

West Chester University
Digital Commons @ West Chester University

Sports Medicine

College of Health Sciences

2014

Tibiofemoral Osteoarthritis After Surgical or Nonsurgical Treatment of Anterior Cruciate Ligament Rupture: A Systematic Review

Kyle P. Harris

Bucks County Community College

Jeffrey B. Driban

Tufts Medical Center

Michael R. Sitler

Bucks County Community College

Nicole M. Cattano

West Chester University of Pennsylvania, ncattano@wcupa.edu

Easwaran Balasubramanian

Temple University

Follow this and additional works at: http://digitalcommons.wcupa.edu/spomed_facpub

 Part of the [Sports Sciences Commons](#)

Recommended Citation

Harris, K. P., Driban, J. B., Sitler, M. R., Cattano, N. M., & Balasubramanian, E. (2014). Tibiofemoral Osteoarthritis After Surgical or Nonsurgical Treatment of Anterior Cruciate Ligament Rupture: A Systematic Review. *Journal of Athletic Training, 49*(3)
<http://dx.doi.org/10.4085/1062-6050-49.3.89>

This Article is brought to you for free and open access by the College of Health Sciences at Digital Commons @ West Chester University. It has been accepted for inclusion in Sports Medicine by an authorized administrator of Digital Commons @ West Chester University. For more information, please contact wccressler@wcupa.edu.

Tibiofemoral Osteoarthritis After Surgical or Nonsurgical Treatment of Anterior Cruciate Ligament Rupture: A Systematic Review

Kyle P. Harris, MS, ATC*; Jeffrey B. Driban, PhD, ATC, CSCS†; Michael R. Sitler, EdD, ATC, FNATA*; Nicole M. Cattano, MPH, ATC‡; Easwaran Balasubramanian, MD§

*Department of Health, Physical Education and Nursing, Bucks County Community College, Newtown, PA; †Division of Rheumatology, Tufts Medical Center, Boston, MA; ‡Department of Sports Medicine, West Chester University, PA; §Department of Clinical Orthopedic Surgery, Temple University, Philadelphia, PA

Objective: To determine if surgical or nonsurgical treatment of anterior cruciate ligament rupture affects the prevalence of posttraumatic tibiofemoral osteoarthritis (OA).

Data Sources: Studies published between 1983 and April 2012 were identified via EBSCOhost and OVID. Reference lists were then screened in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.

Study Selection: Studies were included if (a) treatment outcomes focused on a direct comparison of surgical versus nonsurgical treatment of anterior cruciate ligament rupture, (b) the prevalence of tibiofemoral OA was reported, and (c) they were written in English. Studies were excluded if (a) the included patients were treated with cast immobilization after surgery, (b) the mean follow-up was less than 10 years, or (c) the patients underwent anterior cruciate ligament revision surgery.

Data Extraction: Two independent investigators reviewed the included articles using the Newcastle-Ottawa Scale. Frequency of OA, surgical procedure, nonsurgical treatments, and participant characteristics were extracted and summarized. We calculated prevalence (%) and 95% confidence intervals for treatment groups for each individual study and overall. We

developed 2 × 2 contingency tables to assess the association between treatment groups (*exposed* had surgery, *referent* was nonsurgical treatment) and the prevalence of OA.

Data Synthesis: Four retrospective studies were identified (140 surgical patients, 240 nonsurgical patients). The mean Newcastle-Ottawa Scale score was 5 (range = 4–6 [of 10] points). Average length of follow-up was 11.8 years (range = 10–14 years). The prevalence of OA for surgically treated patients ranged from 32.6% to 51.2% (overall = 41.4%, 95% confidence interval = 35.0%, 48.1%) and for nonsurgical patients ranged from 24.5% to 42.3% (overall = 30.9%, 95% confidence interval = 24.4%, 38.3%).

Conclusions: Although OA prevalence was higher in the surgical treatment group at a mean follow-up of 11.8 years, no definitive evidence supports surgical or nonsurgical treatment after anterior cruciate ligament injury to prevent posttraumatic OA. Current studies have been limited by small sample sizes, low methodologic quality, and a lack of data regarding confounding factors.

Key Words: lower extremity, knee injuries, prevalence

Key Points

- This is the first systematic review to directly compare surgical and nonsurgical treatment of anterior cruciate ligament ruptures.
- No definitive evidence supports surgical or nonsurgical treatment after anterior cruciate ligament injury to prevent posttraumatic osteoarthritis.
- Research to date has been limited by small samples, low methodologic quality, and a lack of data on confounding factors.

Anterior cruciate ligament (ACL) reconstruction is often the treatment recommended after ACL rupture¹ in a physically active individual. The intended outcome of the surgery is to restore knee anatomy and biomechanics to a functional level, thereby reducing shear and torsional stresses on the menisci and articular surfaces^{2–9} and permitting a return to previous physical activities.^{7,10–13} After ACL reconstruction surgery, the short-term functional results appear favorable^{4,14–19}; however, over the long term, at least 28% to 87%^{1,5,7,15–17,19–33}

of these patients develop posttraumatic tibiofemoral osteoarthritis (OA).

Some patients opt for nonsurgical treatment of their ACL injuries.^{34,35} Patients recommended for this type of treatment typically have sufficient dynamic knee stability for their desired level of function^{3,36–39} and no secondary joint injury (eg, meniscal tear, collateral ligament sprain).³⁸ For patients treated nonsurgically, the reported rates of OA range from 11% to 73%.^{23,25–28,32,35,40–46} Although these patients are believed to have less disruption of lower

extremity biomechanics after ACL rupture,⁴⁷ it remains unclear if the likelihood of OA differs with surgical or nonsurgical treatment of the knee.^{15,35,40,44,48,49}

No systematic review has been reported to date on the prevalence of OA in patients with ACL ruptures treated surgically versus nonsurgically. Our purpose was to conduct a systematic review to determine if OA prevalence differed between the treatments. Studies used in this systematic review focused on a direct head-to-head comparison of surgical reconstruction and nonsurgical treatment. The advantage of a head-to-head approach was that the studies used the same criteria, such as the radiographic threshold for determining OA and the patient's previous level of function. Our intent was to inform evidence-based clinical care in the treatment of patients with ACL ruptures.

METHODS

Inclusion and Exclusion Criteria

Studies were included if (a) treatment outcomes focused on a direct comparison of surgical versus nonsurgical treatment of ACL ruptures, (b) the prevalence of tibiofemoral OA was reported, and (c) they were written in English. Studies were excluded if (a) patients were placed in casts after surgery, (b) the mean follow-up was less than 10 years,⁵⁰ or (c) patients underwent ACL revision surgery. Before 1983, the standard postsurgical care was straight-leg casting for 8 to 12 weeks.⁵¹ More contemporary postsurgical treatment calls for mobilization and rehabilitation beginning immediately after surgery.⁵² Because the long-term outcomes may differ between these postsurgical treatment protocols, we excluded articles published before 1983. Randomized control trials were initially considered for inclusion; however, none met the eligibility criteria for inclusion. If the reviewer (K.P.H.) was unsure that a study met all the necessary criteria, the study was reviewed by the other authors, and a consensus was reached.

