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The Role of Athletic Trainers in Preventing and Managing Posttraumatic Osteoarthritis in Physically Active Populations: a Consensus Statement of the Athletic Trainers' Osteoarthritis Consortium[a]

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Objective: To provide athletic trainers with a fundamental understanding of the pathogenesis and risk factors associated with the development of posttraumatic osteoarthritis (PTOA) as well as the best current recommendations for preventing and managing this condition.

Background: Posttraumatic osteoarthritis, or osteoarthritis that develops secondary to joint injury, accounts for approximately 5.5 million US cases annually. A young athlete with a joint injury is at high risk for PTOA before the age of 40, which could lead to the patient living more than half of his or her life with a painful and disabling disorder. Given our frequent contact with physically active people who often sustain traumatic joint injuries, athletic trainers are in a unique position to help prevent and manage PTOA. We can, therefore, regularly monitor joint health in at-risk patients and implement early therapies as necessary.

Recommendations: The recommendations for preventing and managing PTOA are based on the best available evidence. Primary injury prevention, self-management strategies, maintenance of a healthy body weight, and an appropriate level of physical activity should be encouraged among those at risk for PTOA after acute traumatic joint injury. Education of athletic trainers and patients regarding PTOA is also critical for effective prevention and management of this disease.

Key Words: joint injuries, injury prevention, best practices

Osteoarthritis (OA) is a chronic disorder that affects movable joints, compromising the articular cartilage, underlying subchondral bone, and surrounding soft tissues. The condition affects more than 27 million Americans[1] and directly costs more than $185 billion annually,[2] making it a substantial public health burden. Associated with several chronic comorbidities,[3] such as obesity, diabetes, and cardiovascular disease, OA ranks 11th among all forms of disability worldwide.[4] A large portion of the working population is affected by this debilitating musculoskeletal condition. Surgical and nonsurgical interventions are used to manage the symptoms of OA and treat the end stage of the disease. Unfortunately, no cure currently exists for OA, and no accepted disease-modifying treatments have been shown to effectively prevent OA or alter the clinical course of disease progression in an afflicted joint.

[a] The views expressed in this article by the Athletic Trainers' Osteoarthritis Consortium (ATOAC) do not necessarily reflect the views of the National Athletic Trainers' Association.

In the past, OA was considered a disease that affects only the elderly; however, increasing evidence demonstrates that young and middle-aged adults are suffering from OA as well.[5–7] For example, more than half of adults with symptomatic knee OA are younger than 65 years old.[8] Adults with hip or knee OA who are 20 to 55 years old are 4 times more likely to be highly psychologically distressed; furthermore, 67% report OA-related work disability, and women 40 to 49 years old report a reduction in the quality of life.[9] The early onset of OA in younger populations is often attributable to a history of acute traumatic joint injury. In fact, a history of acute traumatic joint injury is a strong predictor of OA risk.[10] This unique type of early-onset OA, which occurs after joint injury, is commonly referred to as *posttraumatic OA* (PTOA). Well-designed, prospective cohort studies indicated that individuals with a history of joint trauma are 3 to 6 times more likely to develop knee OA[11,12] and were diagnosed approximately 10 years earlier than those without any history of injury.[13] For some patients, this may mean living more than half their lives with a chronic and disabling disorder. Although PTOA has been estimated to account for 12% of all OA cases in the

Table 1. Prevention Levels and Definitions

Levels of Prevention	Definition
Primary prevention	Interventions are designed to prevent an injury or disease condition from occurring in the first place. The focus is generally on policies, practices, and behaviors that mitigate risk.
Secondary prevention	Initiatives attempt to recognize or identify an injury or disease at its earliest stage, so that prompt and appropriate management can be implemented to mitigate the secondary effects of the injury or disease and restore function. Successful secondary prevention reduces the effect of the disease in the short term and perhaps also in the long term. The focus is generally on emergency management and initial medical care.
Tertiary prevention	Initiatives focus on reducing or minimizing the long-term consequences of an injury or disease once it has occurred. The goal is to eliminate or delay the onset of complications, morbidity, and long-term disability due to the injury or disease. Most medical interventions fall into this category. The focus is generally on chronic management and health-behavior change.

general population (approximately 5.6 million US cases annually),[13] the incidence and prevalence of PTOA in young athletes and active populations remain unclear; the values are likely much higher in these populations that are at increased risk for joint injury.

Athletic trainers (ATs) are in a unique position to have substantial effects on primary, secondary, and tertiary OA prevention (Table 1). We can promote *primary prevention* (ie, preventing OA from occurring) by encouraging the sports community to adopt injury-prevention programs and promoting physical activity in children to help reduce the risk of obesity. After an injury, ATs can play a critical role in *secondary prevention* of OA, which involves recognizing that the patient may have an early stage of OA and initiating care to reduce the effects of the disease. Finally, if a physically active patient has symptomatic OA, ATs can be crucial health care providers in *tertiary prevention*, which focuses on minimizing the pain, disability, and other consequences of OA. The Athletic Trainers' Osteoarthritis Consortium (ATOAC) contends that ATs must have an up-to-date understanding of OA and be armed with evidence-based strategies to aid in preventing or managing this chronic and often debilitating disorder. Much is still unknown about the initiation, development, prevention, and treatment of OA, but our goal in publishing this review is to provide the current state of the literature regarding the pathophysiology, risk factors, and possible prevention and management strategies for OA.

RECOMMENDATIONS

The ATOAC suggests the following recommendations for ATs to assist with the prevention and management of OA in physically active populations.

1. Athletic training students and ATs should be educated, through professional education and continuing education programs, regarding common risk factors for OA and the current strategies to prevent and manage OA in the physically active populations frequently treated by ATs.
2. Athletic trainers should support and implement evidence-based primary injury-prevention interventions to reduce the risk of acute traumatic joint injuries, which will in turn reduce the risk of OA.
3. Athletic trainers should educate patients with joint injuries about their increased risk of OA, other common risk factors for OA, self-management strategies to minimize the burden of OA, and strategies to regularly monitor changes in joint health.
4. Athletic trainers should be knowledgeable about and apply existing guidelines and recommendations for managing OA among physically active individuals.[14]
5. Athletic trainers should support national public health policies related to OA, particularly recommendations to treat individuals with a history of joint injury as a high-risk patient population for OA using a chronic disease-management model.[15,16]
6. Athletic trainers should encourage athletes to maintain a physically active lifestyle once their participation in competitive sports has ceased in order to reduce the risk of obesity and promote long-term health and wellness.
7. Athletic trainers should encourage, engage in, and conduct well-designed research examining risk factors and management strategies for PTOA, as well as investigations to examine the best approaches to identify and monitor disease progression. A recent review by Emery et al[17] provides strong methodologic recommendations for persons interested in undertaking research in this field.

This review and the following recommendations received full support from the ATOAC Consensus Task Force and Executive Committee, as well as support from 71% (39 of 55 members; 16 members did not vote) of the ATOAC membership.

PATHOGENESIS OF PTOA

The pathologic changes in a joint with PTOA include varying degrees of structural changes in the joint tissues: (1) fibrillation or thinning of articular cartilage, (2) infiltration of the subchondral bone into the articular cartilage, (3) tearing or fibrillation of the menisci and intra-articular ligaments, (4) synovitis, (5) joint capsule fibrosis and thickening, and (6) osteophytes at the joint margins.[18] The pathogenic cascade of events that leads to these structural changes is not well understood. A common characteristic of PTOA is the initial mechanical insult to the joint that results in damage to the articular structures. The intensity of the impact as well as the severity of tissue damage likely affect the onset and progression of PTOA. High-intensity impacts may result in displaced fractures of the articular surface that extend through the cartilage and bone (subchondral and trabecular), whereas low-intensity impacts may result in damage to the chondrocytes and extracellular matrix, macroscopic disruption of the articular cartilage without displaced bony fracture, and traumatic bone marrow lesions.[19] Both high-intensity and low-intensity impacts, therefore, disrupt the articular structures and can lead to PTOA; however, the progressions to PTOA

after these injuries can be vastly different. For example, patients with tibial plafond fractures resulting from low-intensity impacts had minimal risk for the rapid onset (within 2 years) of PTOA, yet patients whose tibial plafond fractures occurred via high-intensity impacts developed PTOA rapidly (within 2 years).[20] Similarly, military service members with high-intensity battlefield trauma to the joint also developed PTOA within 2 years of injury.[21] It should be noted that highly repetitive impact loading of a joint can damage the articular cartilage in the absence of an acute traumatic injury. For example, in animal studies, repetitive running or grasping resulted in inflammation, greater collagen degeneration, decreased lubricin (a glycoprotein that protects against cartilage wear), and decreased glycosaminoglycans (components of the extracellular matrix of cartilage that contribute to lubrication and shock absorption), all of which can be precursors to the development of OA.[22–24] Hence, similar to many other tissues in the body, a joint may be injured by a high-impact single event or excessive repetitive overloading.

Mounting evidence supports the current hypothesis that PTOA pathogenesis may reflect multiple events or processes, which may occur at the time of injury or develop within the joint after injury, including (1) structural changes to joint tissues, (2) molecular changes, and (3) neuromuscular and biomechanical alterations. The traumatic injury causes structural damage to multiple joint tissues, such as the ligaments, bone, synovium, and articular cartilage. Direct trauma to the articular cartilage resulting from impact is common and can cause chondrocyte death in both the impacted and surrounding areas.[25] Subchondral bone injury, as indicated by traumatic bone marrow lesions, is also frequent with joint injury.[26] For example, more than 80% of patients with anterior cruciate ligament (ACL) injuries present with lesions to the lateral femoral condyle or tibial plateau.[27,28] Geographic traumatic bone marrow lesions (characterized as bordering cortical bone and disrupting the overlying articular surface) are associated with osteocyte necrosis, proteoglycan loss, chondrocyte death, and extracellular matrix degradation.[29,30] Therefore, a joint injury can have immediate effects throughout the joint.

