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10

# A pragmatic approach to prevent post-traumatic osteoarthritis after sport or exercise-related joint injury



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### A B S T R A C T

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Lower extremity musculoskeletal injuries are common in sport and exercise, and associated with increased risk of obesity and post-traumatic osteoarthritis (PTOA). Unlike other forms of osteoarthritis, PTOA is common at a younger age and associated with more rapid progression, which may impact career choices, long-term general health and reduce quality of life. Individuals who suffer an activity-related joint injury and present with abnormal joint morphology, elevated adiposity, weak musculature, or become physically inactive are at increased risk of PTOA. Insufficient exercise therapy or incomplete rehabilitation, premature return-to-sport and re-injury, unrealistic expectations, or poor nutrition may further elevate this risk. Delay in surgical interventions in lieu of exercise therapy to optimize muscle strength and neuromuscular control while addressing fear of movement to guarantee resumption of physical activity, completeness of rehabilitation before return-to-sport, education that promotes realistic expectations and self-management, and nutritional counseling are the best approaches for delaying or preventing PTOA.

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## Introduction

Despite numerous benefits, sport and exercise participation is a leading cause of lower extremity musculoskeletal injury. These injuries are associated with a variety of negative consequences including a significantly elevated risk of developing post-traumatic osteoarthritis (PTOA), subsequent comorbidities, and reduced health-related quality of life (QOL). Although a great deal is understood about how to prevent sport and exercise-related musculoskeletal injuries and how to reduce disability in persons with PTOA, there is a paucity of knowledge about how to prevent or delay the onset of PTOA after an injury has occurred. This paper 1) presents a risk profile for PTOA based on a synthesis of findings from investigations that have assessed modifiable risk/protective factors in the interval between joint injury and PTOA onset, and 2) proposes guiding principles for preventing PTOA after a sport or exercise-related lower extremity musculoskeletal injury aimed at pragmatically addressing risk factors that are amenable to intervention. For the purposes of this chapter, PTOA and osteoarthritis (OA) signify a combination of pain, reduced function, and structural OA features (e.g., bone marrow lesions, joint space narrowing, and osteophyte formation), often referred to as symptomatic OA [1], as opposed to structural features alone unless otherwise stated. This definition has been chosen given the considerable discordance between the symptoms and structural features of OA [2,3] and to support the argument that the prevention of OA involves upstream management of the symptoms, functional restrictions, and structural features of this disease.

## Musculoskeletal injury in sport

Sport and recreational activities account for up to 40% of injuries requiring medical attention, with 50% involving the ankle, knee, or hip [4,5]. The most commonly injured joint during sporting activities is the ankle [6] followed by the knee [7]. The highest rates of injury are in youth team sports that involve contact, quick changes of direction, or rapid acceleration and deceleration [8]. Beyond the high prevalence of these injuries, there are an increasing number of signs warning that sport and recreational injuries are on the rise [9].

Sport and exercise-related injuries are associated with a variety of negative consequences. In the short term, they can lead to negative mood states, re-injury anxiety, feelings of isolation, loss of social identity, withdrawal from sport, re-injury, and physical inactivity. For example, individuals currently undergoing rehabilitation for a sport-related injury report frustration, anxiety, and anger associated with missing substantial periods of training and competition, letting their teammates and coaches down, and falling behind in their competitive ability and physical fitness [10]. Further, it is estimated that following a significant injury, such as an Anterior Cruciate Ligament (ACL) and surgical reconstruction (ACLR), only 65% of patients return to pre-injury sport and only 55% return to competitive sport by two years [11]. Overall, 15% of patients who undergo an ACLR suffer a second ACL injury (7% ipsilateral and 8% contralateral), with the re-injury rate increasing to 23% for those who return-to-sport [12]. Further, patients two years post-ACLR spend less time in moderate-to-vigorous physical activity and have lower step counts than uninjured controls [13].