Search Strategy

We conducted a comprehensive literature search from 1983 through April 2012 with the assistance of an experienced reference librarian. Databases searched with EBSCOhost were Academic Search Premier, CAB abstracts, CINAHL, Education Research Complete, Education Resources Information Center, MEDLINE, SPORTDiscus with full text, and Research Starters-Education. Databases searched with OVID were Cochrane Database of Systematic Reviews, ACP Journal Club (1991 to April 2012), Database of Abstracts of Reviews of Effects (second quarter of 2012), Cochrane Library Central Register of Controlled Trials (second quarter of 2012), Cochrane Library Methodology Register, Cochrane Library Health Technology Assessment, Cochrane Library Economic Evaluation Database, Journals@Ovid, Global Health, and Ovid MEDLINE. Key words used in the database searches were *ACL or anterior cruciate ligament and osteoarthritis or osteoarthrosis or degenerative joint disease or arthritis or coxarthrosis or gonarthrosis and reconstruct** or *repair or surgery or replacement and meniscus or menisci or tear or torn or injury or laceration and meniscus or menisci or tear or torn or injury or injuries or injured*. Although we focused on tibiofemoral OA, we did not specify the type of

OA for the literature search to avoid eliminating studies of both tibiofemoral and patellofemoral OA.

Study Selection

The primary search yielded 799 studies, and the lead author (K.P.H.) screened the titles, key words, dates of publication, and abstracts. A total of 759 articles were eliminated because their titles or abstracts indicated that the studies either did not meet the inclusion criteria or they met the exclusion criteria. We then obtained the full text of the 40 remaining articles and further screened them for all inclusion and exclusion criteria. The reference lists of all 40 full-text articles were searched manually to identify any additional articles not located through the electronic database search process; no additional articles were cited. A total of 11 articles were provisionally identified as meeting the inclusion criteria. For 2 studies,^{26,53} it was unclear if patients were placed in casts after surgery. Therefore, we contacted the first author of each and confirmed that patients were treated with casts, so the studies were excluded. Three additional studies⁵⁴⁻⁵⁶ were later excluded during the data-extraction process because prevalence data were not reported. Another study²⁸ was excluded due to a mixed study design (cohort and cross-sectional study design) and the inclusion of patients who did not improve with nonsurgical treatment. One additional study²⁷ was later eliminated for not meeting the inclusion criteria for an adequate length of follow-up. The study-elimination process is shown in the Figure. The final 4 studies are presented in Table 1.

Assessment of Study Quality

Two independent reviewers (K.P.H., N.M.C.) assessed the quality of all included studies using the Newcastle-Ottawa Scale (NOS), which we modified for use in this systematic review (Appendix).⁵⁸ Although not developed specifically for OA research, the NOS has been recommended for the qualitative evaluation of observational studies⁵⁸ because it is easy to use and includes specific items based on study design (eg, case control, cohort).⁵⁸ A recent analysis⁵⁹ of several quality-assessment tools also deemed the NOS appropriate for its intended use in this study. We transferred the NOS to an electronic form for more efficient assessment and operationally defined the NOS criteria to make them applicable to the study population. Specifically, questions that assessed the representativeness of the cohorts (items 1 and 2) were modified to meet the objective of our systematic review. The question regarding the representativeness of the ACL population (item 1) was changed from "representativeness of the community of selection" to "representativeness of the general ACL-deficient population." The question assessing the selection of the nonexposed cohort (item 2) was changed from "drawn from the same community as the exposed cohort" to "drawn from the general ACL-deficient population." The question assessing the ascertainment of exposure of the exposed cohort (item 3) was defined as the "surgical cohort." To assess the exposure of the nonsurgical cohort, a fifth item was added. Items used to assess the comparability of cohorts were defined as follows: (a) Did the study control for secondary injuries (ie, meniscal, ligamentous injuries other than ACL injury)? and b) Did the

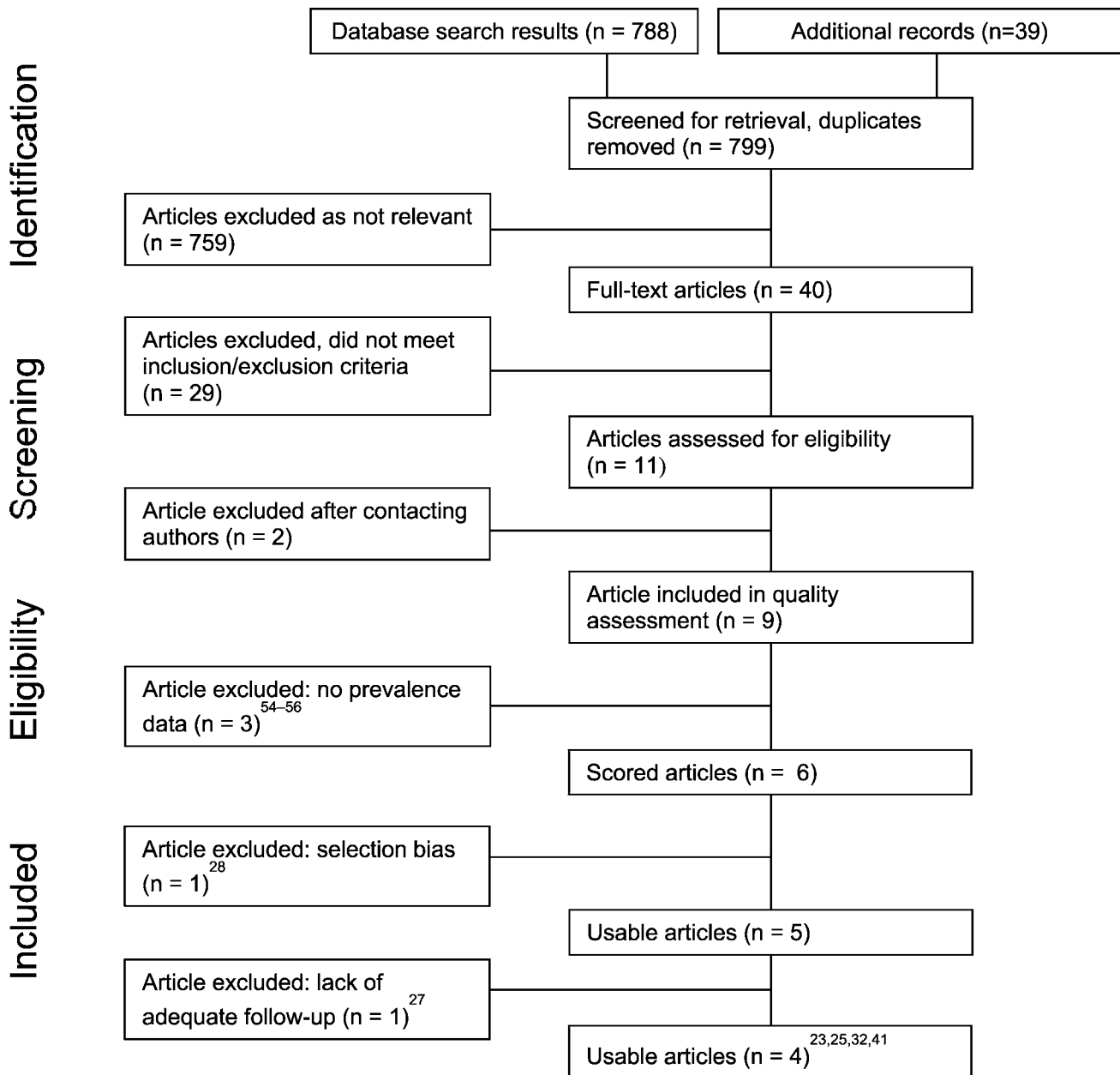


Figure. Study-elimination sequence according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.⁵⁷

study control for the body mass index (BMI) of participants at either baseline or follow-up? This indicated that the authors attempted to control for important, known influences on the development of OA. Of the 3 items designated to assess the outcomes of each study, 2 items were further defined. The adequate follow-up time for the outcome (prevalence of OA) to occur (item 2) was defined as 10 or more years, and the adequacy of follow-up cohorts (item 3) was defined as greater than 80%. The modified total possible score was 10 points, as opposed to 9 in the original instrument.