After an injury, numerous inflammatory mediators (eg, interleukin-6 and tumor necrosis factor-α) are released initially and alter joint homeostasis.[31] Several inflammatory mediators are capable of inducing articular cartilage damage[32,33] and changing the quality of the synovial fluid, which is critical for lubricating the articular cartilage.[34] Within a few days after an ACL injury, assays can often detect biomarkers that reflect proteoglycans (eg, matrix of matrix metalloproteinases 1 and 3, aggrecan fragments) and glycosaminoglycans (chondroitin sulfate epitope [WF6]) degradation; these substances make up the cartilage extracellular matrix and allow the cartilage to withstand compressive loads.[35]

Early changes in bone shape have also been identified after injury and may contribute to PTOA pathogenesis. Within 3 months after ACL injury, for example, the femoral condyles become less convex, and the tibial plateaus become more concave.[36] In adults at high risk for symptomatic knee OA, including those with previous joint injury, subtle alterations in joint shape were linked to the development of radiographic OA.[37]

Although these structural and molecular changes occur in the joint, biomechanical changes are also taking place and may perpetuate future joint damage and cartilage breakdown. For example, rotational knee kinematics are altered after ACL injury[38] and remain affected after reconstruction.[39] This kinematic change may lead to altered joint tissue loading, whereby areas of cartilage not conditioned to receive loads are now experiencing loads, ultimately resulting in cartilage degeneration.[40] Given that normal function of the synovial joints requires extensive cooperation among the various tissues, changes in biological, chemical, and biomechanical processes could promote a vicious cycle of continuous joint damage, resulting in progressive joint degradation and ultimately PTOA. In-depth discussions of PTOA pathogenesis have been published elsewhere,[41–43] and we refer the reader to these articles for a more detailed description. These early changes highlight that the foundation for PTOA is often laid while a physically active individual with a joint injury is under the care of an AT. Understanding the pathogenesis of PTOA may lead to new strategies to predict who will develop PTOA and to identify novel therapies to prevent or delay the onset of PTOA.

RISK FACTORS FOR PTOA

A *risk factor* is any attribute, characteristic, or exposure of an individual that increases the likelihood of developing a disease or injury[44]; specific to this review, a risk factor is any attribute of a person that increases the probability of developing PTOA. We will provide an overview of potential risk factors for PTOA that have at least some evidence linking each risk factor to the postinjury development of OA.

Joint Injury (Location, Type, Severity, and Recurrence)

Location. Although joint injury may increase the risk of PTOA, a joint-specific response to injury may predispose a particular joint to PTOA more than other joints.[45] For example, about 12% of knee[10] and 8% of hip[46] OA are reported to be posttraumatic in nature, whereas 70% to 80% of diagnosed OA at the ankle is considered posttraumatic.[47,48] Based on these data, it would seem the ankle may be more susceptible to PTOA than other lower extremity joints. Why injury may lead to different incidence rates of PTOA across joints remains unknown, but potential explanations could include postinjury biomechanical alterations that are joint specific and different responses of the tissues within each joint to injurious insult (eg, dissipates energy differently, different rates of cell death).[45]

Type. The structures affected can influence the risk for PTOA. In a case-control study[49] of 32 patients who developed radiographic signs of OA during a 2-year period, the authors noted that people who developed OA were more likely to have medial meniscal extrusion (odds ratio [OR] = 3.03) or tears with larger radial involvement (OR = 5.92) at baseline compared with 64 adults without OA. Other types of meniscal injuries (eg, horizontal tear, longitudinal tear) were not associated with incident OA.[49] Patients who sustain fractures also have an increased risk of PTOA. Of 73 patients who received operative treatment for

tibial plateau fractures, 68% had developed radiographic evidence OA in the affected knee and 32% in the contralateral knee at approximately 4.5 years after surgery.[50] Similarly, in a group of 65 patients who were treated surgically for intra-articular fractures of the distal femur, 36% went on to develop PTOA at 3.8 years postoperatively.[51] In contrast, fewer than 20% of patients with tibial spine fractures who were treated with open reduction and internal fixation had developed OA 5 years after surgery.[52]

Severity. The number of concomitant injuries and the degree of ligament injury can also increase PTOA risk. Patients with an isolated ACL injury are less likely to develop OA than those with concomitant injury. Oiestad et al[53] prospectively evaluated patients 10 to 15 years after ACL reconstruction (ACLR) and found joint-space narrowing (a marker of OA) in 80% of patients who presented with accompanying chondral or meniscal damage, whereas only 62% of patients with isolated injuries displayed narrowing. Similar results have been shown for knee medial collateral ligament (MCL) sprains: 45% of knees with an MCL sprain accompanied by a meniscal or ACL tear had radiographic signs of OA 10 years after injury, whereas 0% of knees with an isolated MCL sprain showed such signs.[54] In addition, the degree of ligament rupture seems to play a role in the development of PTOA. Results from several studies[55–57] illustrate that patients who sustained grade II MCL sprains were less likely to demonstrate radiographic evidence of PTOA (0% to 13% of knees) at 9 to 10 years postinjury than patients who experienced grade III MCL sprains (63% to 68% of knees).

Injury Recurrence. Many studies show a high incidence (up to 89%) of glenohumeral OA after repetitive shoulder dislocations (treated both operatively [with various procedures] and nonoperatively).[58–61] Moderate or severe radiographic evidence of OA was present in 29% of patients with recurrent shoulder dislocations but only 17% of patients with 1-time dislocations 25 years after initial injury.[62] Similarly, Vollnberg et al[63] found that patients who had incurred recurrent (2–9) or chronic (10 or more) patellar dislocations were more likely to have patellofemoral OA than those patients with a single dislocation.

Excess Body Weight

Obesity (body mass index [BMI] >30; calculated as kg/m^2) is an established and robust risk factor for knee OA. Results from a recent meta-analysis[64] showed that individuals who were overweight (BMI = 25.0–29.9) were 2.5 times more likely to develop knee OA and those who were obese were 4.6 times more likely to develop knee OA. Further, the risk of knee OA increased by 35% with each increase of 5 in BMI. Being overweight or obese is also a risk factor for hip[46] and hand[65] OA. However, fewer data are available examining body weight as a risk factor for PTOA. Being overweight or obese at the time of surgical repair for an ankle fracture increases the risk of developing moderate to severe (Kellgren and Lawrence grade 3–4) PTOA; those patients who were overweight at the time of surgery were 1.9 times more likely to develop OA and those who were obese were 2.8 times more likely to develop PTOA.[66] In a mouse model of PTOA, obesity increased the severity of OA associated with a traumatic knee fracture.[67] Although a weight gain greater than 15 lb (6.8 kg) within the first 5 years after ACLR was not directly linked to radiographic or cartilage-measured changes in joint structure, Spindler et al[68] observed that it was a predictor of poor patient-perceived outcomes.

Range-of-Motion Deficits

Range-of-motion (ROM) loss after injury is commonplace. More often than not, restoration of ROM is considered complete with appropriate rehabilitation, and as such, ROM is not often studied as a potential risk factor for PTOA. Yet based on the limited available evidence, it would appear that ROM deficits may contribute to PTOA development after ACLR. Roe et al[69] found that flexion contractures were associated with early evidence of PTOA at approximately 7 years after ACLR. Shelbourne and Gray[70] showed that patients with less than normal ROM after ACLR were 2.5 times more likely to have radiographic evidence of PTOA at approximately 10 years than patients with normal ROM. In a subsequent study, Shelbourne et al[71] noted that limited knee-flexion ROM at return to play increased the risk for radiographic evidence of OA.

Range-of-motion deficits subsequent to injuries other than ACL tears have yet to be linked to PTOA. For instance, patients with chronic ankle instability often present clinically with deficits in dorsiflexion ROM.[72,73] These ROM deficits have been linked to altered landing kinematics,[74] but their involvement or lack thereof in the development of PTOA secondary to ankle injury has not been systematically investigated.

Strength Deficits

Muscle weakness is a common consequence of many joint injuries. In particular, quadriceps muscle weakness, which often accompanies ACL injuries,[75,76] is thought to lead to impaired dampening and shock-attenuation abilities, resulting in greater loads being transmitted to the knee-joint structures; therefore, it has been suggested as a risk factor for the development of PTOA.[77–79] Available evidence indeed supports the premise that quadriceps weakness is a risk factor for PTOA. Keays et al[80] demonstrated that 6 years after ACLR, weak quadriceps muscles and low quadriceps-to-hamstrings strength ratios measured at 60°/s trended toward discriminating between participants without OA (Kellgren and Lawrence grade 0 or 1) and those with OA (Kellgren and Lawrence grade 2 or greater). Using a prospective study design, Tourville et al[81] noted that quadriceps strength loss measured within a few months after ACL injury was associated with joint-space narrowing by 4 years after ACLR. Oiested et al[79] found an association between quadriceps strength loss that occurred 2 to 15 years after ACLR and symptomatic knee OA (Kellgren and Lawrence grade 2) but were unable to establish an association between weakness soon after ACL injury and radiographic OA. These data are not conclusive, but they suggest that quadriceps weakness after ACL injury may be associated with the development of PTOA. More research is necessary to determine if muscle weakness that manifests after other joint injuries (eg, ankle sprains, shoulder sprains, and dislocations) contributes to the risk of PTOA.

