudinal population-based study to examine risk factors for heart disease,[17] and 2) a newly recruited Community cohort. The first of these two subgroups, the Offspring cohort, included offspring of participants in the original Framingham Heart Study and spouses of these offspring. Details of this cohort have been described previously.[18]

Participants in the Framingham Community cohort were drawn from a population-based sample of the Framingham community, as described elsewhere.[19] To be included, subjects had to be 50 to 80 years old and ambulatory (use of assistive devices such as canes and walkers was permitted). As in BOKS, a validated survey instrument supplemented by questions about medication use was used to exclude patients with rheumatoid arthritis and other types of inflammatory arthritis.[12] Those with bilateral total knee prostheses or contraindications to MRI were also excluded. In contrast to BOKS, neither of the two FOA subgroups was selected according to the presence or absence of knee OA. Approval for study of the combined FOA cohort was obtained from the IRB at the Boston University Medical Center. Participants were examined in 2002-2005.[14]

MRI examinations of the right knee were performed using a 1.5 T magnet (Siemens Symphony, Erlangen, Germany), unless the right knee had a prosthesis, in which case the left knee was imaged. A positioning device was used to ensure consistent knee placement. In the Offspring subgroup, which had been extensively studied, only participants reporting knee pain, aching, or stiffness underwent MRI of the right knee. In contrast, MRIs were acquired in all members of the Framingham Community subgroup, regardless of knee symptom status. The imaging protocol in the FOA study included sagittal, coronal, and axial turbo spin-echo fat-suppressed intermediate-weighted sequences (TR 3610 ms, TE 40 ms, 3.5 mm slice thickness, 0 mm interslice gap, 256 × 256 matrix, 139 mm² FOV, ETL 6).[15, 16] To characterize PF cartilage damage and its location, we again focused on the axial and sagittal views.

Standing full-limb radiographs were obtained of all eligible knees from participants in the Framingham Community cohort. These radiographs were read for mechanical axis knee alignment among a subsample of participants that had been previously identified for

inclusion in a case-control study of knee OA.[20] A single experienced reader (ICC= 0.99 for intra-rater reliability) measured mechanical axis knee alignment to the nearest 0.1 degree from the digitized images using Efilm software (Merge Healthcare, Inc. Milwaukee, WI, USA).

Multicenter Osteoarthritis Study (MOST)

The Multicenter Osteoarthritis Study (MOST) is a prospective study whose overall goal is to identify risk factors for incident and progressive knee OA in a large sample of adults aged 50 to 79 with either existing knee OA or at high risk of developing the disease. Those considered at high risk included persons who were overweight or obese, had knee pain, aching, or stiffness on most of the last 30 days, had a history of knee injury that made it difficult to walk for at least one week, or had previous knee surgery. Participants were recruited from two communities in the United States: Birmingham, Alabama and Iowa City, Iowa. Details about recruitment are reported elsewhere.[21] The protocol for MOST was approved by IRBs at the University of Iowa in Iowa City, the University of Alabama in Birmingham, the University of California in San Francisco, and Boston University Medical Center.

Among MOST participants who did not have contraindications, MRIs of both knees were obtained at the baseline visit using a 1.0 T system (OrthOne, ONI Inc., Wilmington, MA, USA) and a circumferential extremity coil. The imaging protocol in the MOST study included sagittal and axial turbo spin-echo fat-suppressed intermediate-weighted sequences (TR 4800 ms, TE 35 ms, 3 mm slice thickness, 0 mm interslice gap, 288×192 matrix, 140 mm^2 FOV, echo train length 8) and a coronal STIR sequence (TR 6650 ms, TE 15 ms, TI 100 ms, 3 mm slice thickness, 0 mm interslice gap, 256×192 matrix, 140 mm^2 FOV, ETL 8).

Standing full-limb radiographs were obtained at the baseline MOST visit with the knee fully extended according to the same protocol used in the BOKS and FOA studies.[11] Two readers (inter-reader ICC= 0.99) measured mechanical axis knee alignment to the nearest 0.1 degree from the digitized images.