To standardize the way in which items were scored by the reviewers, we established criteria for specific questions on the NOS. To score the representativeness of the surgical group (item 1), the following criteria were assessed: (a) Were patients randomly or consecutively chosen (reduced

risk of selection bias)? (b) Were patients between 15 and 55 years of age at the time of injury⁶⁰⁻⁶²? (c) Was the cohort mean BMI⁶² less than 30? and (d) Was surgery performed within 8 weeks of injury? Studies that fulfilled all 4 criteria were considered representative of the general population with ACL ruptures. Studies adhering to 3 of the 4 criteria were considered to be somewhat representative. Investigations adhering to 2 or fewer of the 4 criteria were not considered representative of the general population with ACL ruptures. To score the representativeness of the nonsurgical group (item 2), we used 3 criteria: (a) Were patients randomly or consecutively chosen? (b) Were patients between 15 and 55 years of age at the time of injury? and (c) Was the cohort mean BMI less than 30? Studies that fulfilled all 3 criteria were considered representative of the general population with ACL ruptures.

Table 1. Study Design, Participant Characteristics, Exposures, and Outcomes for Studies (n = 4) Included in the Systematic Review Extended on Next Page

Authors (Year)	Cohort Study Design	Participant Characteristics	Exposure Definition: Treatment Type	
			Surgical (n)	Nonsurgical (n)
Lohmander et al ²³ (2004)	Retrospective	Age range, 26–40 y, women only, Sweden, league soccer: identified through insurance company archive	Various reconstruction methods (62% autologous patellar tendon graft), n = 41 with radiographic data	No explanation of nonoperative treatment, n = 26 with radiographic data
Kessler et al ⁴¹ (2008)	Retrospective	Age range = 12.5–54 y, males = 62%, females = 38%, participants matched age, sex, body mass index, Tegner score, follow-up	Bone–patellar tendon–bone reconstruction, procedures performed by various practitioners, radiographic data: n = 60	Standardized, nonoperative treatment program, carried out by various physiotherapists, radiographic data: n = 49
von Porat et al ³² (2004)	Retrospective	Age range = 30–56 y, men only, Swedish league soccer players identified through insurance company archive	Various surgical methods (most common: patellar tendon graft)	No explanation of nonoperative treatment
Meuffels et al ²⁵ (2009)	Retrospective	Mean age = 37.7 y, males = 76%, females = 24%, Netherlands, participants matched by age, sex, Tegner score	Bone–patellar tendon–bone reconstruction, performed by 1 of 2 surgeons, radiographic data: n = 50	Rehabilitation program: edema reduction, range of motion, hamstrings and quadriceps strengthening exercises

Abbreviation: KL, Kellgren-Lawrence Scale.

^a Maximum possible score for modified instrument = 10.

Those adhering to 2 or fewer of the 3 criteria were not considered representative of the general population with ACL ruptures.

Before rating the quality of the 4 included studies, we pilot tested the rating procedures using 3 sample articles that were not included in the analysis. Studies were randomized by a blinded, independent investigator (M.R.S.). Each article was then read and scored independently by 2 raters. Consensus scores were determined for each article. If the scores of rater 1 (K.P.H.) and rater 2 (N.M.C.) agreed, then that score was used as the consensus score. If the scores of raters 1 and 2 differed by 1 point, the raters discussed and agreed on a consensus score. If the scores of raters 1 and 2 scores differed by 2 or more points, a third rater (J.B.D.) reviewed the article, and a final consensus score was agreed on by all 3 raters.

We created a spreadsheet for data extraction. The following information was extracted from each of the studies by the primary investigator (K.P.H.): (a) publication information: first author’s name, journal, and year of publication; (b) study methods: study design, report of meniscal injury, and NOS score; (c) OA outcomes: definition of OA and time of follow-up; (d) patient descriptors: source of exposed (surgical) and nonexposed (nonsurgical) cohorts, matching variables (eg, age, sex, Tegner activity score), sport, level of participation, percentage lost to follow-up, sample size, age, weight, height, BMI; and (e) outcomes measures: frequency of OA, prevalence of OA ($[\# \text{ yes surgical} / \text{total surgical}] * 100$), and adjustment variables (eg, meniscal injury, age, BMI).

For each study, the number of patients with OA at follow-up, stratified by treatment group (surgical, nonsurgical), was populated in a 2 × 2 contingency table. Prevalence (%)

of OA was calculated using the following formula: number with OA in a treatment group/total in the treatment group at baseline. Prevalence ratios (PRs) were calculated using the formula ($\# \text{ surgical and OA} / \text{all surgical}$)/($\# \text{ nonsurgical and OA} / \text{all nonsurgical}$). The nonsurgical treatment group was considered the referent group. We calculated 95% confidence intervals (CIs) for proportions and PRs using standard methods⁶³ and prevalence proportions and PRs for each individual study and for all studies combined (overall prevalence).

RESULTS

All 4 reviewed studies^{23,25,32,41} used a retrospective cohort design. A total of 380 patients (260 men, 120 women), 140 (37%) of whom were treated surgically, were included in the 4 studies.^{23,25,32,41} The mean patient ages reported at follow-up^{25,32} and time of trauma^{23,41} were 37.9 and 24.9 years, respectively. In all 4 studies,^{23,25,32,41} bone–patellar tendon–bone autograft was the primary surgical method. The reported postsurgical follow-up for each study ranged from 10 to 14 years (mean = 11.8 years).

Methodologic Quality

No study fulfilled all of the NOS criteria. The highest score recorded was 6 (of 10 possible points), and the lowest was 4. The mean score for all studies was 5 points. All studies^{23,25,32,41} included patients with meniscal injury; authors of only 1 study²³ adjusted statistically for this using a logistic regression model. Two groups^{25,41} used matching variables (age, sex, and Tegner activity score) in allocating the exposed and nonexposed cohorts. No authors reported

Table 1. Extended From Previous Page

Outcome Definition: Tibiofemoral Osteoarthritis Assessor/Reliability	Original Cohort with Follow-up Radiologic Examinations, %	Length of Follow-up, Mean y	Newcastle-Ottawa Scale Score ^a
KL grade > 2, all radiographs graded by same author without prior knowledge of injured side, full weight-bearing, anteroposterior radiographs at 15° of knee flexion	67/84, 80%	12	4
KL grade > 1, two independent readers, full weight-bearing, anteroposterior radiographs at 0° of knee flexion	109/149, 73%	11	5
KL grade > 2, bilateral posteroanterior radiographs at 15° of knee flexion, single reader of radiographs, blinded to injured side	154/205, 75%	14	5
KL grade > 1, weight-bearing posteroanterior and Rosenberg view radiographs taken; 2 blinded, independent readers evaluated all radiographs	50/50, 100%	10	6

inclusion and exclusion criteria for patients treated nonsurgically or fulfilled the requirement of “representative of the general ACL-deficient population” for either the exposed or nonexposed cohort. All studies^{23,25,32,41} ascertained exposure of the surgically treated cohort through surgical records or a structured interview. No group reported the absence of OA (eg, on radiographs) at the start of the study, mitigating the ability to determine the incidence of OA.