Static Alignment

Joint malalignment has been established as a risk factor for knee OA. In 2 large prospective cohort studies,[82,83] varus knee alignment was associated with 1.5-fold and 2-fold increased risks of knee OA. Although the data linking malalignment to PTOA are not as strong, the current research suggests that malalignment may be a risk factor. In a retrospective study,[50] patients who sustained a tibial plateau fracture and displayed a valgus malalignment of 5° or greater were more likely to develop moderate to severe PTOA (Kellgren and Lawrence grade 3–4). Similarly, varus malalignment of the contralateral, uninjured knee of patients who sustained an ACL injury approximately 15 years earlier predicted the risk of OA in the ACL-injured knee (OR = 3.9; 95% confidence interval = 1.0, 15.8).[84]

Static malalignment is also a common indication for a high tibial osteotomy, which is often performed for a varus-aligned knee with isolated medial compartment OA (PTOA or primary OA). The surgery reduces the load on the medial knee compartment by shifting the mechanical axis to the lateral compartment.[85] The goals of this procedure are to reduce symptoms and potentially prevent further overload of the medial compartment in an attempt to alter or slow disease progression. Pain and function improve with high tibial osteotomy,[82] yet no evidence shows a beneficial effect in slowing disease progression. Studies are needed to examine disease-related outcomes of high tibial osteotomy and nonoperative treatment to better understand the effectiveness of the surgery.

Physical Activity and Sports Participation

The health benefits associated with physical activity strongly support the importance of a physically active lifestyle in children and adults. Physical activity may be beneficial to joints because it encourages strength, endurance, neuromuscular control, and weight management; however, certain high-level physical activities may expose joints to repetitive overloading and a greater risk of injury or osteoarthritis. In a large prospective study, more than 5000 individuals (minimum age = 20 years, 2/3 of the study population was between 40 and 60 years old) were surveyed regarding physical activity (frequency, duration, intensity, and type) and then radiographs were taken 4, 9, and 13 years after the survey.[86] No association was evident between OA development and running, walking, or physical activity. Hansen et al[87] compiled the evidence related to running and OA and were unable to support an association between short-distance to moderate-distance running and OA; however, the evidence was inconclusive regarding high-volume running (>20 miles [32 km] per week). There may indeed be a dose-response relationship between physical activity and OA (ie, high-level physical activity is more risky and low-level activity is safe). Michaelsson et al[88] found that cross-country skiers participating in 5 or more high-level races had a 70% higher rate of OA than skiers who completed only a single race. Further, skilled skiers (ie, those with faster racing times) were also more likely to develop OA.

Not only is low to moderate physical activity safe for joints but it may also protect a joint from OA.[89] For example, over a 2-year period, Wijayaratne et al[90] observed that retropatellar cartilage volume was less in sedentary women than in physically active women. Similarly, Mosher et al[91] demonstrated increased femoral cartilage volume in marathon runners compared with nonrunners.

With regard to specific sports, Driban et al[92] quantitatively analyzed 17 studies and found that the prevalence of knee OA in former sport participants (n = 3759) was 7.7%, compared with 7.3% among nonexposed control participants (n = 4730, OR = 0.9). Athletes who were involved in certain sports were more likely to have knee OA: competitive weight lifting (OR = 6.9), wrestling (OR = 3.8), soccer (OR = 3.5), or elite-level long-distance running (OR = 3.3), suggesting a 3 to 7 times higher prevalence of knee OA in these sports. It should be noted that the authors were unable to control for a previous history of joint injury and, thus, we do not know if the higher OA rates were due to higher rates of joint injury or the result of participation in the activities themselves. However, in this same systematic review, the authors were able to control for joint injury in a smaller subset (4 studies) and found similar results. Uninjured participants in elite-level (soccer or orienteering) and nonelite-level (soccer or American football) sports had a higher prevalence of knee OA (OR = 9.46 and 3.75, respectively) than nonexposed control participants. Supporting the contention that an interaction exists between competition level and injury and the risk for PTOA is work by Roos et al,[93] who compared elite and nonelite soccer players with or without a history of injury. The prevalence of knee OA was highest in injured elite-level soccer players (33.3%) and lowest in uninjured control participants (1.3%) and nonelite soccer players (2.7%).

The level of physical activity after acute traumatic joint injury could play a role in the initiation and progression of PTOA, yet conflicting evidence makes it difficult to establish a clear association. Among a young and physically active population, individuals with a history of knee-joint injury reported lower scores on the Knee Injury and Osteoarthritis Outcome Score (KOOS)[94,95] and Western Ontario and McMaster University Osteoarthritis Index (WOMAC)[96] scales, indicating more symptoms and impaired function, despite reporting higher levels of physical activity (as measured by the Marx Activity Rating Scale) than uninjured control participants.[97,98] Similar results were recently reported in collegiate athletes.[99] If symptoms persist, structural alterations are present, or physical function is not regained after joint injury, returning to a high level of physical activity could exacerbate symptoms and prove detrimental to long-term joint health. Therefore, low-impact activities may be an appropriate alternative. Further research is needed to determine when it is safe to return to physical activity after joint injury and whether certain types of physical activity (eg, excessive loading, cutting) are associated with the initiation and progression of PTOA after joint injury.

Neuromuscular Deficits

Neuromuscular deficits, such as an inability to control muscle force or activate a muscle completely, could be related to the onset of PTOA; however, little evidence is available to verify this hypothesis. After ACLR, patients who performed a single-legged landing had greater hamstrings coactivation and higher tibiofemoral compressive forces than uninjured, control participants.[100] Similar-

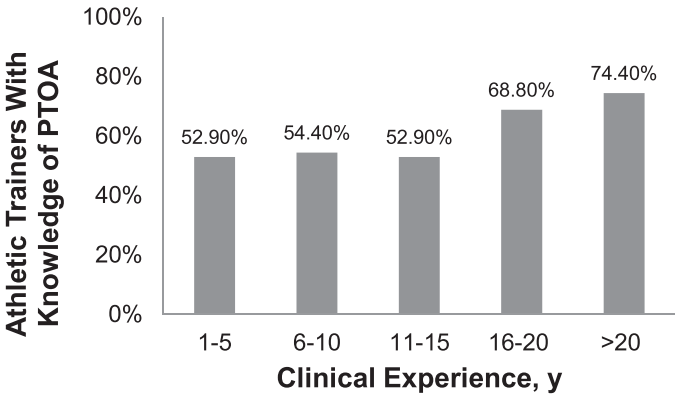

Figure 1. Athletic trainers' knowledge of posttraumatic osteoarthritis (PTOA) by years of clinical experience.[104]

ly, when patients who had undergone ACLR were asked to complete a force-matching task approximately 18 months after their surgery, they displayed less accurate quadriceps force control and more quadriceps and hamstrings coactivation than healthy individuals.[101] Aberrant loading at the knee joint (eg, overloading or underloading) resulting from altered muscle activation or diminished quadriceps control after ACLR could influence joint-loading patterns and lead to joint degeneration. In the same way, modified knee-joint kinematics, such as altered knee flexion and rotation, may result in greater loads on previously unloaded regions of articular cartilage and lead to the biological changes that result in PTOA.[40,102]

Arthrogenic muscle inhibition, an inability to completely activate a muscle voluntarily, is another neuromuscular deficiency that could play a role in PTOA.[78] Arthrogenic inhibition affects a muscle's ability to contract even in the absence of direct trauma to that muscle. This occurs commonly in the quadriceps after ACL injury or reconstruction. The inhibition is due to diminished motoneuron activity and may contribute to the lingering strength deficits that have been noted after ACLR and that have been linked to PTOA.

Although the aforementioned neuromuscular deficits and alterations have been identified after joint injury, a direct link between them and the development of PTOA has yet to be shown. Future research is necessary to support or refute the hypothesis that altered neuromuscular control after joint injury contributes to the onset of PTOA.

ATHLETIC TRAINERS' AWARENESS OF PTOA

One practice domain of athletic training is injury/illness prevention and wellness protection,[103] and ATs generally take pride in their ability to keep patients safe and minimize injury during physical activity. Historically, the profession has been focused on primary injury prevention, acute treatment, rehabilitation, and return to play; less attention has been given to secondary and tertiary prevention and longer-term outcomes that may result from joint injury. Athletic trainers need to be aware of the association between acute traumatic joint injury and OA and the significantly higher risk that joint injury poses for the development of OA later in life. As ATs, we are well positioned to take an active role in educating patients about this risk and discussing management strategies that may preserve joint health over time. Such education can be challenging, particularly when patients, families, and coaches are focused on short-term outcomes after injury, when the primary goal is to return to sport as fast as possible, with little concern about the long-term consequences. However, as health care practitioners, we must remain objective and provide the information patients and their families need to make informed decisions about their care.

For ATs to educate patients about the risk of PTOA and discuss evidence-based management strategies after joint injury, we must have a solid understanding of how acute traumatic joint injury is associated with PTOA. However, the results of a recent study[104] suggested that ATs may have some misperceptions about the link between joint injury and PTOA. Pietrosimone et al[104] surveyed more than 400 ATs about their knowledge and perceptions of PTOA after knee-joint injury. In general, ATs agreed that the risk of OA increased after acute traumatic knee-joint injury, but they underestimated the percentage of patients who were likely to develop PTOA in the first or second decade after injury. They overestimated the effect that ACLR or meniscal surgery would have in preventing PTOA. Though many ATs agreed that the OA risk increased after injury, only about 70% reported that they discussed this risk and strategies to mitigate it with their patients. Nearly 40% of ATs indicated that they had no knowledge of PTOA; they were less likely to explain the risk of developing OA after acute traumatic knee-joint injury or to discuss strategies to mitigate this risk with athletes. Finally, ATs' knowledge of PTOA was associated with their years of clinical experience (Figure 1): those with greater than 15 years of clinical experience were more likely to report knowledge of PTOA. This finding suggests that knowledge of PTOA is likely not gained through professional education but through multiple years of clinical experiences and observing long-term outcomes in patients with significant joint injuries. As a result, it is critical to incorporate information about the association between joint injury and PTOA into entry-level training and continuing education opportunities for ATs.