MRI Readings for Patellofemoral Cartilage Damage

In the BOKS, FOA, and MOST studies, the same two experienced musculoskeletal radiologists (AG and FWR) executed identical reading protocols. Cartilage morphology was assessed separately on the medial and lateral patellar and trochlear surfaces using WORMS. [10] The WORMS cartilage scale ranges from 0-6, with scores defined as follows: 0=normal thickness and signal; 1=normal thickness but increased signal on T2-weighted images; 2.0=partial thickness focal defect <1 cm in greatest width; 2.5=full thickness focal defect <1 cm in greatest width; 3=multiple areas of partial thickness (Grade 2.0) defects intermixed with areas of normal thickness, or a Grade 2.0 defect wider than 1 cm but <75% of the region; 4=diffuse (75% of the region) partial thickness loss; 5=multiple areas of full thickness loss (grade 2.5) or a grade 2.5 lesion wider than 1 cm but <75% of the region; 6=diffuse (75% of the region) full thickness loss. During blinded reassessment, weighted kappa for inter-reader reliability in WORMS scoring of patellar and trochlear regions was

0.63 (95% CI: 0.59, 0.67) among 67 knees in BOKS, 0.73 (0.71, 0.75) among 170 knees in FOA, and 0.78 (0.73, 0.82) among 10 knees in MOST.

It should be noted that WORMS geographic demarcations assign the nadir of the patella (the crista) to the medial patella, even though the crista may not, strictly speaking, be part of either the medial or lateral PF compartments. To assess the extent to which our findings may have been influenced by this aspect of the WORMS protocol, we blinded one of the radiologists (AG) to the hypothesis being tested and had him re-read a random selection of 26 MRIs from among knees that had been previously identified as having at least partial thickness (WORMS = 2) medial patellar cartilage damage. During this re-reading, we instructed the radiologist to score the medial patella separately from the crista.

There is currently no consensus definition specifying how severely cartilage must be damaged before OA is diagnosed on MRI. We applied four different cartilage morphology score thresholds to define PF cartilage damage that might be indicative of OA, and compared the prevalence of medial and lateral PF disease using each of these four diagnostic cutpoints. Thus, WORMS scores = 2 (any cartilage thickness defect), = 3 (multiple areas of partial thickness loss), = 4 (diffuse partial thickness loss), and = 5 (extensive full thickness loss) were all variously considered for the identification of cartilage damage that might be indicative of medial or lateral PF OA.

RESULTS

All three OA study populations had a mean age in the mid-60s (Table 1). Consistent with the fact that FOA study participants were recruited from the community without regard to the presence of knee pain or OA, these participants also had a slightly lower mean body mass index (BMI) (29.2 kg/m² in FOA vs. 30.7 kg/m² in BOKS and 30.0 kg/m² in MOST) and a lower prevalence of knee pain (29.6% in FOA vs. 94.0% in BOKS and 33.2% in MOST) and radiographic PF OA (15.7% in FOA vs. 64.0% in BOKS and 18.0% in MOST) than did participants in the other two studies. Because BOKS participants were recruited largely from among military veterans in the Veterans Administration Healthcare System, the proportion of females was lower in BOKS (41.1% in BOKS vs. 56.0% in FOA and 51.0% in MOST).

The prevalence of medial PF cartilage damage exceeded the prevalence of lateral PF cartilage damage in all three OA study populations and according to nearly every definition of PF disease (Table 2). When applying a threshold commonly used to identify cartilage damage suggestive of OA (WORMS = 2), the prevalence of medial PF damage exceeded that of lateral damage ($p < 0.01$) by 11-19% across the cohorts. This was true regardless of whether the patella alone was considered (left side of Table 2), or whether the corresponding trochlear surface of the femur was also considered (right side of Table 2). Only when consideration was limited to the most severely involved knees (WORMS = 4 or = 5) did the prevalence of lateral PF cartilage damage approximate that of medial PF cartilage damage.

In comparisons of the prevalence of isolated medial, isolated lateral, and mixed medial and lateral PF cartilage damage (Table 3), the most common finding was that of mixed medial and lateral damage. This was especially true when lower WORMS score thresholds (= 2 or

3) were used to define the presence of cartilage damage. When more restrictive thresholds were applied (WORMS scores of 4 or 5), both isolated medial and isolated lateral damage increased in prevalence relative to the prevalence of mixed PF damage. However, the prevalence of isolated medial PF cartilage damage still exceeded that of isolated lateral damage in most instances, regardless of whether the patella alone was considered or both the patella and trochlea femoris were considered (compare left and right sides of Table 3). As with the previous set of findings, it was only when the most restrictive threshold definitions of disease were applied (WORMS 4 or 5) that the prevalence of isolated lateral PF cartilage damage approximated that of isolated medial PF cartilage damage. In all studies, medial PF cartilage damage remained as or more prevalent than lateral damage within separate BMI (normal or underweight vs. overweight or obese), age (< 70 vs. > 70 years), and gender strata.