Prevalence of Tibiofemoral OA

Overall, OA prevalence for the 4 studies^{23,25,32,41} ranged from 24.5% to 51.2%. The OA prevalence for surgically treated patients ranged from 32.6% to 51.2% (overall = 41.4%, 95% CI = 35.0, 48.1) and for nonsurgical patients ranged from 24.5% to 42.3% (overall = 30.9%, 95% CI = 24.4, 38.3). Because all studies included patients with compromised menisci, we did not report prevalence data comparing isolated ACL ruptures with nonisolated ACL ruptures.

Prevalence Ratios of Tibiofemoral OA

Across the 4 studies, the PRs of tibiofemoral OA among surgical patients compared with nonsurgical controls ranged from 1.01 to 1.84. The surgical group had a 34% higher prevalence of tibiofemoral OA (PR = 1.34; 95% CI = 1.01, 1.77) compared with the nonsurgical group (Table 2).

Radiologic Classification System

To quantify radiographic OA, authors of all 4 included studies^{23,25,32,41} used the Kellgren-Lawrence score.⁶⁴ Two studies^{25,41} defined the presence of OA as greater than or

equal to Kellgren-Lawrence grade 1. Combined, these studies demonstrated an OA prevalence of 45.9% (95% CI = 35.7, 56.4) for surgically treated patients and 25.7% (95% CI = 17.1, 36.6) for nonsurgically treated patients. The surgical group had a higher prevalence of tibiofemoral OA than did the nonsurgically treated patients (PR = 1.79, 95% CI = 1.14, 2.81). Two studies^{23,32} defined OA as a Kellgren-Lawrence score of greater than or equal to grade 2. Use of this scale resulted in a reported OA prevalence of 38.5% (95% CI = 30.5, 47.0) for surgically treated patients and 35.1% (95% CI = 26.1, 45.3) for nonsurgically treated patients. Based on these 2 studies, the prevalence of tibiofemoral OA was similar among both treatment groups (PR = 1.09, 95% CI = 0.77, 1.56). Furthermore, radiographic OA was determined using at least 2 patient positions. Two studies^{25,41} used full-weight-bearing anteroposterior radiographs with the knee at 0° of extension, and the other 2 studies^{23,32} used full-weight-bearing anteroposterior radiographs with the knee in 15° of flexion.

DISCUSSION

This is the first systematic review to report on the prevalence of OA in patients with ACL ruptures treated surgically or nonsurgically based on studies directly comparing these treatments.^{23,25,27,28,32,41,54–56} This approach allowed us to use the PR to compare OA prevalence between the treatments. Unfortunately, because the included studies did not rule out the presence of OA at baseline, the incidence of tibiofemoral OA could not be determined. This limited our systematic review to the evaluation of OA prevalence and the association (ie, the PR) between the treatments. Based on these results, the prevalence of tibiofemoral OA may be greater among surgically treated ACL-deficient patients than among nonsurgically treated

Table 2. Frequency, Prevalence (%), and Unadjusted Prevalence Ratios of Tibiofemoral Osteoarthritis by Treatment (Surgical or Nonsurgical) Among Athletes with Anterior Cruciate Ligament Ruptures

Author (Year)	Frequency			Prevalence, ^a % (95% Confidence Interval)	Crude Prevalence Ratios (95% Confidence Interval)
	Yes	No	Total		
Tibiofemoral Osteoarthritis					
Lohmander et al ²³ (2004)					
Surgical	21	20	41	51.2 (36.4, 65.8)	1.21 (0.71, 2.08), 1.00 (referent)
Nonsurgical	11	15	26	42.3 (25.5, 61.0)	
Total	32	35	67	47.8 (36.3, 59.5)	
Kessler et al ⁴¹ (2008)					
Surgical	27	33	60	45.0 (33.1, 57.5)	1.84 (1.04, 3.24), 1.00 (referent)
Nonsurgical	12	37	49	24.5 (14.6, 38.1)	
Total	39	70	109	35.8 (27.4, 45.1)	
Von Porat et al ³² (2004)					
Surgical	29	60	89	32.6 (23.7, 42.9)	1.01 (0.64, 1.60), 1.00 (referent)
Nonsurgical	21	44	65	32.3 (22.2, 44.4)	
Total	50	104	154	32.5 (25.6, 40.2)	
Meuffels et al ²⁵ (2009)					
Surgical	12	13	25	48.0 (30.0, 66.5)	1.71 (0.81, 3.63), 1.00 (referent)
Nonsurgical	7	18	25	28.0 (14.3, 47.6)	
Total	19	31	50	38.0 (25.9, 51.8)	
Overall					
Surgical	89	126	215	41.4 (35.0, 48.1)	1.34 (1.01, 1.77), 1.00 (referent)
Nonsurgical	51	114	165	30.9 (24.4, 38.3)	
Total	140	240	380	36.8 (32.1, 41.8)	

^a Prevalence percentages calculated using the following formula: (#surgical with osteoarthritis/total surgical)*100. Prevalence ratios calculated as (#surgical with osteoarthritis/all surgical)/(#nonsurgical with osteoarthritis/all nonsurgical).

ACL-deficient patients. Unfortunately, the lack of methodologic quality and insufficient data in these studies prohibit a conclusive statement. Regardless, the tibiofemoral OA prevalence rates for both treatments were higher than for the general population,^{65,66} which may suggest that an optimal long-term treatment strategy for preventing tibiofemoral OA after ACL injury is yet to be determined.

Surgical ACL treatments focus on reconstructing the damaged ligament to restore normal knee biomechanics. In contrast, nonsurgical ACL treatment consists of joint mobility training to regain full range of motion, muscle strengthening, and neuromuscular training to promote the restoration of knee function.⁶⁷ Shortly after injury, both surgical^{4,14-19} and nonsurgical^{35,46} treatment options appear favorable for the athlete who wants to return to activity. Although the short-term results are promising, the long-term results (10 years or more) of either treatment are less clear. Initial return-to-activity rates for nonsurgical patients appear encouraging, but only 10% to 14% of patients actually returned to their preinjury activity level without limitations.^{35,45} Furthermore, the rates of ACL-deficient patients who are unable to adequately cope with knee instability and later opt for surgery range from 12% to 39%.^{35,42,47,68}

One explanation for the increased long-term prevalence of OA after ACL injury is the disruption of joint biochemistry. Immediately after injury, the joint undergoes a cascade of changes (eg, increase in inflammatory mediators and cartilage turnover markers)^{10,49,69,70} that disrupt the equilibrium between synthesis and catabolism of articular cartilage,⁷¹ influencing how articular cartilage and subchondral bone respond to new loading patterns.^{3,10,49,72} Elevated levels of C-telopeptide fragments

in synovial fluid (a biomarker of cartilage degeneration) and matrix metalloproteinases (catabolic enzymes involved in the degradation of the extracellular matrix^{72,73}) occur within hours of ACL rupture; they gradually decrease over the next year but never return to preinjury levels.^{52,73} In the nonsurgically treated ACL-deficient knee, type II collagen cleavage begins to return to normal at 12 months after injury, whereas type II collagen synthesis remains elevated at 12 and 24 months postinjury.¹⁰ At the time of ACL reconstruction surgery, biochemical synthesis and degeneration of type II collagen were elevated in the injured knee compared with the normal knee.⁵² At 12 months postsurgery, although cleavage of type II collagen has returned to normal limits, synthesis of type II collagen is elevated and remains so through 24 months. At 24 months, aggrecan turnover begins to approach normal levels but is still elevated. Unfortunately, current treatment strategies do not address these biochemical changes to the joint and, therefore, both surgically and nonsurgically treated patients may be susceptible to tibiofemoral OA.