PREVENTION AND MANAGEMENT OF PTOA

There is much the medical community still does not know about PTOA, including why it develops in some people postinjury and not in others. What is clear, however, is that those who sustain acute traumatic joint injuries are more likely to develop OA after injury and at a faster rate. Thus, the primary prevention of acute traumatic joint injuries could go a long way in reducing the risk of PTOA in physically active populations. Despite our best efforts, joint injuries will continue to occur in young athletes and physically active populations. In injured patients, we must shift our focus to secondary and tertiary prevention efforts to mitigate the risk of PTOA after joint injury (Table 1). Although it would be ideal to fully understand the underlying mechanisms and modifiable risk factors associated with PTOA before designing and implementing prevention strategies, members of the ATOAC and other professional organizations, such as the Chronic Osteoarthritis Management Initiative (COAMI) and the Osteoarthritis Action Alliance, believe that sufficient evidence supports prevention efforts for PTOA in several areas that

are aligned with the practice domains and educational competencies of athletic training. In this section, we will discuss how ATs can play a critical role in the primary prevention of acute traumatic joint injuries (and, by extension, PTOA) and in secondary and tertiary prevention by potentially delaying the onset of PTOA or mitigating the effects of the disease after injury.

Primary Injury Prevention

To aid in the prevention of PTOA, ATs should continue to promote, deliver, and evaluate the efficacy and effectiveness of primary injury-prevention programs designed to reduce the risk of acute traumatic joint injuries. Compared with the uninjured population, knee injury increases the risk of developing OA 4-fold and hip injury appears to increase the risk of PTOA 5-fold.[105] Therefore, by preventing the initial insult to the joint, it is likely that we can also prevent the long-term consequences of joint injury (eg, PTOA). Even though it is impossible to prevent all joint injuries, reducing a moderate number of these injuries would be a substantial step toward reducing the incidence and prevalence of PTOA in the young and active patient populations often treated by ATs. For example, if we were able to prevent 30% (or 75 000) of the roughly 250 000[106] ACL injuries that occur each year in the United States, we could prevent approximately 37 500 cases of PTOA if we assume that half of the persons who sustain ACL ruptures would go on to develop PTOA.

Neuromuscular training, which includes balance, strength, plyometric, and agility exercises, along with education regarding proper movement control, delivered as a preseason program or a warm-up during the season, appears to be effective in preventing lower extremity injury. In their recent meta-analysis, Emery et al[107] compiled outcomes from 25 investigations in which neuromuscular training was delivered to youth (aged 19 years or younger) who participated in team sports. The authors found that neuromuscular training had a protective effect on lower extremity injury risk, noting a 36% risk reduction. They also identified a preventive effect of neuromuscular training in reducing knee injuries by 26%, but this number did not reach statistical significance.[107] Sugimoto et al[108] observed a larger preventive effect of neuromuscular training for female athletes: 73.4% relative risk reduction. Specifically, a 73.4% relative risk reduction (ie, 73% less likely to experience a noncontact ACL injury) was evident in females who participated in a neuromuscular training program compared with those who did not. Herman et al[109] conducted a meta-analysis of 9 studies involving more than 13 000 participants to examine the effectiveness of neuromuscular warm-up strategies on injury prevention and demonstrated similar findings. Their study highlighted that, in general, neuromuscular training programs can reduce the risk of lower extremity and knee injuries. Further, it showed that protocols (eg, Anterior Knee Pain Prevention Training Program) are available to reduce the incidence of anterior knee pain in military recruits. Also of interest from their results is that neuromuscular training programs did not produce significant reductions in hip or thigh injuries. In summary, neuromuscular training appears to reduce lower extremity injury risk and, thus, we encourage ATs to incorporate such programs into sports and activities in which lower extremity injuries are common.

Although our intent was not to review neuromuscular training programs, we highlight the following key aspects of most effective programs[110]: (1) Neuromuscular training programs that incorporate multiple components (lower extremity and core-muscle–strengthening exercises, plyometric exercises, balance exercises, and consistent feedback to participants on proper technique) appear to demonstrate the greatest overall protective effect[111]; (2) making the programs sport specific and securing coach involvement appear to be important for successful implementation[112]; (3) completing the program at an appropriate dosage (ie, over at least 3 months and at all training sessions during that period[109] or 15 sessions over 6 weeks with minimum 15-minute exposures, started before the season)[110]; (4) no to minimal equipment is required to complete the program[110]; and (5) some ongoing maintenance training may be necessary to retain the benefits of these programs over time (ie, across multiple years or seasons).[113,114] More research is needed in many areas related to neuromuscular training programs, but implementation research that integrates theories of health-behavior change is a top priority. The focus needs to be on how to encourage people to change their attitudes and behaviors toward these neuromuscular training programs so that athletes and the physically active are more likely to adopt and maintain participation in the programs over time, as compliance is critical to program effectiveness.[115] Theories of health-behavior change have been applied to a number of health conditions to improve participation and compliance; however, many studies of musculoskeletal injury prevention, including ACL injury-prevention programs, have failed to integrate these theories. Also, research related to the necessary timing, dose, and strategies for effective retention is needed.

Proprioceptive training programs, which consist of exercises that challenge a patient's ability to target, detect, and react to varying joint positions,[116] are popular interventions for ankle-injury prevention. This type of training includes exercises such as balancing on a wobble or balance board, reacting to a sport-specific stimulus (eg, catching or throwing a ball) while standing on a single limb, and balancing on a single leg with the eyes closed. A recent meta-analysis[117] demonstrated that compared with various other control training interventions, proprioceptive training reduced ankle-sprain risk by 35% (relative risk = 0.65, 95% confidence interval = 0.55, 0.77). Similar findings were demonstrated among patients with or without a history of ankle sprain, reinforcing the importance of primary injury-prevention efforts. Although proprioceptive training may be effective for combating ankle injuries, evidence supporting its ability in isolation to reduce the overall risk of lower extremity injury, similar to neuromuscular training, is lacking. As such, we recommend adopting a neuromuscular training program that incorporates proprioceptive training over proprioceptive training alone so that any protective effects potentially span more than 1 joint.

No evidence to detail the effectiveness of primary prevention programs for upper extremity injuries is currently available. This is most likely because knowledge of upper extremity injuries lags behind that of lower

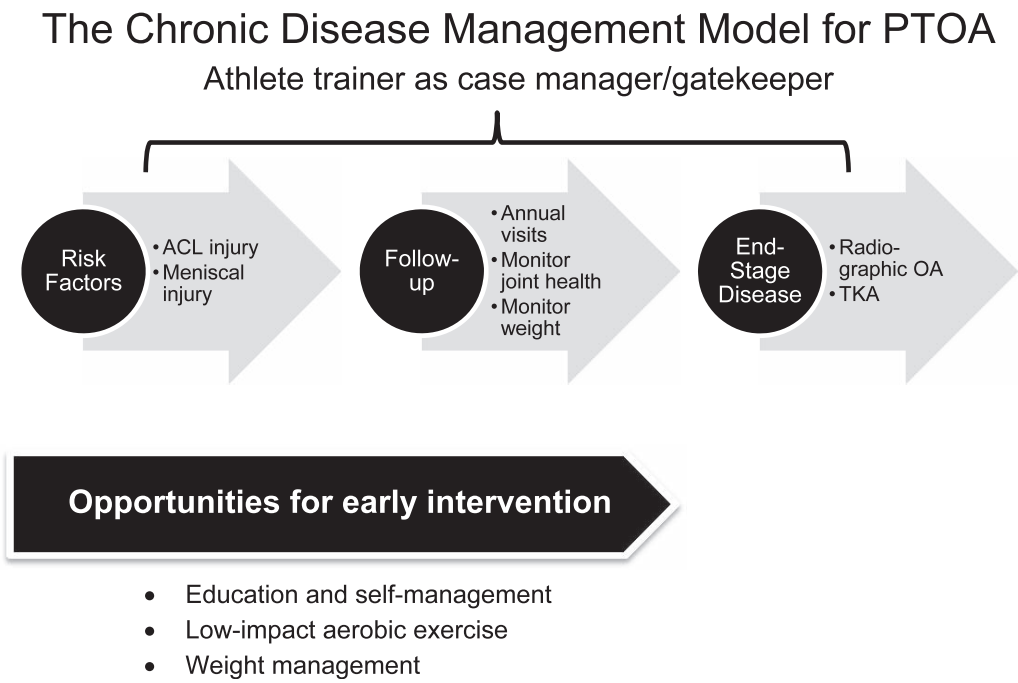

Figure 2. The chronic disease management model with an athletic trainer as case manager or gatekeeper. Abbreviations: ACL, anterior cruciate ligament; OA, osteoarthritis; PTOA, posttraumatic osteoarthritis; TKA: total knee arthroplasty.

extremity injuries. For prevention programs to be developed, we must determine risk factors for the shoulder, elbow, wrist, and hand and test programs aimed at targeting the modifiable risk factors for these upper extremity joint injuries.

Secondary and Tertiary Prevention

Traditionally, the management of acute traumatic joint injuries has ended when patients are deemed fit to return to activity. Treatment has focused on healing joint tissues, restoring anatomic structures, and improving functional capabilities through surgical repair or rehabilitation (or both). Patients are typically discharged from follow-up care within several weeks to more than 9 months, depending on the injury severity and treatment approach. However, as noted earlier, for many patients, these injuries are the starting point for a cascade of progressive pathologic joint changes (eg, PTOA) that, over the course of several years, leading to chronic pain and loss of function, resulting in limitations that affect both physical performance and activities of daily living. The paradigm in which the initial injury is treated and the long-term consequences are ignored must change.