With these findings in mind, we then determined the extent to which the high prevalence of medial PF cartilage damage could be explained by its association with varus knee malalignment. Among knees with varus malalignment (<-2 degrees), there was indeed a higher prevalence of medial than lateral PF cartilage damage in all three OA studies and according to every threshold (left side of Figure 2). However, even among knees with neutral alignment (-2 to 2 degrees), the high prevalence of medial PF cartilage damage persisted (middle of Figure 2). Only when identification of cartilage damage was restricted to the most severely involved knees (WORMS 4 or 5) did the prevalence of lateral PF cartilage damage approximate that of medial PF damage among knees with neutral alignment. Even more striking was the high prevalence of medial PF cartilage damage among knees with valgus malalignment (> 2 degrees) (right side of Figure 2). Contrary to expectations, it was only when the threshold used to identify cartilage damage was specific to the most severely involved knees (WORMS 4 or 5) that the prevalence of lateral PF cartilage damage consistently surpassed that of medial damage in knees with valgus malalignment. These findings were strongly evident in the MOST study where large numbers of full-limb radiographs were available for analysis.

To determine the extent to which involvement of the patellar crista might account for the high prevalence of medial PF cartilage damage identified using WORMS, we re-read a random selection of 26 knee MRIs that had previously been identified as having medial patellar disease according to a commonly used MRI definition of OA (WORMS cartilage morphology score 2). In 23 of these 26 knees (88.5%; 95% CI: 76.2%, 100%), regions of the medial patella other than the crista had cartilage damage, indicating that crista involvement alone was not sufficient to account for the high prevalence of medial PF cartilage damage in these three OA studies.

DISCUSSION

Contrary to the assertions made by previous interpreters of the Law of Valgus, our findings indicate that medial PF cartilage damage is highly prevalent among older adults, and may even be more prevalent than cartilage damage in the lateral PF compartment. Only when identification of relevant cartilage damage was limited to the most severely involved knees (WORMS cartilage morphology scores 4 or 5) did the prevalence of lateral PF cartilage

damage approximate (or slightly exceed) that of medial PF damage. These findings were consistent across three large OA studies and regardless of whether neutrally aligned, varus malaligned, or valgus malaligned knees were considered. Scoring the patellar crista as part of the medial PF compartment appears to have had minimal influence on these results.

The patella is a sesamoid bone embedded within the tendon of the quadriceps muscle. Forces acting on the patella include the proximal pull of the four quadriceps muscles and the distal pull of the patellar tendon. The direction of the proximal pull is approximated with a line connecting the center of the patella to the anterior superior iliac spine. The direction of the distal pull is represented with a line connecting the center of the patella to the tibial tubercle. Where these two lines deviate from 180 degrees, we are left with a quadriceps or “Q” angle (Figure 1). In a normally aligned adult limb, the Q angle is valgus.

Acknowledging this, interpreters of the Law of Valgus have supposed that a higher prevalence of lateral than medial PF OA should result from the increased lateral loading that is produced. According to Ficat and Hungerford, medial PF disease should occur only rarely and then only in the presence of varus knee malalignment.

By outfitting knees with retropatellar pressure sensors under varying Q angle conditions, cadaveric studies[22, 23] have corroborated the assumption that increased valgus Q angles produce increased loading of the lateral PF compartment during the mid-range of knee flexion. However, valgus Q angles provide only one route by which compartmental loading of the PF joint can vary. The high prevalence of medial PF cartilage damage in the BOKS, FOA, and MOST studies suggests that some other common, but as yet unspecified, static or dynamic factors must be acting to increase the prevalence of medial PF joint damage in these older adult populations. Varus knee malalignment is likely to be one of these factors, as suggested several years ago by Elahi and colleagues[24] and confirmed in our data. However, varus knee malalignment did not account for all of the medial PF cartilage damage observed in these three OA studies. In fact, even among knees with neutral or valgus alignment, the prevalence medial PF cartilage damage remained high and often exceeded that of lateral PF damage in all three studies. Other factors must therefore be contributing to the high prevalence of medial PF disease.