In the long term, sport and exercise-related injuries are associated with obesity, reduced QOL, and PTOA. Youth who suffer a sport-related knee injury demonstrate higher total body and abdominal adiposity within 3–10 years following injury than uninjured controls [14]. This is concerning given that markers of obesity in adolescence are associated with high adiposity and increased risk of morbidity and mortality later in life [15], and adiposity is known to contribute to the development of knee OA through mechanical [16] and systemic [17] mechanisms. With regard to QOL, injured adolescent athletes report reductions in the physical and social domains of health-related QOL compared to uninjured peers [18]. Further, individuals who have undergone ACLR 5–26 years previously demonstrate reduced knee-related QOL [19], with further reductions seen in the presence of symptomatic radiographic OA [20]. Potential explanations for reductions in QOL in the ACLR population include failure to return-to-sport, subsequent surgery following primary ACLR, and elevated body mass index (BMI) [21].

Beyond adiposity and QOL, there is an increased incidence of early-onset symptomatic and/or radiographic- or MRI-defined PTOA associated with sport-related joint injuries [22]. Specifically, joint injury is associated with a fourfold (3.9 95% CI 2.7, 5.6) increased risk of radiographic knee OA [23] and a

fivefold (5.0 95% CI 1.9, 13.3) increased risk of clinically diagnosed hip OA [24]. Unlike the knee and hip joint, where non-traumatic OA is more common than PTOA, up to 90% of ankle OA is post-traumatic in nature [25], with 13% of patients with chronic ankle instability demonstrating radiographic OA 20 years following injury. Distinct to other forms of OA, PTOA (which accounts for up to 12% of OA cases) [26] is associated with an early age of onset and more rapid progression to end-stage disease, thereby resulting in a greater number of years lived with disability (YLD) and socioeconomic burden [26–28]. In fact, patients with a history of previous knee surgery, including ACLR, undergo total knee arthroplasty (end-stage treatment), on average, seven and nine years younger respectively, than those without this history [29].

## The epidemiology and burden of osteoarthritis

Osteoarthritis ranks 11th out of 291 in terms of YLD and is a major source of pain, mobility disability, and socioeconomic cost worldwide [30]. Although the onset of OA is multifactorial, the two most established risk factors are previous joint injury and obesity [23,31,32]. Specifically, 20–50% of people develop symptomatic radiographic OA following joint trauma [33], while the lifetime risk of symptomatic radiographic OA increases with increasing BMI [31].

Osteoarthritis is projected to become the fourth leading cause of disability worldwide by 2020 [34], with one-quarter of North Americans diagnosed with the disease by 2040 [35,36]. The increasing prevalence and expanding burden of OA has been attributed to lifestyle factors (i.e., inactivity and nutrition) [37], changing socio-demographics, earlier age of onset, and the increased prevalence of joint injury [9,38] and obesity [39]. The increasing burden of OA is worrisome for many reasons. At an individual level, the pain and mobility disability associated with knee and hip OA, which account for 83% and 17% of the OA burden, respectively [40], are a leading reason for becoming inactive with age [41]. This inactivity and associated obesity contribute to CV disease [42], reduced health-related QOL [43], and an increased risk of all-cause morbidity [44], with a recent individual patient meta-analysis demonstrating that symptomatic radiographic OA of the knee is associated with a 19% increased association with premature death independent of age, sex, and race compared to pain and radiographic OA-free controls [45]. At a societal level, OA is associated with increased health care use (including physician visits, medication use, and surgical procedures) [46,47], disability compensation/benefits [48], and losses to work force productivity [49]. Given the growing prevalence and burden of OA, it is essential that we shift our approach to management upstream and focus on prevention to reduce the burden of this disease [32].