In September 2012, COAMI issued a call to action highlighting the need to adopt a chronic management model for OA, similar to the models used to treat heart disease, diabetes, and hypertension.[15] The treatment goals of this chronic management model are to detect and modify risk factors early, before symptoms develop, so that the debilitating outcomes associated with PTOA can be mitigated or prevented entirely (Figure 2). The chronic management model typically focuses on secondary and tertiary prevention of PTOA after joint injury. In 2014, the American Orthopaedic Association conducted a symposium to examine emerging management strategies after acute traumatic joint injury in order to shift the clinical approach to PTOA from palliation to prevention.[118] A primary emphasis was the diagnosis and management of preosteoarthritis in the time between joint injury and the traditional radiographic diagnosis of OA: on average, 10 to 15 years after injury. It noted that orthopaedic surgeons who treat the full spectrum of joint injuries should have "an awareness of the paradigm shift toward the prevention of OA which is critical to the promotion of improved clinical care."[118] Athletic trainers who care for patients with acute traumatic joint injuries should also be aware of this paradigm shift focusing on the secondary and tertiary prevention of PTOA after joint injury.

Athletic trainers are in an ideal position to serve as case managers in the chronic management model for PTOA, particularly in young and physically active patients at risk for joint injury. We are also well positioned to detect and modify risk factors for PTOA as soon as possible after joint injury, preferably before symptoms appear, so that this debilitating condition can be managed more effectively or prevented entirely. It is important to point out that the traditional approach of waiting until radiographic or symptomatic OA is diagnosed to implement treatment is too late, as the disease process cannot be reversed; therefore, ATs must take steps to mitigate PTOA before it occurs. An overview of a possible chronic disease management model for PTOA after acute traumatic knee joint injury, in which an AT serves as a case manager, is shown in Figure 2. As noted previously, the AT is typically present when an athlete or patient sustains an acute traumatic knee-joint injury, and he or she commonly manages the care and referrals for treatment in the early stages of injury and throughout rehabilitation and return to play. During this time, ATs can also play a critical role in educating the patient about the risk of PTOA postinjury and discussing evidence-based management strategies to potentially mitigate or prevent the development and progres-

Table 2. Guidelines for the Management of Osteoarthritis (OA)[14]

1. Provide or refer patients to self-management education or exercise programs.
2. Advise patients to engage in low-impact aerobic exercise and, if overweight, to lose weight.
3. Walking aids and other assistive devices to improve activities of daily living are recommended for patients as needed. Based on current data, evidence is inconclusive for bracing or medial or lateral heel wedges for knee OA and for splints for thumb-base OA.
4. Thermal modalities are recommended for hand, knee, and hip OA; therapeutic ultrasound is not recommended for use; and insufficient evidence currently exists to provide a general recommendation regarding acupuncture, tai chi, or transcutaneous electrical nerve stimulation.
5. Joint replacement is recommended for appropriate patients with knee or hip OA.
6. Acetaminophen should be used as first-line therapy in symptomatic OA. Second-line agents should include topical agents (capsaicin and topical nonsteroidal anti-inflammatory drugs) and oral nonsteroidal anti-inflammatory drugs (with appropriate risk stratification and use of gastroprotective strategies). For refractory symptoms, tramadol is recommended, and consideration can be given to opioids or possibly duloxetine.
7. Intra-articular corticosteroids are recommended for knee and hip OA. Insufficient evidence currently exists to provide a general recommendation regarding intra-articular hyaluronans.

Note: These recommendations were generated by Nelson et al[14] as part of the Chronic Osteoarthritis Management Initiative of the US Bone and Joint Initiative. The recommendations were drafted for management of OA in general but can also be applied to posttraumatic OA. In some instances, we slightly modified the recommendations.

sion of PTOA (eg, self-management, weight management). Athletic trainers are also well positioned to monitor joint health and other known risk factors over the life span after joint injury. This can be accomplished through annual visits or preparticipation screening. The goals of these annual assessments are to monitor joint pain and function over time and to refer patients who report increased levels of pain or decreased levels of function, as these may be signs of preosteoarthritis. If we can identify these patients early, PTOA could be more effectively managed and perhaps further joint damage would not ensue or symptoms would not worsen. In other words, early identification would provide an opportunity for early interventions or behavior modifications (eg, weight management, activity modifications, exercise programs). Athletic trainers could also work with patients on appropriate exercise prescriptions and weight-management strategies to reduce symptoms, improve function, and potentially limit the development and progression of PTOA. These practices are in alignment with evidence-based recommendations for the management of OA.[14] The ultimate goal of secondary and tertiary prevention efforts is to push the end stage of disease as far off in the timeline as possible in an attempt to delay total joint failure and the need for total knee replacement surgery.

Evidence-Based Management Recommendations

Recently, Nelson et al[14] conducted a systematic review of treatment and management recommendations and guidelines for OA. They evaluated treatment recommendations for OA from all professional medical associations and societies that had published recommendations or guidelines at the time of their review. A summary of their recommendations is provided in Table 2. A key finding was agreement on many recommendations for the management of OA across the multiple professional societies and organizations making such recommendations; however, how these recommendations were being disseminated and implemented in clinical practice varied considerably. Although these recommendations were primarily focused on managing patients currently suffering from the symptoms and consequences of OA, some may be beneficial in the secondary and tertiary prevention of PTOA after acute traumatic joint injury and before the onset of symptoms or dysfunction. For example, self-management strategies for monitoring joint health over time, managing regular exercise and the type of physical activity patients engage in, and maintaining a healthy weight likely play important roles in the secondary prevention of PTOA after joint injury. The remaining recommendations are probably more appropriate for tertiary prevention to manage pain and symptoms in order to delay end-stage disease and total joint arthroplasty. We will elaborate on the recommendations for secondary PTOA prevention (weight management, physical activity, and self-management strategies), as they will likely be critical in improving the quality of life for those with PTOA (Figure 3). These recommendations were generated by the Centers for Disease Control and Prevention as management and intervention strategies for OA in general,[16] but they are also applicable to secondary prevention of PTOA.

Maintaining a healthy body weight is important. People who maintain a healthy weight are less likely to develop symptomatic knee OA,[119,120] and this appears to apply to PTOA as well. Englund and Lohmander[121] found that obesity was associated with symptomatic tibiofemoral PTOA after a meniscectomy in persons 15 to 22 years old. Greater body weight increases the loads transmitted across weight-bearing joints and can, therefore, increase the pain and symptoms associated with OA. In fact, Messier et al[122] observed that for each pound of weight lost, the load exerted on the knee per step during daily activities in adults with knee OA who were overweight or obese was reduced 4-fold. Further, in a subsequent randomized clinical trial, Messier et al[123] found that adults with knee OA who were overweight or obese and who participated in a diet and exercise regimen not only reduced compressive forces at the knee but also had reductions in inflammatory markers and self-reported pain as well as improvements in physical function. Therefore, ATs should encourage athletes to maintain a healthy weight (BMI <25) after joint injury. In addition, patients who are overweight (BMI ≥ 25) or obese (BMI ≥ 30) should be encouraged to manage weight through an individualized eating plan, physical activity and exercise regimen, and, if needed, referral to appropriate members of the sports medicine team.

Living a sedentary lifestyle could increase the risk for PTOA and other lifestyle-related diseases, such as diabetes and cardiovascular disease. Regular physical activity has been shown to decrease pain and disability and improve function in all forms of arthritis.[124] Low-impact, moderate-intensity aerobic physical activity (eg, cycling, swimming, walking) and muscle strengthening are considered the safest and most effective exercises for adults who have

Figure 3. Posttraumatic osteoarthritis secondary prevention recommendations from the Centers for Disease Control and Prevention.[16]

OA.[16] Although this guideline is likely also appropriate for patients at risk for PTOA, it may be difficult to implement, especially in younger athletes who participate in high-impact or elite-level sports. In these instances, until more research is available to support or refute sport participation and high-intensity physical activity as a risk factor for PTOA, patients should be educated to monitor their symptoms during activity and to report to a health care provider (AT, physician) if participation in their normal physical activities is causing them difficulty. In addition to aerobic activity, strengthening programs should be adopted to maximize muscle strength (eg, achieve preinjury strength values equivalent to the contralateral limb or strength values that allow a high level of physical function). This could not only help to prevent PTOA but also to reduce pain and increase joint function in those experiencing PTOA symptoms.[125] Maintaining muscle strength after achieving strength goals is critical to continued success of the initial program and, therefore, continued participation in strengthening exercises is warranted. Thus, even if preinjury strength levels are achieved, participation in strengthening programs should be continued at least 2 times per week.[16] Hence, patients should be encouraged to continue aerobic and strengthening exercises not only during their competitive careers but also throughout their lives.

Self-management education is a process in which patients with a chronic disease are taught how to manage their condition, prevent its consequences, and achieve the best possible quality of life. Several arthritis self-management programs are available. The Centers for Disease Control and Prevention currently recommend the use of the Arthritis Self-Management Program and the Chronic Disease Self-Management Program (each has an English and Spanish version available), as sufficient research supports their effectiveness.[126] Generally speaking, both programs contain information regarding how to deal with problems associated with arthritis and chronic disease; appropriate exercise regimens; appropriate use of medications; suggestions on how to communicate effectively with family, friends, and health care providers regarding arthritis and chronic disease; nutritional information; and guidelines on how to evaluate new treatments and therapies.[127,128] The research behind both programs shows that participation in these programs improves patients' confidence in managing their conditions, reduces depression and anxiety related to the condition, reduces pain, improves quality of life, and can lead to increased exercise participation.[129] Athletic trainers need to highlight to patients that the choices they make after joint injury will likely affect the initiation and progression of PTOA and that part of their self-management strategy is to understand the risk factors for PTOA and to make educated decisions with regard to physical activity, diet and weight management, and monitoring of joint health.