Recent studies have used dynamic MRI to provide detailed assessments of patellofemoral kinematics.[25–28] While findings have generally been consistent with the Law of Valgus in demonstrating slight lateral displacement and tilting of the patella during the mid-ranges of knee flexion (even in healthy knees), these studies also describe medial displacement of the patella during the initiation of knee flexion. The relative positioning of the trochlear prominences and patella during the first 20 degrees of knee flexion may be critical in determining whether or not microtrauma and eventual cartilage damage is sustained with repeated knee bending activities.[29] In full extension, the patella is positioned superior and slightly lateral of the trochlear groove. As the knee begins to flex therefore, the patella must descend into the groove by first moving medially in relation to the femur.[30] Once situated in the groove and fully engaged with the medial and lateral trochlear prominences, the position of the patella is largely stabilized and alterations in patellar alignment (lateral displacement and tilting) are substantially limited by bony architecture. However, at the initiation of knee flexion, prior to full engagement of the patella with the trochlear groove,

patellofemoral alignment remains highly vulnerable to changes in tibial and/or femoral position.[31-33] Forces that displace the patella further medially during this critical early phase of knee flexion could contribute to medial PF cartilage damage. One such force may be foot pronation which can constrain the tibia to rotate internally[34, 35] and could thereby displace the tibial tubercle and inferior patella further medially, perhaps increasing load over the medial PF joint. Excessive foot pronation has been posited to contribute to frequent knee pain and cartilage damage among both older[36] and younger adults.[37-39]

Alternatively, the relative position of the trochlea and patella could be affected by rotation or torsion of the femur.[38] Excessive internal rotation or anteversion of the femur may contribute to heightened lateral PF stresses and lateral PF disorders.[40, 41] On the other hand, biomechanical theories seeking to explain medial PF disease with reference to excessive external rotation or retroversion of the femur are challenged by reports that these impairments may be uncommon among both younger[42, 43] and older adults with knee pain.[44, 45] An exception is the toe-out walking posture that is frequently observed among older adults with symptomatic medial tibiofemoral OA.[46] Externally rotating the entire limb and walking in an exaggerated toe-out posture can decrease load[47] and protect against OA progression[48] in the medial tibiofemoral compartment. However, its effects on the medial PF compartment are not known. If toe-out walking increases load on the medial PF compartment, then future investigations to determine the relationship of this potentially modifiable gait pattern to medial PF disease are essential.

Regardless of whether an explanation of the current findings is apparent, these results could still have major implications for the treatment of PF OA using biomechanical interventions. First, the Macquet procedure, a widely used surgical procedure, attempts to unload the symptomatic PF joint in patients with PF disease by realigning the patella medially. Such a procedure would clearly not be indicated if PF joint damage was predominantly medial, which, as we have shown in this study, is likely to be true for a substantial proportion of knees. Second, many physical therapy exercises for PF OA preferentially strengthen the medial quadriceps musculature (the vastus medialis obliquus or VMO) with the goal of drawing the patella medially during knee bending activities. This may or may not be effective, but it is clearly not indicated if the primary pathology involves injury to the medial PF compartment. Lastly, many PF braces and taping procedures use straps that attempt to pull the patella medially and away from the supposed site of OA in the lateral PF compartment. If, however, disease is localized to the medial rather than the lateral PF compartment, or if it affects both the medial and lateral PF compartments together, this strategy may not work. While more specific advice should await the results of investigations focused on painful knees that have been selected for biomechanical intervention, the findings of the present study nevertheless caution against the unconsidered prescription of any treatments that assume PF OA to be exclusively or predominantly lateral.

An additional assumption is called into question by our findings. Ficat and Hungerford presumed that PF pain syndrome and PF OA shared a similar etiology, one that involves excessive loading of the lateral PF joint. The findings of the current study suggest either that the elements of this presumed etiology are true only of PF pain syndrome while PF OA

follows a substantially different causal pathway (i.e., the two conditions are less related than previously supposed), or that a rethinking of PF pain syndrome is also in order.

A limitation of this study is that the PF joint was read using the WOMS scale, for which the medial patellar region includes the crista. To evaluate the extent to which this regional definition influenced our findings, we had knees with medial patellar cartilage damage re-read. We confirmed that the great majority (88.5%; 95% CI: 76.2%, 100%) of these knees had cartilage damage in regions of the medial patella that were distinct from the crista. Extrapolated to our larger study population, cartilage damage of the crista alone might explain no more than 24% (upper bounds of the 95% CI) of the observed prevalence of medial patellar damage, still leaving a very large proportion (76% to 100%) of the prevalence unexplained by conventional theories.