## Prevention of osteoarthritis

Best evidence suggests that OA is amenable to prevention and early-stage treatment [32]. However, prevention of OA can take many forms. Strategies aimed at preventing or reducing risk factors in susceptible populations (e.g., populations prone to joint injury or obesity) are referred to as primary prevention. Strategies aimed at identifying and slowing down the onset of symptomatic OA in pre-clinical populations fall under the umbrella of secondary prevention, and strategies concerned with improving function and reducing disability in those with symptomatic OA are referred to as tertiary prevention. In the context of PTOA, primary prevention would refer to strategies aimed at preventing joint injuries in populations susceptible to joint injury (e.g., individuals who participate in sport and exercise), secondary prevention would refer to strategies aimed at delaying or halting the onset of symptomatic OA after joint injury, and tertiary prevention would refer to strategies aimed at improving function in those who have already developed symptomatic PTOA (Fig. 1).

Given that sport and exercise are leading causes of joint injury, individuals who suffer a sport or exercise-related joint injury represent an easily identifiable subset of “at risk” individuals to target with strategies aimed at preventing OA. Currently, there is high-level evidence guiding interventions aimed at preventing sport-related joint injuries (primary prevention) [50] and improving function/reducing disability (tertiary prevention) [51] in persons with symptomatic radiographic PTOA. Despite the progress that has been made in the primary and tertiary prevention of PTOA, there remain considerable gaps in our understanding of what can be done to delay or halt the onset of symptomatic PTOA after joint injury (secondary prevention) [52,53]. The reasons for these gaps include factors that make it difficult to execute

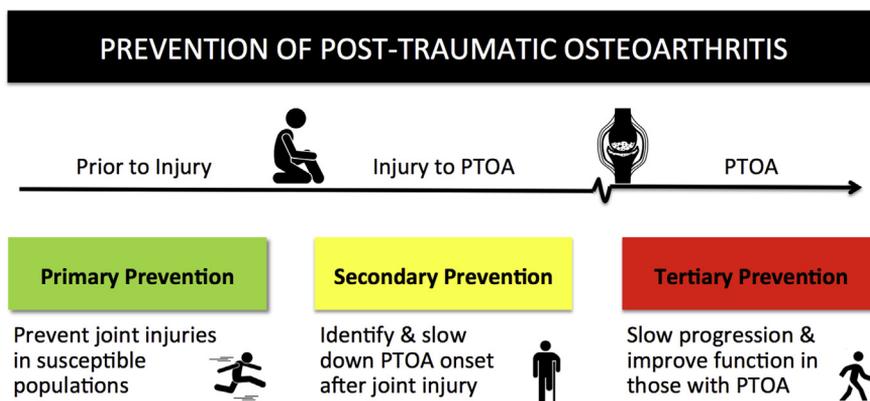


Fig. 1. A continuum for preventing symptomatic post-traumatic osteoarthritis.

high-quality randomized clinical trials (e.g., length of time between joint injury and PTOA onset); the lack of accepted definitions for early symptomatic and/or structural (i.e., MRI-defined or radiographic) PTOA; and heterogeneity in anatomical morphology, precipitating injury, treatment (e.g., access, and completeness of rehabilitation and surgical history), joint loading, and subsequent injury [54,55]. Despite these challenges, there has been considerable knowledge accumulated investigating post-traumatic populations in shorter windows of time since injury using a variety of clinical, functional, and structural surrogate (self-reported disability, MRI-defined OA, or QOL) or interim (i.e., re-injury or return-to-sport) outcomes. Some of these studies have focused on understanding the extent to which joint injury leads to symptomatic or structural OA and have emphasized nonmodifiable risk factors such as age, sex, and injury type. These studies have been essential to understanding the increased risk of developing symptomatic or structural OA after an ACL tear, ACLR, and combined injuries. Beyond investigating these nonmodifiable risk factors, attention has also been paid on numerous potential risk factors that may be amenable to intervention. Although the quality and level of supporting evidence vary across these potentially modifiable risk factors, there are some common threads that are consistent with clinical reports and what is known to be effective for primary and tertiary prevention of symptomatic OA. These parallels point to a risk profile for developing PTOA after injury and possible treatment targets.