ROLE OF THE AT IN THE MANAGEMENT OF ADVANCED-STAGE OA

The focus of this review has been primarily on PTOA and the role of the AT in the prevention and management of PTOA, but the ATOAC contends that it is important to point out that ATs are also qualified to assist in the management of physically active patients who are experiencing advanced-stage PTOA or OA (ie, patients who have been diagnosed by a physician as already having the disorder, regardless of its origin). For example, the guidelines for the management of OA (Table 2) include the delivery of thermal and electrical modalities, the application of which are within the scope of practice for ATs. Furthermore, given our emphasis on and expertise with physically active individuals, we are in an ideal position to understand the unique challenges these patients present (eg, desire to return to sport, desire for high level of physical functioning).

SUMMARY

This review and our recommendations are intended to provide ATs with a basic knowledge of PTOA and the best evidence-based guidance for preventing and managing this condition. Athletic trainers should implement injury-prevention programs that reduce the number of joint-related injuries and thereby reduce the incidence and prevalence of PTOA. For patients who have sustained a joint injury, ATs should educate them about the risks of joint injury and schedule regular follow-up visits to monitor joint health and ensure adequate rehabilitation and appropriate return to play. Lastly, ATs should encourage patients to maintain a healthy weight and participate in regular low-impact aerobic exercise and strength training in an effort to prevent PTOA or minimize its symptoms in those who have already been diagnosed with the condition.

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REFERENCES

1. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States, part II. *Arthritis Rheum*. 2008;58(1):26–35.
2. Kotlarz H, Gunnarsson CL, Fang H, Rizzo JA. Insurer and out-of-pocket costs of osteoarthritis in the US: evidence from national survey data. *Arthritis Rheum*. 2009;60(12):3546–3553.
3. Suri P, Morgenroth DC, Hunter DJ. Epidemiology of osteoarthritis and associated comorbidities. *PM R*. 2012;4(suppl 5):S10–S19.
4. Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012;380(9859):2163–2196.
5. Gelber AC, Hochberg MC, Mead LA, Wang NY, Wigley FM, Klag MJ. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Ann Intern Med*. 2000;133(5):321–328.
6. Roos EM. Joint injury causes knee osteoarthritis in young adults. *Curr Opin Rheumatol*. 2005;17(2):195–200.
7. 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.
8. Deshpande BR, Katz JN, Solomon DH, et al. The number of persons with symptomatic knee osteoarthritis in the United States: impact of race and ethnicity, age, sex, and obesity. *Arthritis Care Res (Hoboken)*. 2016;68(12):1743–1750.
9. Ackerman IN, Bucknill A, Page RS, et al. The substantial personal burden experienced by younger people with hip or knee osteoarthritis. *Osteoarthritis Cartilage*. 2015;23(8):1276–1284.
10. Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. *Am J Sports Med*. 2007;35(10):1756–1769.
11. Felson DT, Zhang Y. An update on the epidemiology of knee and hip osteoarthritis with a view to prevention. *Arthritis Rheum*. 1998; 41(8):1343–1355.
12. Muthuri SG, McWilliams DF, Doherty M, Zhang W. History of knee injuries and knee osteoarthritis: a meta-analysis of observational studies. *Osteoarthritis Cartilage*. 2011;19(11):1286–1293.
13. Brown TD, Johnston RC, Saltzman CL, Marsh JL, Buckwalter JA. Posttraumatic osteoarthritis: a first estimate of incidence, prevalence, and burden of disease. *J Orthop Trauma*. 2006;20(10):739–744.
14. Nelson AE, Allen KD, Golightly YM, Goode AP, Jordan JM. A systematic review of recommendations and guidelines for the management of osteoarthritis: the chronic osteoarthritis management initiative of the U.S. bone and joint initiative. *Semin Arthritis Rheum*. 2014;43(6):701–712.
15. A call to action from the Chronic Osteoarthritis Management Initiative (COAMI). United States Bone and Joint Initiative Web site. http://www.usbji.org/sites/default/files/COAMI Call to action.pdf. Published 2012. Accessed April 5, 2016.
16. A national public health agenda for osteoarthrtis, 2010. Centers for Disease Control and Prevention Web site. http://www.cdc.gov/arthritis/docs/OAAagenda.pdf. Accessed April 1, 2016.
17. Emery CA, Roos EM, Verhagen E, et al. OARSI Clinical Trials Recommendations: design and conduct of clinical trials for primary prevention of osteoarthritis by joint injury prevention in sport and recreation. *Osteoarthritis Cartilage*. 2015;23(5):815–825.
18. Little CB, Hunter DJ. Post-traumatic osteoarthritis: from mouse models to clinical trials. *Nat Rev Rheumatol*. 2013;9(8):485–497.
19. Anderson DD, Chubinskaya S, Guilak F, et al. Post-traumatic osteoarthritis: improved understanding and opportunities for early intervention. *J Orthop Res*. 2011;29(6):802–809.
20. Thomas TP, Anderson DD, Mosqueda TV, et al. Objective CT-based metrics of articular fracture severity to assess risk for posttraumatic osteoarthritis. *J Orthop Trauma*. 2010;24(12):764–769.
21. Rivera JC, Wenke JC, Buckwalter JA, Ficke JR, Johnson AE. Posttraumatic osteoarthritis caused by battlefield injuries: the primary source of disability in warriors. *J Am Acad Orthop Surg*. 2012;20(suppl 1):S64–S69.
22. Ni GX, Lei L, Zhou YZ. Intensity-dependent effect of treadmill running on lubricin metabolism of rat articular cartilage. *Arthritis Res Ther*. 2012;14(6):R256.
23. Arokoski J, Kiviranta I, Jurvelin J, Tammi M, Helminen HJ. Long-distance running causes site-dependent decrease of cartilage glycosaminoglycan content in the knee joints of beagle dogs. *Arthritis Rheum*. 1993;36(10):1451–1459.
24. Driban JB, Barr AE, Amin M, Sitler MR, Barbe MF. Joint inflammation and early degeneration induced by high-force reaching are attenuated by ibuprofen in an animal model of work-related musculoskeletal disorder. *J Biomed Biotechnol*. 2011;2011: 691412.
25. Kramer WC, Hendricks KJ, Wang J. Pathogenetic mechanisms of posttraumatic osteoarthritis: opportunities for early intervention. *Int J Clin Exp Med*. 2011;4(4):285–298.
26. Roemer FW, Frobell R, Hunter DJ, et al. MRI-detected subchondral bone marrow signal alterations of the knee joint: terminology, imaging appearance, relevance and radiological differential diagnosis. *Osteoarthritis Cartilage*. 2009;17(9):1115–1131.
27. Frobell RB, Roos HP, Roos EM, et al. The acutely ACL injured knee assessed by MRI: are large volume traumatic bone marrow lesions a sign of severe compression injury? *Osteoarthritis Cartilage*. 2008;16(7):829–836.
28. Theologis AA, Kuo D, Cheng J, et al. Evaluation of bone bruises and associated cartilage in anterior cruciate ligament-injured and -reconstructed knees using quantitative t(1rho) magnetic resonance imaging: 1-year cohort study. *Arthroscopy*. 2011;27(1):65–76.
29. Fang C, Johnson D, Leslie MP, Carlson CS, Robbins M, Di Cesare PE. Tissue distribution and measurement of cartilage oligomeric matrix protein in patients with magnetic resonance imaging-detected bone bruises after acute anterior cruciate ligament tears. *J Orthop Res*. 2001;19(4):634–641.
30. Johnson DL, Urban WP Jr, Caborn DN, Vanarthos WJ, Carlson CS. Articular cartilage changes seen with magnetic resonance imaging-detected bone bruises associated with acute anterior cruciate ligament rupture. *Am J Sports Med*. 1998;26(3):409–414.
31. Lee JH, Ort T, Ma K, et al. Resistin is elevated following traumatic joint injury and causes matrix degradation and release of inflammatory cytokines from articular cartilage in vitro. *Osteoarthritis Cartilage*. 2009;17(5):613–620.