Although ACL reconstruction does not address the aforementioned biochemical concerns, it may correct the disrupted joint kinematics. However, the long-term outcomes are yet to be determined.^{13,74-76} Tashman et al¹³ used a 3-dimensional radiographic stereophotogrammetric motion-analysis system to determine joint kinematics in 6 ACL-reconstructed patients during downhill running. In all reconstructed knees, the femur was more externally rotated and adducted relative to the tibia than in the uninjured contralateral knee. Vertical loading during heel strike and loading rate directed in line with the tibia were less in patients with reconstructed ACLs than in healthy control participants.⁷⁵ Collectively, these findings demonstrate

persistent altered kinematics and biochemical changes not only postinjury but also postsurgery.

The findings of all studies included in this systematic review were limited by the fact that the type and incidence of meniscal injury were not controlled. Patients who have undergone a meniscal repair or meniscectomy have a higher prevalence of OA than those with no meniscal injury.^{2,19,22,24,26,31,33,34,45,76–81} In an ACL-deficient knee, which is already experiencing biomechanical and biochemical joint changes, meniscal injuries compound the risk of developing OA by further altering the mechanical loading and contact points on the articular cartilage.^{12,14,45,75} Meniscal status is important to the long-term OA outcomes of patients with ACL ruptures.^{2,15,19–22,24,26,31,33,34,41,77,81–83} In addition, although partial meniscectomies resulted in a greater risk of radiographic changes, the risk was still lower than with total meniscectomies, and substantial function may remain in the residual meniscus.⁸⁴ However, the potential effect of this remaining tissue on OA risk remains unknown.

The large age range in this systematic review indicates that these results are generalizable to the population with ACL ruptures, but it limits our ability to make inferences about specific groups of patients (eg, high school or college athletes). Although the authors of 2 studies^{25,41} matched patients according to their Tegner activity scores, authors of the other studies included patients ranging from high-level European soccer players to more sedentary patients injured in motor vehicle accidents or falls. Patients with various activity levels may react differently to treatment options and may have different risks of OA based on their activity levels (eg, sedentary lifestyle, high-level competition).^{84,85} Another limitation introduced by the large age range is that some patients may have already had joint degeneration at the time of surgery. Therefore, we could not assess whether incidence rates were different between nonsurgical and surgical treatments for ACL injuries.

It is also important to note that we identified only studies of patients with bone–patellar tendon–bone grafts. Other surgical techniques or grafts, such as the hamstrings tendon graft, were not investigated. Future researchers should evaluate the long-term effectiveness of new surgical approaches and graft selections because evidence suggests that patients reconstructed with hamstrings tendon grafts have lower rates of knee OA than those receiving bone–patellar tendon–bone grafts.^{86–88} Unfortunately, a critical challenge to performing long-term follow-up studies is that new treatment strategies may be adopted as the standard of care before high-quality, long-term follow-up studies can assess their long-term efficacy. This was evident during the study-selection process: a number of groups compared surgical and nonsurgical treatments yet included patients who were immobilized in casts after surgery. This practice is no longer the standard of care, but it was discarded recently enough that an insufficient amount of time has passed to allow degenerative changes to become detectable.

Both the number and quality of studies identified as eligible for inclusion in the systematic review were low. The small number of articles may reflect a publication bias (eg, papers without significant findings were not published). Among the 4 studies, the mean NOS score was 5 of 10 possible points, which was attributed to methodologic weakness and inadequate reporting. Although we redefined

certain NOS criteria in an effort to standardize the scoring, this could have resulted in lower study scores because some groups reported details concerning the item being assessed but did not provide enough information to meet the necessary number of criteria to qualify for credit on the NOS.

Although the Kellgren-Lawrence score is the most common method for detecting OA, disagreement exists as to the threshold for determining OA, which has been shown to affect the overall classification of OA.^{89–91} One strength of this systematic review was that we included only articles with direct comparisons: both treatment groups were evaluated using the same diagnostic criteria.^{23,25,32,41} A limitation to the radiographic OA classification system is the possibility of false negatives, especially in mild cases, when the diseased compartment or joint is compared with internal controls (ie, opposite compartment [medial versus lateral], contralateral knee). No authors of studies included in this systematic review used diagnostic magnetic resonance imaging, which is more costly than radiographic analysis but also more sensitive to degenerative changes.⁹²

Furthermore, if we do not know whether tibiofemoral OA was present at baseline, we cannot truly determine the risk of tibiofemoral OA. This is also problematic with respect to previous or subsequent knee injuries and is especially limiting when assessing OA in a strictly athletic population. Future researchers must not only include patients with no signs of radiographic OA at baseline but also be diligent in collecting and reporting both this information and a comprehensive history of previously sustained injuries.

Although the systematic review is designed to present the body of literature concerning a specific topic, our systematic review was limited by insufficient reporting on a number of factors, which, greatly limits the results from being generalized to the population of those with ACL ruptures. Authors of the included studies did not report on many factors associated with the development of OA (eg, osteochondral lesions, previous injury). Higher-quality study designs would aid our understanding of how OA develops after surgical or nonsurgical ACL treatment. A randomized control trial⁹³ comparing these 2 treatment options is thus far limited to a 2-year follow-up. We need more randomized clinical trials with sufficient posttreatment follow-up and effective control of confounding factors to increase our understanding of surgical versus nonsurgical management of ACL and the incidence of knee OA.

It is also important for future investigators to consider as subcohorts patients who do not undergo reconstruction but instead modify their level of activity (copers, noncopers, and adapters). Assessing the difference between surgical and nonsurgical treatment of ACL ruptures in copers and noncopers can lead to an improved understanding of the true effectiveness of the 2 treatment options by helping us to identify which treatment effectively decreases the episodes of instability and the occurrence of OA. Although some research has been completed to date,⁹³ lengthier follow-up is needed to better assess the development of OA.

CONCLUSIONS

To date, no definitive evidence supports surgical or nonsurgical treatment after ACL injury to prevent post-

traumatic OA. The prevalence of OA in the included studies was slightly higher in surgically treated than in nonsurgically treated ACL patients at follow-up of approximately 12 years. However, large, overlapping confidence intervals indicate that there is no clear difference. This finding may have clinical importance, but the available studies were methodologically weak. Therefore, a significant relationship cannot be determined between having or not having ACL reconstruction surgery and developing tibiofemoral OA. The current studies were limited by small numbers, low methodologic quality, and a lack of data on confounding factors. Future authors should account for the presence of OA at baseline and focus on directly comparing the surgical and nonsurgical treatment of ACL ruptures while controlling for confounding factors (eg, age, meniscal status, BMI, physical activity).