### Secondary prevention of post-traumatic osteoarthritis

The prevention of OA after joint injury is contingent upon understanding who is at-risk (target population) and the availability of interventions capable of mitigating potentially modifiable risk factors (treatment targets). Although a variety of theoretically modifiable risk factors for symptomatic PTOA have been identified, there is limited evidence as to what extent they are modifiable, if modifying them is effective in delaying or halting the onset of the disease, and the associated costs [56]. However, given the considerable negative individual health-related outcomes and exponentially growing societal burden of OA, there is an urgent need for preventative efforts. The following sections will present a “risk profile” for symptomatic PTOA and suggestions for intervention strategies based on evidence synthesis. The content of these sections is heavily weighted on the findings of investigations in sport-related post-traumatic knee injury populations but may be broadly applicable to populations with traumatic hip and ankle injury. With that said, hip OA is less commonly associated with trauma and more likely to be attributed to accumulated load and/or morphological factors [57], while ankle OA is commonly associated with greater morbidity given an earlier age of onset and lower success rates with joint arthroplasty in comparison to knee OA [25].

#### *Risk factors for post-traumatic osteoarthritis after a sport-related joint injury*

For the purposes of informing targeted treatment to prevent symptomatic PTOA after a sport-related MSK joint injury, a “risk profile” for PTOA is best characterized by considering potentially

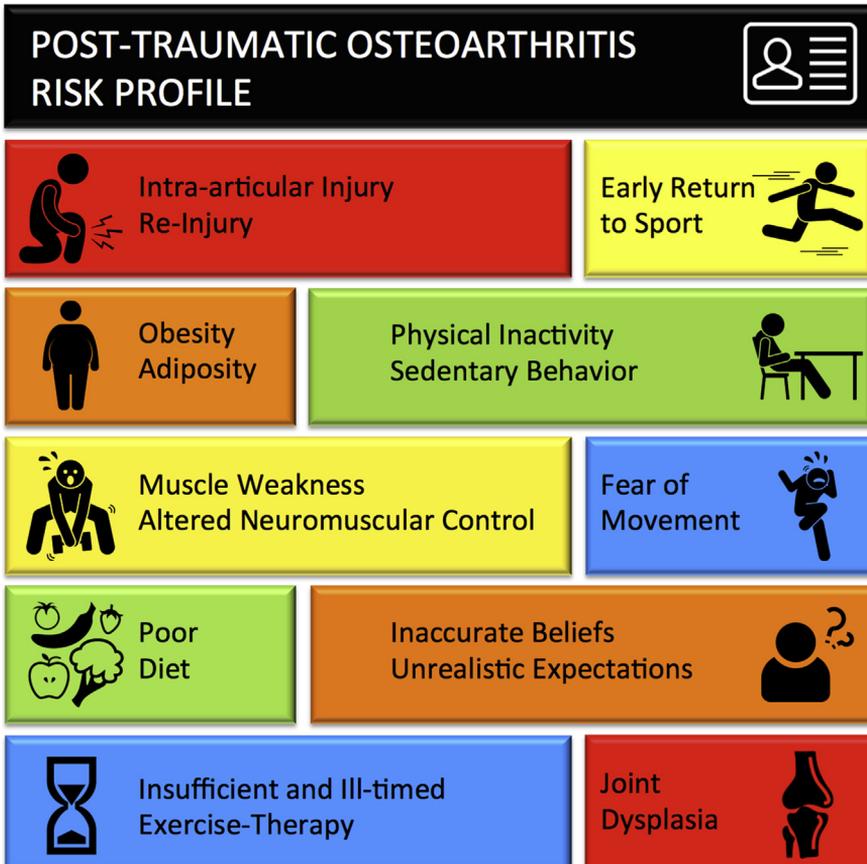


Fig. 2. A risk profile for post-traumatic osteoarthritis.

modifiable factors that have been associated with either increasing the risk of, or protecting against, PTOA (Fig. 2).