32. Nietfeld JJ, Wilbrink B, Helle M, et al. Interleukin-1-induced interleukin-6 is required for the inhibition of proteoglycan synthesis by interleukin-1 in human articular cartilage. *Arthritis Rheum*. 1990; 33(11):1695–1701.
33. Rowan AD, Koshy PJ, Shingleton WD, et al. Synergistic effects of glycoprotein 130 binding cytokines in combination with interleukin-1 on cartilage collagen breakdown. *Arthritis Rheum*. 2001; 44(7):1620–1632.
34. Elsaid KA, Machan JT, Waller K, Fleming BC, Jay GD. The impact of anterior cruciate ligament injury on lubricin metabolism and the effect of inhibiting tumor necrosis factor alpha on chondroprotection in an animal model. *Arthritis Rheum*. 2009;60(10):2997–3006.
35. Harkey MS, Luc BA, Golightly YM, et al. Osteoarthritis-related biomarkers following anterior cruciate ligament injury and reconstruction: a systematic review. *Osteoarthritis Cartilage*. 2015;23(1):1–12.
36. Hunter DJ, Lohmander LS, Makovey J, et al. The effect of anterior cruciate ligament injury on bone curvature: exploratory analysis in the KANON trial. *Osteoarthritis Cartilage*. 2014;22(7):959–968.
37. Neogi T, Bowes MA, Niu J, et al. Magnetic resonance imaging-based three-dimensional bone shape of the knee predicts onset of knee osteoarthritis: data from the osteoarthritis initiative. *Arthritis Rheum*. 2013;65(8):2048–2058.
38. 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.
39. Tashman S, Collon D, Anderson K, Kolowich P, Anderst W. Abnormal rotational knee motion during running after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2004;32(4): 975–983.
40. Chaudhari AM, Briant PL, Bevill SL, Koo S, Andriacchi TP. Knee kinematics, cartilage morphology, and osteoarthritis after ACL injury. *Med Sci Sports Exerc*. 2008;40(2):215–222.
41. Cattano NM, Barbe MF, Massicotte MR, et al. Joint trauma initiates knee osteoarthritis through biochemical and biomechanical processes and interactions. *OA Musculskelet Med*. 2013;1(1):3.
42. Lotz MK, Kraus VB. New developments in osteoarthritis. Posttraumatic osteoarthritis: pathogenesis and pharmacological treatment options. *Arthritis Res Ther*. 2010;12(3):211.
43. Riordan EA, Little C, Hunter D. Pathogenesis of post-traumatic OA with a view to intervention. *Best Pract Res Clin Rheumatol*. 2014; 28(1):17–30.
44. Risk factors, 2016. World Health Organization Web site. http://www.who.int/topics/risk_factors/en. Accessed February 23, 2016.
45. Palmer-Green DS, Batt ME, Scammell BE. Simple advice for a simple ankle sprain? The not so benign ankle injury. *Osteoarthritis Cartilage*. 2016;24(6):947–948.
46. Cooper C, Inskip H, Croft P, et al. Individual risk factors for hip osteoarthritis: obesity, hip injury, and physical activity. *Am J Epidemiol*. 1998;147(6):516–522.
47. Saltzman CL, Salamon ML, Blanchard GM, et al. Epidemiology of ankle arthritis: report of a consecutive series of 639 patients from a tertiary orthopaedic center. *Iowa Orthop J*. 2005;25:44–46.
48. Valderrabano V, Horisberger M, Russell I, Dougall H, Hintermann B. Etiology of ankle osteoarthritis. *Clin Orthop Relat Res*. 2009; 467(7):1800–1806.
49. Badlani JT, Borrero C, Golla S, Harner CD, Irrgang JJ. The effects of meniscus injury on the development of knee osteoarthritis: data from the osteoarthritis initiative. *Am J Sports Med*. 2013;41(6): 1238–1244.
50. Parkkinen M, Madanat R, Mustonen A, Koskinen SK, Paavola M, Lindahl J. Factors predicting the development of early osteoarthritis following lateral tibial plateau fractures: mid-term clinical and radiographic outcomes of 73 operatively treated patients. *Scand J Surg*. 2014;103(4):256–262.
51. Rademakers MV, Kerkhoffs GM, Sierevelt IN, Raaymakers EL, Marti RK. Intra-articular fractures of the distal femur: a long-term follow-up study of surgically treated patients. *J Orthop Trauma*. 2004;18(4):213–219.
52. Rademakers MV, Kerkhoffs GM, Kager J, Goslings JC, Marti RK, Raaymakers EL. Tibial spine fractures: a long-term follow-up study of open reduction and internal fixation. *J Orthop Trauma*. 2009; 23(3):203–207.
53. Oiestad BE, Holm I, Engebretsen L, Aune AK, Gunderson R, Risberg MA. The prevalence of patellofemoral osteoarthritis 12 years after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 2013;21(4):942–949.
54. Lundberg M, Messner K. Ten-year prognosis of isolated and combined medial collateral ligament ruptures: a matched comparison in 40 patients using clinical and radiographic evaluations. *Am J Sports Med*. 1997;25(1):2–6.
55. Lundberg M, Messner K. Long-term prognosis of isolated partial medial collateral ligament ruptures: a ten-year clinical and radiographic evaluation of a prospectively observed group of patients. *Am J Sports Med*. 1996;24(2):160–163.
56. Kannus P. Long-term results of conservatively treated medial collateral ligament injuries of the knee joint. *Clin Orthop Relat Res*. 1988;226:103–112.
57. Kannus P, Jarvinen M. Osteoarthrosis in a knee joint due to chronic posttraumatic insufficiency of the medial collateral ligament: nine-year follow-up. *Clin Rheumatol*. 1988;7(2):200–207.
58. Allain J, Goutallier D, Glorion C. Long-term results of the Latarjet procedure for the treatment of anterior instability of the shoulder. *J Bone Joint Surg Am*. 1998;80(6):841–852.
59. Berendes TD, Wolterbeek R, Pilot P, Verburg H, te Slaa RL. The open modified Bankart procedure: outcome at follow-up of 10 to 15 years. *J Bone Joint Surg Br*. 2007;89(8):1064–1068.
60. Buscayret F, Edwards TB, Szabo I, Adeleine P, Coudane H, Walch G. Glenohumeral arthrosis in anterior instability before and after surgical treatment: incidence and contributing factors. *Am J Sports Med*. 2004;32(5):1165–1172.
61. Plath JE, Aboalata M, Seppel G, et al. Prevalence of and risk factors for dislocation arthropathy: radiological long-term outcome of arthroscopic bankart repair in 100 shoulders at an average 13-year follow-up. *Am J Sports Med*. 2015;43(5):1084–1090.
62. Hovelius L, Olofsson A, Sandstrom B, et al. Nonoperative treatment of primary anterior shoulder dislocation in patients forty years of age and younger: a prospective twenty-five-year follow-up. *J Bone Joint Surg Am*. 2008;90(5):945–952.
63. Vollnberg B, Koehlitz T, Jung T, et al. Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation. *Eur Radiol*. 2012;22(11):2347–2356.
64. Zheng H, Chen C. Body mass index and risk of knee osteoarthritis: systematic review and meta-analysis of prospective studies. *BMJ Open*. 2015;5(12):e007568.
65. Grotle M, Hagen KB, Natvig B, Dahl FA, Kvien TK. Obesity and osteoarthritis in knee, hip and/or hand: an epidemiological study in the general population with 10 years follow-up. *BMC Musculoskelet Disord*. 2008;9:132.
66. Lubbeke A, Salvo D, Stern R, Hoffmeyer P, Holzer N, Assal M. Risk factors for post-traumatic osteoarthritis of the ankle: an eighteen year follow-up study. *Int Orthop*. 2012;36(7):1403–1410.
67. Louer CR, Furman BD, Huebner JL, Kraus VB, Olson SA, Guilak F. Diet-induced obesity significantly increases the severity of posttraumatic arthritis in mice. *Arthritis Rheum*. 2012;64(10):3220–3230.
68. Spindler KP, Warren TA, Callison JC Jr, Secic M, Fleisch SB, Wright RW. Clinical outcome at a minimum of five years after reconstruction of the anterior cruciate ligament. *J Bone Joint Surg Am*. 2005;87(8):1673–1679.