REFERENCES

- Jarvela T, Kannus P, Jarvinen M. Anterior cruciate ligament reconstruction in patients with or without accompanying injuries: a re-examination of subjects 5 to 9 years after reconstruction. *Arthroscopy*. 2001;17(8):818–825.
- Allen CR, Livesay GA, Wong EK, Woo SL. Injury and reconstruction of the anterior cruciate ligament and knee osteoarthritis. *Osteoarthritis Cartilage*. 1999;7(1):110–121.
- Barrance PJ, Williams GN, Snyder-Mackler L, Buchanan TS. Do ACL-injured copers exhibit differences in knee kinematics? An MRI study. *Clin Orthop Relat Res*. 2007;454:74–80.
- Andriacchi TP, Dyrby CO. Interactions between kinematics and loading during walking for the normal and ACL deficient knee. *J Biomech*. 2005;38(2):293–298.
- Howe JG, Johnson RJ, Kaplan MJ, Fleming B, Jarvinen M. Anterior cruciate ligament reconstruction using quadriceps patellar tendon graft, part 1: long-term followup. *Am J Sports Med*. 1991;19(5):447–457.
- Kaplan MJ, Howe JG, Fleming B, Johnson RJ, Jarvinen M. Anterior cruciate ligament reconstruction using quadriceps patellar tendon graft, part 2: a specific sport review. *Am J Sports Med*. 1991;19(5):458–462.
- Lebel B, Hulet C, Galaud B, Burdin G, Locker B, Vielpeau C. Arthroscopic reconstruction of the anterior cruciate ligament using bone–patellar tendon–bone autograft: a minimum 10-year follow-up. *Am J Sports Med*. 2008;36(7):1275–1282.
- Frost-Christensen LN, Mastbergen SC, Vianen ME, et al. Degeneration, inflammation, regeneration, and pain/disability in dogs following destabilization or articular cartilage grooving of the stifle joint. *Osteoarthritis Cartilage*. 2008;16(11):1327–1335.
- Glasson SS, Blanchet TJ, Morris EA. The surgical destabilization of the medial meniscus (DMM) model of osteoarthritis in the 129/SvEv mouse. *Osteoarthritis Cartilage*. 2007;15(9):1061–1069.
- Nelson F, Billingham RC, Pidoux I, et al. Early post-traumatic osteoarthritis-like changes in human articular cartilage following rupture of the anterior cruciate ligament. *Osteoarthritis Cartilage*. 2006;14(2):114–119.
- Buckland-Wright JC, Lynch JA, Dave B. Early radiographic features in patients with anterior cruciate ligament rupture. *Ann Rheum Dis*. 2000;59(8):641–646.
- Andriacchi TP, Briant PL, Beville SL, Koo S. Rotational changes at the knee after ACL injury cause cartilage thinning. *Clin Orthop Relat Res*. 2006;442:39–44.
- Tashman S, Collon D, Anderson L, Kolowich P, Anderst W. Abnormal rotational knee motion during running after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2004;32(4):975–983.
- Andreisek G, White LM, Sussman MS, et al. Quantitative MR imaging evaluation of the cartilage thickness and subchondral bone area in patients with ACL-reconstructions 7 years after surgery. *Osteoarthritis Cartilage*. 2009;17(7):871–878.
- Ferretti A, Conteduca F, De Carli A, Fontana M, Mariani PP. Osteoarthritis of the knee after ACL reconstruction. *Int Orthop*. 1991;15(4):367–371.
- Hertel P, Behrend H, Cierpinski T, Musahl V, Widjaja G. ACL reconstruction using bone–patellar tendon–bone press-fit fixation: 10-year clinical results. *Knee Surg Sports Traumatol Arthrosc*. 2005;13(4):248–255.
- Jomha NM, Borton DC, Clingeleffer AJ, Pinczewski LA. Long-term osteoarthritic changes in anterior cruciate ligament reconstructed knees. *Clin Orthop Relat Res*. 1999;358:188–193.
- Arendt EA, Agel J, Dick R. Anterior cruciate ligament injury patterns among collegiate men and women. *J Athl Train*. 1999;34(2):86–92.
- Sommerlath K, Lysholm J, Gillquist J. The long-term course after treatment of acute anterior cruciate ligament ruptures: a 9 to 16 year follow-up. *Am J Sports Med*. 1991;19(2):156–162.
- Hart AJ, Buscombe J, Malone A, Dowd GS. Assessment of osteoarthritis after reconstruction of the anterior cruciate ligament: a study using single-photon emission computed tomography at ten years. *J Bone Joint Surg Br*. 2005;87(11):1483–1487.
- Jonsson H, Riklund-Ahlstrom K, Lind J. Positive pivot shift after ACL reconstruction predicts later osteoarthritis: 63 patients followed 5–9 years after surgery. *Acta Orthop Scand*. 2004;75(5):594–599.
- Keays SL, Newcombe PA, Bullock-Saxton JE, Bullock MI, Keays AC. Factors involved in the development of osteoarthritis after anterior cruciate ligament surgery. *Am J Sports Med*. 2010;38(3):455–463.
- Lohmander LS, Ostergren A, Englund M, Roos H. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. *Arthritis Rheum*. 2004;50(10):3145–3152.
- Maletius W, Messner K. Eighteen- to twenty-four-year follow-up after complete rupture of the anterior cruciate ligament. *Am J Sports Med*. 1999;27(6):711–717.
- Meuffels DE, Favejee MM, Vissers MM, Heijboer MP, Reijman M, Verhaar JA. Ten year follow-up study comparing conservative versus operative treatment of anterior cruciate ligament ruptures: a matched-pair analysis of high level athletes. *Br J Sports Med*. 2009;43(5):347–351.
- Meunier A, Odensten M, Good L. Long-term results after primary repair or non-surgical treatment of anterior cruciate ligament rupture: a randomized study with a 15-year follow-up. *Scand J Med Sci Sports*. 2007;17(3):230–237.
- Myklebust G, Maehlum S, Engebretsen L, Bahr R. Clinical, functional, and radiologic outcome in team handball players 6 to 11 years after anterior cruciate ligament injury: a follow-up study. *Am J Sports Med*. 2003;31(6):981–989.
- Neuman P, Englund M, Kostogiannis I, Friden T, Roos H, Dahlberg LE. Prevalence of tibiofemoral osteoarthritis 15 years after nonoperative treatment of anterior cruciate ligament injury: a prospective cohort study. *Am J Sports Med*. 2008;36(9):1717–1725.
- Pinczewski LA, Lyman J, Salmon LJ, Russell VJ, Roe J, Linklater J. A 10-year comparison of anterior cruciate ligament reconstructions with hamstring tendon and patellar tendon autograft: a controlled, prospective trial. *Am J Sports Med*. 2007;35(4):564–574.
- Ruiz AL, Kelly M, Nutton RW. Arthroscopic ACL reconstruction: a 5–9 year follow-up. *Knee*. 2002;9(3):197–200.
- Ait Si Selmi T, Fithian D, Neyret P. The evolution of osteoarthritis in 103 patients with ACL reconstruction at 17 years follow-up. *Knee*. 2006;13(5):353–358.
- von Porat A, Roos EM, Roos H. High prevalence of osteoarthritis 14 years after an anterior cruciate ligament tear in male soccer players: a