#### *Injury type and re-injury*

There is overwhelming evidence that those who suffer an intra-articular injury such as ACL, meniscal or labral tear, osteochondral lesion, or intra-articular fracture, particularly if in combination, are at high risk of radiographic OA [58]. Specifically, it is estimated that the relative risk of radiographic OA following an ACL tear is 3.89 (95% CI 2.72, 5.57) [23], that the odds of OA increase with concomitant injuries [59], and that ACL injury is associated with a 7-fold (OR 6.96, 95% CI 4.73, 10.31) increased odds of total knee arthroplasty compared to those in the general population [60].

Given that intra-articular injury is a risk factor for symptomatic OA, it is logical to hypothesize that reoccurrence of an intra-articular injury will similarly increase the risk for symptomatic PTOA. Although injury and subsequent re-injury are likely less of an issue in hip OA, it is estimated that 23% of individuals who undergo ACLR suffer a second tear [12] and 20% of individuals who sustain an ankle sprain will proceed to develop recurrent instability [61]. Further, re-injury after ACLR is associated with worse five-year outcomes [62]. There is a clear link between meeting return-to-sport criteria and risk of re-injury [63] as evidenced by the observation that athletes failing to meet return-to-sport criteria being at a fourfold (OR 4.32, 95% CI 1.0, 18.4) increased odds of suffering an ACL graft rupture than athletes that meet return-to-sport criteria [64]. Accordingly, it seems reasonable to assume that

individuals who return-to-sport after injury without meeting return-to-sport criteria are potentially at a higher risk for re-injury and subsequent symptomatic OA.

### *Obesity and adiposity*

Alongside joint injury, the most established risk factor for OA is obesity, which contributes to the development of symptomatic knee OA through both mechanical [16] and systemic [17] mechanisms. The lifetime risk of OA increases with increasing BMI with two-thirds of obese adults developing symptomatic radiographic OA [31]. Preliminary evidence suggests that young female athletes gain more fat mass over 1 year following ACL injury than uninjured teammates [65] and that by 3–10 years following a youth sport-related knee injury; young adults have a 2.4 (95% CI 1.2, 4.6) greater odds of being rated as overweight/obese by BMI and are 3.8 (95% CI 1.5, 9.8) times more likely to be in the upper quartile of total percent fat mass and have greater abdominal fat (mean within-pair difference; 0.46 kg, 95% CI 0.23, 0.69) than age-, sex-, and sport-matched uninjured controls [14]. This is concerning given that the association between BMI and OA is primarily mediated by fat mass [66] and suggests that in the context of joint injury, gain in adiposity is likely to increase the risk of future symptomatic OA.

### *Physical inactivity*

Although the link between physical activity and onset of OA in humans is not as well established as some of the above-mentioned risk factors, a recent cadaveric study has elegantly shown that lifestyle factors (e.g., including physical activity and possibly dietary habits) ubiquitous with the postindustrial era have contributed to a doubling of the prevalence of knee OA since the mid-20th century independent of body mass index and age [37]. In addition to this, approximately 8% of Australian youth drop out of recreational sporting activities after injury [67], and up to 20% of individuals who tear their ACL do not return to any sport (with 35% not returning to pre-injury levels of sport and 45% not returning to competitive sport) [11]. Individuals who undergo ACLR demonstrate less time in moderate-to-vigorous physical activity and have lower step counts than uninjured controls [13], and individuals who suffered a youth sport-related knee injury 3–10 years previously self-report being less physically active than uninjured matched controls [14]. Further, there is evidence that youth who suffer a sport-related injury may become disengaged from sport [68]. Given the clear associations between objective measures of physical activity (including sedentary behavior), energy expenditure, and adiposity [69], it is likely that physical inactivity is a risky behavior that may contribute to the development of symptomatic OA after joint injury.