69. Roe J, Pinczewski LA, Russell VJ, Salmon LJ, Kawamata T, Chew M. A 7-year follow-up of patellar tendon and hamstring tendon grafts for arthroscopic anterior cruciate ligament reconstruction: differences and similarities. *Am J Sports Med*. 2005;33(9):1337–1345.
70. Shelbourne KD, Gray T. Minimum 10-year results after anterior cruciate ligament reconstruction: how the loss of normal knee motion compounds other factors related to the development of osteoarthritis after surgery. *Am J Sports Med*. 2009;37(3):471–480.
71. Shelbourne KD, Urch SE, Gray T, Freeman H. Loss of normal knee motion after anterior cruciate ligament reconstruction is associated with radiographic arthritic changes after surgery. *Am J Sports Med*. 2012;40(1):108–113.
72. Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. *J Athl Train*. 2002;37(4):364–375.
73. Hoch MC, Staton GS, Medina McKeon JM, Mattacola CG, McKeon PO. Dorsiflexion and dynamic postural control deficits are present in those with chronic ankle instability. *J Sci Med Sport*. 2012;15(6):574–579.
74. Hoch MC, Farwell KE, Gaven SL, Weinhandl JT. Weight-bearing dorsiflexion range of motion and landing biomechanics in individuals with chronic ankle instability. *J Athl Train*. 2015;50(8):833–839.
75. Palmieri-Smith RM, Thomas AC, Wojtys EM. Maximizing quadriceps strength after ACL reconstruction. *Clin Sports Med*. 2008;27(3):405–424, vii–ix.
76. Lepley LK. Deficits in quadriceps strength and patient-oriented outcomes at return to activity after ACL reconstruction: a review of the current literature. *Sports Health*. 2015;7(3):231–238.
77. Palmieri-Smith RM, Kreinbrink J, Ashton-Miller JA, Wojtys EM. Quadriceps inhibition induced by an experimental knee joint effusion affects knee joint mechanics during a single-legged drop landing. *Am J Sports Med*. 2007;35(8):1269–1275.
78. Palmieri-Smith RM, Thomas AC. A neuromuscular mechanism of posttraumatic osteoarthritis associated with ACL injury. *Exerc Sport Sci Rev*. 2009;37(3):147–153.
79. Oiestad BE, Holm I, Gunderson R, Myklebust G, Risberg MA. Quadriceps muscle weakness after anterior cruciate ligament reconstruction: a risk factor for knee osteoarthritis? *Arthritis Care Res (Hoboken)*. 2010;62(12):1706–1714.
80. 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.
81. Tourville TW, Jarrell KM, Naud S, Slauterbeck JR, Johnson RJ, Beynnon BD. Relationship between isokinetic strength and tibiofemoral joint space width changes after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2014;42(2):302–311.
82. Brouwer GM, van Tol AW, Bergink AP, et al. Association between valgus and varus alignment and the development and progression of radiographic osteoarthritis of the knee. *Arthritis Rheum*. 2007;56(4):1204–1211.
83. Felson DT, Niu J, Gross KD, et al. Valgus malalignment is a risk factor for lateral knee osteoarthritis incidence and progression: findings from the Multicenter Osteoarthritis Study and the Osteoarthritis Initiative. *Arthritis Rheum*. 2013;65(2):355–362.
84. Sward P, Friden T, Boegard T, Kostogiannis I, Neuman P, Roos H. Association between varus alignment and post-traumatic osteoarthritis after anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc*. 2013;21(9):2040–2047.
85. Laprade RF, Spiridonov SI, Nystrom LM, Jansson KS. Prospective outcomes of young and middle-aged adults with medial compartment osteoarthritis treated with a proximal tibial opening wedge osteotomy. *Arthroscopy*. 2012;28(3):354–364.
86. Hootman JM, Macera CA, Helmick CG, Blair SN. Influence of physical activity-related joint stress on the risk of self-reported hip/knee osteoarthritis: a new method to quantify physical activity. *Prev Med*. 2003;36(5):636–644.
87. Hansen P, English M, Willick SE. Does running cause osteoarthritis in the hip or knee? *PM R*. 2012;4(suppl 5):S117–S121.
88. Michaelsson K, Byberg L, Ahlbom A, Melhus H, Farahmand BY. Risk of severe knee and hip osteoarthritis in relation to level of physical exercise: a prospective cohort study of long-distance skiers in Sweden. *PLoS One*. 2011;6(3):e18339.
89. Urquhart DM, Tobing JF, Hanna FS, et al. What is the effect of physical activity on the knee joint? A systematic review. *Med Sci Sports Exerc*. 2011;43(3):432–442.
90. Wijayaratne SP, Teichtahl AJ, Wluka AE, et al. The determinants of change in patella cartilage volume—a cohort study of healthy middle-aged women. *Rheumatology (Oxford)*. 2008;47(9):1426–1429.
91. Mosher TJ, Liu Y, Torok CM. Functional cartilage MRI T2 mapping: evaluating the effect of age and training on knee cartilage response to running. *Osteoarthritis Cartilage*. 2010;18(3):358–364.
92. Driban JB, Hootman JM, Sitler MR, Harris KP, Cattano NM. Is participation in certain sports associated with knee osteoarthritis? A systematic review. *J Athl Train*. 2017;52(6):497–506.
93. Roos H, Lindberg H, Gardsell P, Lohmander LS, Wingstrand H. The prevalence of gonarthrosis and its relation to meniscectomy in former soccer players. *Am J Sports Med*. 1994;22(2):219–222.
94. Roos EM, Lohmander LS. The Knee injury and Osteoarthritis Outcome Score (KOOS): from joint injury to osteoarthritis. *Health Qual Life Outcomes*. 2003;1:64.
95. Roos EM, Toksvig-Larsen S. Knee injury and Osteoarthritis Outcome Score (KOOS) - validation and comparison to the WOMAC in total knee replacement. *Health Qual Life Outcomes*. 2003;1:17.
96. Bellamy N, Buchanan WW, Goldsmith CH, Campbell J, Stitt LW. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. *J Rheumatol*. 1988;15(12):1833–1840.
97. Cameron KL, Peck KY, Thompson BS, Svoboda SJ, Owens BD, Marshall SW. Reference values for the Marx Activity Rating Scale in a young athletic population: history of knee ligament injury is associated with higher scores. *Sports Health*. 2015;7(5):403–408.
98. Cameron KL, Thompson BS, Peck KY, Owens BD, Marshall SW, Svoboda SJ. Normative values for the KOOS and WOMAC in a young athletic population: history of knee ligament injury is associated with lower scores. *Am J Sports Med*. 2013;41(3):582–589.
99. Lam KC, St Thomas S, Snyder Valier AR, Valovich McLeod TC, Bay RC. Previous knee injury and health-related quality of life in collegiate athletes. *J Athl Train*. 2017;52(6):534–540.
100. Tsai LC, McLean S, Colletti PM, Powers CM. Greater muscle co-contraction results in increased tibiofemoral compressive forces in females who have undergone anterior cruciate ligament reconstruction. *J Orthop Res*. 2012;30(12):2007–2014.
101. Perraton L, Clark R, Crossley K, et al. Impaired voluntary quadriceps force control following anterior cruciate ligament reconstruction: relationship with knee function. *Knee Surg Sports Traumatol Arthrosc*. In press.
102. 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.
103. *Practice Analysis*. 7th ed. Omaha, NE: Board of Certification Inc; 2015.
104. Pietrosimone B, Blackburn JT, Golightly YM, et al. Certified athletic trainers' knowledge and perceptions of posttraumatic osteoarthritis after knee injury. *J Athl Train*. 2017;52(6):541–559.
105. Richmond SA, Fukuchi RK, Ezzat A, Schneider K, Schneider G, Emery CA. Are joint injury, sport activity, physical activity, obesity,

or occupational activities predictors for osteoarthritis? A systematic review. *J Orthop Sports Phys Ther*. 2013;43(8):515–B519.
106. Griffin LY, Albohm MJ, Arendt EA, et al. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am J Sports Med*. 2006;34(9):1512–1532.
107. Emery CA, Roy TO, Whittaker JL, Nettel-Aguirre A, van Mechelen W. Neuromuscular training injury prevention strategies in youth sport: a systematic review and meta-analysis. *Br J Sports Med*. 2015;49(13):865–870.
108. Sugimoto D, Myer GD, McKeon JM, Hewett TE. Evaluation of the effectiveness of neuromuscular training to reduce anterior cruciate ligament injury in female athletes: a critical review of relative risk reduction and numbers-needed-to-treat analyses. *Br J Sports Med*. 2012;46(14):979–988.
109. Herman K, Barton C, Malliaras P, Morrissey D. The effectiveness of neuromuscular warm-up strategies, that require no additional equipment, for preventing lower limb injuries during sports participation: a systematic review. *BMC Med*. 2012;10:75.
110. Consensus opinion for best practice features of lower limb injury prevention programs. Osteoarthritis Action Alliance Web site. http://oaaction.unc.edu/resource-library/for-public/injury-prevention. Accessed April 1, 2016.
111. Lauersen JB, Bertelsen DM, Andersen LB. The effectiveness of exercise interventions to prevent sports injuries: a systematic review and meta-analysis of randomised controlled trials. *Br J Sports Med*. 2014;48(11):871–877.
112. Bizzini M, Junge A, Dvorak J. Implementation of the FIFA 11+ football warm up program: how to approach and convince the Football associations to invest in prevention. *Br J Sports Med*. 2013; 47(12):803–806.
113. Padua DA, DiStefano LJ, Marshall SW, Beutler AI, de la Motte SJ, DiStefano MJ. Retention of movement pattern changes after a lower extremity injury prevention program is affected by program duration. *Am J Sports Med*. 2012;40(2):300–306.
114. DiStefano LJ, Marshall SW, Padua DA, et al. The effects of an injury prevention program on landing biomechanics over time. *Am J Sports Med*. 2016;44(3):767–776.
115. Sugimoto D, Myer GD, Bush HM, Klugman MF, Medina McKeon JM, Hewett TE. Compliance with neuromuscular training and anterior cruciate ligament injury risk reduction in female athletes: a meta-analysis. *J Athl Train*. 2012;47(6):714–723.
116. Lephart SM, Pincivero DM, Giraldo JL, Fu FH. The role of proprioception in the management and rehabilitation of athletic injuries. *Am J Sports Med*. 1997;25(1):130–137.
117. Schiftan GS, Ross LA, Hahne AJ. The effectiveness of proprioceptive training in preventing ankle sprains in sporting populations: a systematic review and meta-analysis. *J Sci Med Sport*. 2015;18(3): 238–244.
118. Chu CR, Millis MB, Olson SA. Osteoarthritis: from palliation to prevention: AOA critical issues. *J Bone Joint Surg Am*. 2014; 96(15):e130.
119. Sridhar MS, Jarrett CD, Xerogeanes JW, Labib SA. Obesity and symptomatic osteoarthritis of the knee. *J Bone Joint Surg Br*. 2012; 94(4):433–440.
120. Felson DT. Weight and osteoarthritis. *Am J Clin Nutr*. 1996; 63(suppl 3):430S–432S.
121. Englund M, Lohmander LS. Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy. *Arthritis Rheum*. 2004;50(9):2811–2819.
122. Messier SP, Gutekunst DJ, Davis C, DeVita P. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. *Arthritis Rheum*. 2005;52(7):2026–2032.
123. Messier SP, Mihalko SL, Legault C, et al. Effects of intensive diet and exercise on knee joint loads, inflammation, and clinical outcomes among overweight and obese adults with knee osteoarthritis: the IDEA randomized clinical trial. *JAMA*. 2013;310(12):1263–1273.
124. *Physical Activity Guidelines Advisory Committee Report*. Washington, DC: US Department of Health and Human Services; 2008.
125. Pietrosimone B, Lepley AS, Harkey MS, et al. Quadriceps strength predicts self-reported function post ACL reconstruction. *Med Sci Sports Exerc*. 2016;48(9):1671–1677.
126. Self-management education for arthritis, 2015. Centers for Disease Control and Prevention Web site. www.cdc.gov/arthritis/interventions/self-manage.htm. Accessed April 7, 2016.
127. Lorig KR, Ritter PL, Plant K. A disease-specific self-help program compared with a generalized chronic disease self-help program for arthrtis patients. *Arthritis Rheum*. 2005;53(6):950–957.
128. Lorig KR, Sobel DS, Stewart AL, et al. Evidence suggesting that a chronic disease self-management program can improve health status while reducing hospitalization: a randomized trial. *Med Care*. 1999; 37(1):5–14.
129. *Sorting Through the Evidence for the Arthritis Self-Management Program and the Chronic Disease Self-Management Program: Executive Summary of ASMP/CDSMP Meta-Analysis*. Atlanta, GA: Centers for Disease Control and Prevention; 2011:1–24.

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