- study of radiographic and patient relevant outcomes. *Ann Rheum Dis.* 2004;63(3):269–273.
33. Seon JK, Song EK, Park SJ. Osteoarthritis after anterior cruciate ligament reconstruction using a patellar tendon autograft. *Int Orthop.* 2006;30(2):94–98.
 34. McDaniel WJ, Dameron TB. Untreated ruptures of the anterior cruciate ligament: a follow-up study. *J Bone Joint Surg Am.* 1980; 62(5):696–705.
 35. Hawkins RJ, Misamore GW, Merritt TR. Follow-up of the acute nonoperated isolated anterior cruciate ligament tear. *Am J Sports Med.* 1986;14(3):205–210.
 36. Chmielewski TL, Hurd WJ, Rudolph KS, Axe MJ, Snyder-Mackler L. Perturbation training improves knee kinematics and reduces muscle co-contraction after complete unilateral anterior cruciate ligament rupture. *Phys Ther.* 2005;85(8):740–749.
 37. Eastlack ME, Axe MJ, Snyder-Mackler L. Laxity, instability, and functional outcome after ACL injury: copers versus noncopers. *Med Sci Sports Exerc.* 1999;31(2):210–215.
 38. Moksnes H, Snyder-Mackler L, Risberg MA. Individuals with an anterior cruciate ligament-deficient knee classified as noncopers may be candidates for nonsurgical rehabilitation. *J Orthop Sports Phys Ther.* 2008;38(10):586–595.
 39. Powell B, Hurd W, Snyder-Mackler L. Nonoperative patient management after acute, isolated anterior cruciate ligament injury. *Athl Ther Today.* 2006;11(2):24–27.
 40. Kannus P, Jarvinen M. Conservatively treated tears of the anterior cruciate ligament. Long-term results. *J Bone Joint Surg Am.* 1987; 69(7):1007–1012.
 41. Kessler MA, Behrend H, Henz S, Stutz G, Rukavina A, Kuster MS. Function, osteoarthritis and activity after ACL-rupture: 11 years follow-up results of conservative versus reconstructive treatment. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(5):442–448.
 42. McDaniel WJ, Dameron TB. The untreated anterior cruciate ligament rupture. *Clin Orthop Relat Res.* 1983;172:158–163.
 43. Pattee GA, Fox JM, Del Pizzo W, Friedman MJ. Four to ten year follow-up of unreconstructed anterior cruciate ligament tears. *Am J Sports Med.* 1989;17(3):430–435.
 44. Noyes FR, Mooar PA, Matthews DS, Butler DL. The symptomatic anterior cruciate-deficient knee, part I: the long-term functional disability in athletically active individuals. *J Bone Joint Surg Am.* 1983;65(2):154–162.
 45. Fowler PJ, Regan WD. The patient with symptomatic chronic anterior cruciate ligament insufficiency: results of minimal arthroscopic surgery and rehabilitation. *Am J Sports Med.* 1987;15(4):321–325.
 46. Giove TP, Miller SJ, Kent BE, Sanford TL, Garrick JG. Nonoperative treatment of the torn anterior cruciate ligament. *J Bone Joint Surg Am.* 1983;65(2):184–192.
 47. Noyes FR, Matthews DS, Mooar PA, Grood ES. The symptomatic anterior cruciate-deficient knee. Part II: the results of rehabilitation, activity modification, and counseling on functional disability. *J Bone Joint Surg Am.* 1983;65(2):163–174.
 48. Kostogiannis I, Ageberg E, Neuman P, Dahlberg L, Friden T, Roos H. Activity level and subjective knee function 15 years after anterior cruciate ligament injury: a prospective, longitudinal study of nonreconstructed patients. *Am J Sports Med.* 2007;35(7):1135–1143.
 49. Marks PH, Donaldson ML. Inflammatory cytokine profiles associated with chondral damage in the anterior cruciate ligament-deficient knee. *Arthroscopy.* 2005;21(11):1342–1347.
 50. Roos H, Adalberth T, Dahlberg L, Lohmander LS. Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. *Osteoarthritis Cartilage.* 1995;3(4):261–267.
 51. Noyes FR, Mangine RE, Barber S. Early knee motion after open and arthroscopic anterior cruciate ligament reconstruction. *Am J Sports Med.* 1987;15(2):149–160.
 52. Beynon BD, Uh BS, Johnson RJ, et al. Rehabilitation after anterior cruciate ligament reconstruction: a prospective, randomized, double-blind comparison of programs administered over 2 different time intervals. *Am J Sports Med.* 2005;33(3):347–359.
 53. Andersson C, Odensten M, Good L, Gillquist J. Surgical or non-surgical treatment of acute rupture of the anterior cruciate ligament: a randomized study with long-term follow-up. *J Bone Joint Surg Am.* 1989;71(7):965–974.
 54. Daniel DM, Stone ML, Dobson BE, Fithian DC, Rossman DJ, Kaufman KR. Fate of the ACL-injured patient: a prospective outcome study. *Am J Sports Med.* 1994;22(5):632–644.
 55. O'Brien WR. Degenerative arthritis of the knee following anterior cruciate ligament injury: role of the meniscus. *Sports Med Arthrosc Rev.* 1993;1(2):114–118.
 56. Zysk SP, Refior HJ. Operative or conservative treatment of the acutely torn anterior cruciate ligament in middle-aged patients: a follow-up study of 133 patients between the ages of 40 and 59 years. *Arch Orthop Trauma Surg.* 2000;120(1–2):59–64.
 57. The Cochrane Collaboration Review Group on HIV Infection and AIDS. Editorial policy: inclusion and appraisal of experimental and non-experimental (observational) studies. <http://www.igh.org/Cochrane2009>. Accessed June 2014.
 58. Hootman JM, Driban JB, Sitler MR, Harris KP, Cattano NM. Reliability and validity of three quality rating instruments for systematic reviews of observational studies. *Res Synth Methods.* 2011;2(2):110–118.
 59. Beynon BD, Johnson RJ, Naud S, et al. Accelerated versus nonaccelerated rehabilitation after anterior cruciate ligament reconstruction: a prospective, randomized, double-blind investigation evaluating knee joint laxity using roentgen stereophotogrammetric analysis. *Am J Sports Med.* 2011;39(12):2536–2548.
 60. Bowers AL, Spindler KP, McCarty EC, Arrigain S. Height, weight, and BMI predict intra-articular injuries observed during ACL reconstruction: evaluation of 456 cases from a prospective ACL database. *Clin J Sport Med.* 2005;15(1):9–13.
 61. Li RT, Lorenz S, Xu Y, Harner CD, Fu FH, Irrgang JJ. Predictors of radiographic knee osteoarthritis after anterior cruciate ligament reconstruction. *Am J Sports Med.* 2011;39(12):2595–2603.
 62. Portney LG, Watkins MP. *Foundations of Clinical Research: Applications to Practice.* Norwalk, CT: Appleton & Lange; 1993.
 63. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthritis. *Ann Rheum Dis.* 1957;16(4):494–502.
 64. Jordan JM, Helmick CG, Renner JB, et al. Prevalence of knee symptoms and radiographic and symptomatic knee osteoarthritis in African Americans and Caucasians: the Johnston County Osteoarthritis Project. *J Rheumatol.* 2007;34(1):172–180.
 65. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W, Meenan RF. The prevalence of knee osteoarthritis in the elderly: the Framingham Osteoarthritis Study. *Arthritis Rheum.* 1987;30(8): 914–918.
 66. von Porat A, Henriksson M, Holmström E, Roos EM. Knee kinematics and kinetics in former soccer players with a 16-year-old ACL injury: the effects of twelve weeks of knee-specific training. *BMC Musculoskelet Disord.* 2007;8:35.
 67. McErlain DD, Appleton CT, Litchfield RB, et al. Study of subchondral bone adaptations in a rodent surgical model of OA using in vivo micro-computed tomography. *Osteoarthritis Cartilage.* 2008;16(4):458–469.
 68. Lohmander LS, Atley LM, Pietka TA, Eyre DR. The release of crosslinked peptides from type II collagen into human synovial fluid is increased soon after joint injury and in osteoarthritis. *Arthritis Rheum.* 2003;48(11):3130–3139.
 69. Cameron ML, Fu FH, Paessler HH, Schneider M, Evans CH. Synovial fluid cytokine concentrations as possible prognostic indicators in the ACL-deficient knee. *Knee Surg Sports Traumatol Arthrosc.* 1994;2(1):38–44.