### *Muscle weakness and altered neuromuscular control*

Based on meta-analyses of involving over 5000 participants, the odds of developing symptomatic radiographic OA was 1.65 (95% CI 1.23, 2.21) times greater for persons with weak knee extensors than strong extensors [70], and recent work from a tertiary prevention perspective has shown that knee extensor strength gains mediate pain and physical function improvements in persons with symptomatic radiographic OA of the knee [71]. In addition to the knee extensors, there is emerging evidence that knee flexor strength [72] and neuromuscular control of the quadriceps and hamstrings [73] may also be important in preventing re-injury. Finally, there is evidence that reduced functional performance (i.e., number of one-leg rises from sitting) is associated with an increased odds of radiographic knee OA five years later [74]. Accordingly, muscle weakness (particularly knee extensor weakness) [75] and poor performance on functional tasks should be considered a risk factor for symptomatic OA.

### *Fear of movement*

From a clinical perspective, there is emerging evidence that fewer symptoms and fear of movement within the first six months following ACLR are associated with better long-term outcomes [76]. Further, there is cross-sectional evidence that individuals diagnosed with symptomatic radiographic OA who have undergone ACLR in the past report poorer knee confidence and higher fear of movement than persons who have undergone ACLR and have not been diagnosed with symptomatic radiographic OA [77]. Consequently, it is possible that more symptoms and greater fear of movement will contribute to an elevated risk of OA. With that said, there is insufficient information to determine whether the

association between rehabilitation or early symptoms and OA is a result of injury severity (e.g., more severe injuries are associated with increased symptoms, kinesiophobia, and OA) or whether these associations are a result of how these factors impact physical activity levels [78].

#### *Inaccurate beliefs and unrealistic expectations*

There have been a series of recent investigations that point to other potential risk factors for PTOA associated with patients' expectations and beliefs related to return-to-sport, future risk of OA, how to interpret and manage flare-ups, and how to pace and/or modify their activity levels [10,79–81]. For example, a survey of patients undergoing ACLR revealed that 91% expected to return-to-sport at the same level by one year post-surgery when estimates are known to be closer to 63% [82], and only 2% thought that they had a risk for future symptomatic PTOA despite average estimates of 50% [79,83]. Beyond the unrealistic degrees of acceptance regarding the impact of the injury on sporting ability and future PTOA risk, individuals who suffer a sport or exercise-related knee injury are highly motivated to recover and despite meeting their injury with resilience many may not pace themselves well [81]. This disconnection between expectations and their real-world injury experiences likely contribute to an evolution in "athletic identity." [81] Although there have been no direct links between patient's knowledge and beliefs, and development of PTOA, there is evidence that unrealistic patient expectations can lead to negative outcomes [84]. Therefore, it is likely that a lack of knowledge and inappropriate beliefs may also have a place in a risk profile for PTOA.

#### *Poor diet*

An unbalanced macronutrient (i.e., carbohydrate, fat, and protein) intake combined with inadequate physical activity is associated with obesity and subsequent OA. Further, interventions incorporating energy-restricted diets that lead to reductions in body weight in the order of 10% have been longitudinally associated with decreased pain and increased function and in persons with knee OA [85]. In addition to macronutrient intake, certain micronutrients (i.e., calcium; vitamins C, D, E, and K; omega-3 fatty acids; and fiber) have been shown to play integral roles in joint and bone health and may mitigate the risk of symptomatic OA [86,87]. For example, a total dietary fiber intake of approximately 25 g/day has been shown to lower the risk of developing moderate or severe knee pain over time in a sample of 4470 individuals at risk of knee OA [88]. Further, there is evidence from the OA Initiative database showing a greater prevalence of symptomatic radiographic OA in persons with a high dietary inflammatory index, indicating a more pro-inflammatory diet [89]. Given the associations between dietary intake, the micro-inflammatory environment of the human body, obesity, and bone and joint health, it is plausible that an unbalanced and inadequate diet is a contributor to PTOA.