A consistent finding was that the prevalence of lateral PF damage approximated (or slightly exceeded) that of medial damage only when the most restrictive thresholds were used to identify relevant cartilage damage (WOMS 4 or 5). This finding might indicate that, while medial PF cartilage damage has a higher prevalence overall, the factors leading to lateral PF damage more frequently result in severe disease than do the factors leading to medial damage. In an 18-month longitudinal study of radiographic OA, Cahue et al[49], found that medial PF OA progression was less common than lateral PF OA progression (15% vs. 30% of knees). This finding could suggest that lateral PF disease, when present, is driven to progress more readily than medial PF disease. It is a limitation of the current cross-sectional study that no determination can be made of the relative rates of progression in medial and lateral PF cartilage damage.

In conclusion, these findings indicate that it is not safe to assume that PF cartilage damage and OA affect mainly the lateral joint compartment. Indeed, many persons with PF cartilage damage appear to have medial disease, either solely or in combination with lateral PF disease. A rethinking of the etiology of PF OA needs to account for the high prevalence of medial PF cartilage damage and offer a more realistic guide for biomechanical approaches to therapy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Fig. 1.

Fig. 2

Table 1
Description of BOKS, FOA, and MOST Participants

	BOKS 224 knees from 224 subjects	FOA 428 knees from 350 subjects	MOST 1621 knees from 1381 subjects
Age, mean (SD), year	66.7 (9.4)	65.0 (8.9)	62.1 (8.0)
Female, N (%)	92 (41.1)	196 (56.0)	843 (61.0)
BMI, mean (SD), kg/m ²	30.7 (4.8)	29.2 (5.5)	30.0 (4.9)
TF Alignment, N (%)			
Varus	140 (62.5)	359 (83.9)	777 (47.9)
Neutral	48 (21.4)	49 (11.5)	532 (32.8)
Valgus	36 (16.1)	20 (4.7)	312 (19.3)
Knee Pain, N (%)	200 (94.0)	124 (29.6)	537 (33.2)
Radiographic PF OA, N (%)	142 (64.0)	67 (15.7)	288 (18.0)
K&L Grade, N (%)			
0	17 (7.6)	220 (51.4)	667 (41.2)
1	40 (17.9)	19 (4.4)	301 (18.6)
2	51 (22.8)	98 (22.9)	316 (19.5)
3	70 (31.2)	59 (13.8)	276 (17.1)
3.5-4	46 (20.5)	32 (7.5)	59 (3.6)
PF JSN Grade, N (%)			
0	109 (49.1)	403 (94.6)	1396 (87.1)
1	68 (30.6)	12 (2.8)	84 (5.2)
2-2.5	30 (13.5)	8(1.9)	80 (5.0)
3	3 (6.8)	3 (0.7)	43 (2.7)

Table 2
Prevalence of medial and lateral patellofemoral (PF) cartilage damage in BOKS, FOA, and MOST studies

		Definition of PF Cartilage Damage							
		Patellar Cartilage Morphology Score				Either Patellar or Trochlear Cartilage Morphology Score			
		2	3	4	5	2	3	4	5
BOKS	Medial PF Damage % (n/N knees)	75.00 (168/ 224)	68.75 (154/ 224)	48.21 (108/ 224)	26.79 (60/ 224)	84.38 (189/ 224)	79.02 (177/ 224)	55.80 (125/ 224)	30.36 (68/ 224)
	Lateral PF Damage % (n/N knees)	61.61 (138/ 224)	58.04 (130/ 224)	45.54 (102/ 224)	24.11 (54/ 224)	73.66 (165/ 224)	68.75 (154/ 224)	54.46 (122/ 224)	28.57 (64/ 224)
FOA	Medial PF Damage % (n/N knees)	64.72 (277/ 428)	50.00 (214/ 428)	23.36 (100/ 428)	17.76 (76/ 428)	69.95 (298/ 426)	57.51 (245/ 426)	27.00 (115/ 426)	21.13 (90/ 426)
	Lateral PF Damage % (n/N knees)	50.93 (218/ 428)	43.22 (185/ 428)	16.59 (71/ 428)	10.98 (47/ 428)	55.40 (236/ 426)	47.65 (203/ 426)	21.60 (92/ 426)	16.43 (70/ 426)
MOST	Medial PF Damage % (n/N knees)	62.74 (1017/ 1621)	51.63 (837/ 1621)	20.30 (329/ 1621)	18.63 (302/ 1621)	69.19 (1116/ 1613)	56.11 (905/ 1613)	21.64 (349/ 1613)	19.78 (319/ 1613)
	Lateral PF Damage % (n/N knees)	45.34 (735/ 1621)	38.43 (623/ 1621)	19.74 (320/ 1621)	16.59 (269/ 1621)	51.08 (824/ 1613)	43.15 (696/ 1613)	23.56 (380/ 1613)	20.52 (331/ 1613)