70. Andriacchi TP, Mundermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. *Ann Biomed Eng.* 2004;32(3):447–457.
71. Jarvela T, Paakkala T, Kannus P, Jarvinen M. The incidence of patellofemoral osteoarthritis and associated findings 7 years after anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft. *Am J Sports Med.* 2001;29(1):18–24.
72. Pearle AD, Warren RF, Rodeo SA. Basic science of articular cartilage and osteoarthritis. *Clin Sports Med.* 2005;24(1):1–12.
73. Shefelbine SJ, Ma CB, Lee KY, et al. MRI analysis of in vivo meniscal and tibiofemoral kinematics in ACL-deficient and normal knees. *J Orthop Res.* 2006;24(6):1208–1217.
74. Timoney JM, Inman WS, Quesada PM, et al. Return of normal gait patterns after anterior cruciate ligament reconstruction. *Am J Sports Med.* 1993;21(6):887–889.
75. Kinds MB, Vincken KL, Hoppinga TN, et al. Influence of variation in semiflexed knee positioning during image acquisition on separate quantitative radiographic parameters of osteoarthritis, measured by Knee Images Digital Analysis. *Osteoarthritis Cartilage.* 2012;20(9):997–1003.
76. Marcacci M, Zaffagnini S, Giordano G, Iacono F, Presti ML. Anterior cruciate ligament reconstruction associated with extra-articular tenodesis: a prospective clinical and radiographic evaluation with 10- to 13-year follow-up. *Am J Sports Med.* 2009;37(4):707–714.
77. Shelbourne KD, Gray T. Anterior cruciate ligament reconstruction with autogenous patellar tendon graft followed by accelerated rehabilitation: a two- to nine-year follow-up. *Am J Sports Med.* 1997;25(6):786–795.
78. Hunter DJ. Imaging insights on the epidemiology and pathophysiology of osteoarthritis. *Rheum Dis Clin North Am.* 2009;35(3):447–463.
79. Kessler MA, Stutz G, Behrend H, Rukavina A, Kuster M. Is ACL reconstruction necessary to prevent osteoarthritis? 12 year follow up results after non-operative treatment of ACL rupture [abstract]. *Br J Sports Med.* 2005;39(6):393.
80. Lynch MA, Henning CE, Glick KR. Knee joint surface changes: long-term follow-up meniscus tear treatment in stable anterior cruciate ligament reconstructions. *Clin Orthop Relat Res.* 1983;172:148–153.
81. Walla DJ, Albright JP, McAuley E, Martin RK, Eldridge V, El-Khoury G. Hamstring control and the unstable anterior cruciate ligament-deficient knee. *Am J Sports Med.* 1985;13(1):34–39.
82. Englund M, Guermazi A, Lohmander LS. The meniscus in knee osteoarthritis. *Rheum Dis Clin North Am.* 2009;35(3):579–590.
83. Scarvell JM, Smith PN, Refshauge KM, Galloway H, Woods K. Comparison of kinematics in the healthy and ACL injured knee using MRI. *J Biomech.* 2005;38(2):255–262.
84. Snyder-Mackler L, Fitzgerald GK, Bartolozzi AR, Ciccotti MG. The relationship between passive joint laxity and functional outcome after anterior cruciate ligament injury. *Am J Sports Med.* 1997;25(2):191–195.
85. Rudolph KS, Eastlack ME, Axe MJ, Snyder-Mackler L. 1998 Basmajian Student Award Paper: Movement patterns after anterior cruciate ligament injury: a comparison of patients who compensate well for the injury and those who require operative stabilization. *J Electromyogr Kinesiol.* 1998;8(6):349–362.
86. Leys T, Salmon L, Waller A, Linklater J, Pinczewski L. Clinical results and risk factors for reinjury 15 years after anterior cruciate ligament reconstruction: a prospective study of hamstring and patellar tendon grafts. *Am J Sports Med.* 2012;40(3):595–605.
87. Sajovic M, Strahovnik A, Dernovsek MZ, Skaza K. Quality of life and clinical outcome comparison of semitendinosus and gracilis tendon versus patellar tendon autografts for anterior cruciate ligament reconstruction: an 11-year follow-up of a randomized controlled trial. *Am J Sports Med.* 2011;39(10):2161–2169.
88. Spector TD, Cooper C. Radiographic assessment of osteoarthritis in population studies: whither Kellgren and Lawrence? *Osteoarthritis Cartilage.* 1993;1(4):203–206.
89. Altman R, Asch E, Bloch D, et al. Development of criteria for the classification and reporting of osteoarthritis: classification of osteoarthritis of the knee. Diagnostic and Therapeutic Criteria Committee of the American Rheumatism Association. *Arthritis Rheum.* 1986;29(8):1039–1049.
90. Schipf D, de Klerk BM, Kerkhof HJ, et al. Impact of different descriptions of the Kellgren and Lawrence classification criteria on the diagnosis of knee osteoarthritis. *Ann Rheum Dis.* 2011;70(8):1422–1427.
91. Frobell RB, Le Graverand MP, Buck R, et al. The acutely ACL injured knee assessed by MRI: changes in joint fluid, bone marrow lesions, and cartilage during the first year. *Osteoarthritis Cartilage.* 2009;17(2):161–167.
92. Frobell RB, Roos EM, Roos HP, Ranstam J, Lohmander LS. A randomized trial of treatment for acute anterior cruciate ligament tears. *N Engl J Med.* 2010;363(4):331–342.
93. Moher D, Liberati A, Tetzlaff J, Altman DG, PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *J Clin Epidemiol.* 2009;62(10):1006–1012.

Address correspondence to Kyle P. Harris, MS, ATC, Department of Health, Physical Education and Nursing, Bucks County Community College, 275 Swamp Road, Newtown, PA 18940. Address e-mail to kpfh85@gmail.com.

Appendix. Newcastle-Ottawa Scale Scores

Scale Item	Lohmander et al ²³ (2004)	Kessler et al ⁴¹ (2008)	von Porat et al ³² (2004)	Meuffel et al ²⁵ (2009)
Selection (maximum = 1 point)				
1. Representativeness of the general ACL-deficient population	0	0	0	0
(a) Truly representative of the general ACL-deficient population				
(b) Somewhat representative of the general ACL-deficient population				
(c) Selected group of users (eg, nurses, volunteers)				
(d) No description of the derivation of the cohort				
2. Selection of the nonexposed cohort (nonsurgical) (maximum = 1 point)	0	0	0	0
(a) Drawn from the general ACL-deficient population				
(b) Drawn from a different source				
(c) No description				
3. Ascertainment of exposure (surgical) (maximum = 1 point)	1	1	1	1
(a) Secure records (eg, surgical records)				
(b) Structured interview				
(c) Written self-report				
(d) No description				
4. Ascertainment of nonexposure (nonsurgical) (maximum = 1 point)	1	1	1	1
(a) Secure records (eg, surgical records)				
(b) Structured interview				
(c) Written self-report				
(d) No description				
5. Demonstration that the outcome of interest was not present at start of study	0	0	0	0
(a) Yes				
(b) No				
Comparability (maximum = 2 points)				
1. Comparability of the cohorts on the basis of design or analysis	0	1	2	1
(a) Study controls for secondary injury (ie, meniscal, ligamentous other than ACL)				
(b) Study controls for body mass index				
Outcome (maximum = 1 point each for items 1–3)				
1. Assessment of outcome	1	1	0	1
(a) Independent blind assessment				
(b) Record linkage				
(c) Self-report				
(d) No description				
2. Was follow-up long enough for outcomes to occur?	1	1	0	1
(a) Yes (10 y or more)				
(b) No				
3. Adequacy of cohort follow-up	0	0	1	1
(a) Complete follow-up: all participants accounted for				
(b) Participants lost to follow-up unlikely to bias (small number lost or >80% follow-up or description provided of those lost)				
(c) Follow-up rate <80% and no description of those lost				
(d) No statement				
Overall consensus score (out of possible 10)	4	5	5	6
Time to read and score each article, average min	9	11	9	9

Abbreviation: ACL, anterior cruciate ligament.