#### *Joint dysplasia*

Abnormal joint morphology has long been linked to pathological loading patterns that are believed to contribute to the development of OA with time [90]. Joint morphology is most relevant for the development of hip OA where morphological abnormalities seen with developmental dysplasia and femoroacetabular impingement have been associated with elevated risk of radiographic OA [57]. Potential methods to alter joint morphology and pathological loading patterns include surgery and exercise therapy interventions. With that said, high-quality clinical trials that determine the effectiveness of these approaches to protect against OA are needed. Despite a lack of evidence for effective means to address abnormal joint morphology, individuals who have suffered a sport-related joint injury that demonstrates abnormal joint morphology should be considered at increased risk of PTOA.

#### *Insufficient and ill-timed exercise therapy*

There is clinical evidence that prehabilitation [91] and early exercise therapy for those undergoing ACLR are associated with better two-to five-year function [62,92]. Although ACLR with or without meniscectomy (as applicable) is a standard of care for individuals who suffer a sport or exercise-related ACL tear and/or concomitant meniscal injury, it is currently unclear whether surgery aimed at addressing these lesions mitigates the elevated risk for OA [93], with 28–50% of individuals who undergo ACLR developing OA [94]. In contrast, there is compelling evidence that patients treated nonsurgically with supervised neuromuscular control exercise therapy and activity modification may

not progress to radiographic or symptomatic OA by 15 years following ACL injury [95]. Further, in the only high-quality randomized controlled trial comparing early exercise therapy (with a later option of ACLR) with early ACLR (followed by the same exercise therapy) for young adults with an acute ACL injury, Frobell and colleagues [96] demonstrated that patients assigned to early exercise therapy did not differ significantly in patient-reported or radiographic outcomes from those assigned to early ACLR. Although it is unknown what characteristics should be used to determine the order and timing of joint injury treatments to mitigate the onset of OA, clinicians and active patients should be aware that there are viable, and in some cases possibly superior, nonsurgical options.

#### *Risk profile for post-traumatic osteoarthritis*

In summary, a synthesis of the existing evidence suggests that individuals who suffer an intra-articular joint injury, exhibit greater adiposity, are less physically active, have strength deficits, or have abnormal joint morphology are likely at an elevated risk of PTOA after a sport or exercise-related knee injury. Further, there is evidence that insufficient prehabilitation, lack of completeness of rehabilitation prior to return-to-sport, unbalanced or inadequate nutrition, and unrealistic expectations and beliefs may contribute to this elevated risk. In the absence of high-quality clinical trials of prevention interventions, this “risk profile” for PTOA points to potential treatment targets and a pragmatic approach to prevention.

#### *Secondary prevention interventions*

Given the paucity of high-quality clinical trials to guide the prevention of symptomatic PTOA after joint injury [55] and the urgent need to reduce the growing burden of OA, it is prudent to propose guiding principles to pragmatically address treatment targets that are amenable to intervention. In addition to considering what is known about factors that increase the risk for PTOA following joint injury, guiding principles aimed at preventing symptomatic PTOA can benefit from considering what has been shown to be effective for preventing joint injuries (primary prevention) and optimizing function in persons with PTOA (tertiary prevention). Specifically, injury prevention programs consisting of running exercises combined with active stretching, controlled partner contacts, planting and cutting movements, and conditioning exercises that incorporate strength, ability and balance, alongside education that emphasizes movement (landing) patterns and fair play have been shown to significantly reduce sport-related lower extremity injuries [97]. Similarly, exercise programs focusing on neuromuscular control and strength alongside education that delivers key principles required for self-management have been shown to improve pain, QOL, physical function, and physical activity in persons with knee and hip OA [52]. Given the nature of the “risk profile” for PTOA and combined success of exercise therapy and education programs for primary and tertiary prevention of PTOA, it is very plausible that a similar approach would be appropriate for preventing symptomatic PTOA following a sport or exercise-related injury.