Table 3
Prevalence of isolated medial, isolated lateral, and mixed medial and lateral patellofemoral (PF) cartilage damage in BOKS, FOA, and MOST studies

		Definition of PF Cartilage Damage							
		Patellar Cartilage Morphology Score				Either Patellar or Trochlear Cartilage Morphology Score			
		2	3	4	5	2	3	4	5
BOKS	No PF Damage % (n/N)	18.75 (42/ 224)	23.66 (53/ 224)	42.41 (95/ 224)	63.84 (143/ 224)	10.27 (23/ 224)	16.07 (36/ 224)	29.46 (66/ 224)	58.04 (130/ 224)
	Isolated Medial PF Damage % (n/N)	19.64 (44/ 224)	18.30 (41/ 224)	12.05 (27/ 224)	12.05 (27/ 224)	16.07 (36/ 224)	15.18 (34/ 224)	16.07 (36/ 224)	13.39 (30/ 224)
	Isolated Lateral PF Damage % (n/N)	6.25 (14/ 224)	7.59 (17/ 224)	9.38 (21/ 224)	9.38 (21/ 224)	5.36 (12/ 224)	4.91 (11/ 224)	14.73 (33/ 224)	11.61 (26/ 224)
	Mixed PF Damage % (n/N)	55.36 (124/ 224)	50.45 (113/ 224)	36.16 (81/ 224)	14.73 (33/ 224)	68.30 (153/ 224)	63.84 (143/ 224)	39.73 (89/ 224)	16.96 (38/ 224)
FOA	No PF Damage % (n/N)	30.37 (130/ 428)	44.86 (192/ 428)	71.73 (307/ 428)	77.57 (332/ 428)	25.82 (110/ 426)	38.73 (165/ 426)	66.43 (283/ 426)	72.54 (309/ 426)
	Isolated Medial PF Damage % (n/N)	18.69 (80/ 428)	11.92 (51/ 428)	11.68 (50/ 428)	11.45 (49/ 428)	18.78 (80/ 426)	13.62 (58/ 426)	11.97 (51/ 426)	11.03 (47/ 426)
	Isolated Lateral PF Damage % (n/N)	4.91 (21/ 428)	5.14 (22/ 428)	4.91 (21/ 428)	4.67 (20/ 428)	4.23 (18/ 426)	3.76 (16/ 426)	6.57 (28/ 426)	6.34 (27/ 426)
	Mixed PF Damage % (n/N)	46.03 (197/ 428)	38.08 (163/ 428)	11.68 (50/ 428)	6.31 (27/ 428)	51.17 (218/ 426)	43.90 (187/ 426)	15.02 (64/ 426)	10.09 (43/ 426)
MOST	No PF Damage % (n/N)	29.73 (482/ 1621)	40.84 (482/ 1621)	68.60 (1112/ 1621)	71.56 (1160/ 1621)	23.37 (377/ 1613)	36.58 (590/ 1613)	64.97 (1048/ 1613)	67.70 (1092/ 1613)
	Isolated Medial PF Damage % (n/N)	24.92 (404/ 1621)	20.73 (404/ 1621)	11.66 (189/ 1621)	11.84 (192/ 1621)	25.54 (412/ 1613)	20.27 (327/ 1613)	11.47 (185/ 1613)	11.78 (190/ 1613)
	Isolated Lateral PF Damage % (n/N)	7.53 (122/ 1621)	7.53 (122/ 1621)	11.10 (180/ 1621)	9.81 (159/ 1621)	7.44 (120/ 1613)	7.32 (118/ 1613)	13.39 (216/ 1613)	12.52 (202/ 1613)
	Mixed PF Damage % (n/N)	37.82 (613/ 1621)	30.91 (501/ 1621)	8.64 (140/ 1621)	6.79 (110/ 1621)	43.65 (704/ 1613)	35.83 (578/ 1613)	10.17 (164/ 1613)	8.00 (129/ 1613)