#### *Exercise program*

A key component of an exercise program for preventing PTOA after a sport or exercise-related joint injury is restoring, maintaining, or improving muscle function. In the case of knee PTOA quadriceps, muscles strength is vital. In addition to the quadriceps neuromuscular or strength training that incorporates exercises to optimize the strength and capacity of all the leg and trunk muscles is likely beneficial [98]. It is important to consider that strength alone is likely not sufficient, as it is critical that patients can perform functional tasks that are relevant and important to them, whether that be in the context of their lifestyle, sport, or occupation. A key component to the success of any exercise program is the need to address any existing fear of movement or re-injury anxiety through confronting provocative movements and myths regarding weight bearing and joint loading. Finally, it is critical that exercise therapy programs focused on delaying or preventing PTOA promote, or if possible, incorporate return to recommended daily levels of physical activity.

### *Education*

From an educational perspective, it is critical to provide patients with information that allows them to develop realistic expectations regarding return-to-sport, re-injury, and OA risk within the context of the severity of their injury. This will likely involve helping patients to let go of unrealistic expectations that they have picked up from other sources (i.e., social media, teammates, coaches, and healthcare practitioners not employing evidence-based treatments). Another important topic for patient education is helping patients to balance their needs for physical activity, rehabilitation, and sport while pacing and managing flare-ups. Additionally, it will be crucial to address how to avoid re-injury and the importance of return-to-sport criteria. If there is a need to modify a patient's physical activity or sport, it is essential to understand what their preferences (e.g., land-based, water-based, team, or sole endeavor sports) are to help them find an alternative that they do not become frustrated or disengaged with. Finally, there is likely a need for education related to weight management and diet. Targets might include balancing caloric intake with physical activity patterns, micronutrient supplementation, and minimizing pro-inflammatory food consumption.

### *Surgical and pharmaceutical interventions*

Despite attempts to optimize (reconstruct or remove) damaged tissues or joint morphology and mechanics with surgery, or the acute joint injury inflammatory response with pharmaceuticals, there is currently insufficient evidence to suggest that these interventions mitigate the risk of PTOA [55]. This is not to say that these interventions do not have a role to play in the management of patients with sport or exercise-related injuries; however, it is important to highlight that surgical interventions that take place soon after an acute injury may prolong the inflammatory response seen with trauma, which is, in some cases, associated with cartilage damage and bone remodeling [99]. Further, there is evidence that suggests that responses to early ACLR [62], meniscectomy [100], and arthroscopic hip surgery [57] are individual and are not always associated with favorable long-term outcomes. As different treatment approaches are associated with different prognoses, an individualized (personalized medicine) approach to acute joint treatment and PTOA prevention is recommended.

### *Philosophy and therapeutic alliance*

A vital component to any prevention strategy is the philosophy underlying a healthcare provider's approach to their patient. It is incumbent upon healthcare providers to have early conversations with patients adhering to the good practice sign posted by the Choosing Wisely campaign (see chapter 2). Success will likely hinge upon an approach that is based on co-management and a therapeutic alliance where healthcare providers are willing to have early difficult conversations related to rehabilitation, surgery, return-to-sport, activity modification, and long-term consequences while balancing the need for realistic expectations and over medicalizing the situation. Healthcare practitioners must be willing to challenge a default favoring of high-technological and invasive treatments and acknowledge exercise therapy as an evidence-based first-line treatment. This does not imply that exercise therapy will be the only treatment, as there will likely be adjuncts along the way including for example, modalities to relieve pain, manual therapy to increase range of motion and surgery to address recurrent instability. Considering the body of evidence implying the important role of exercise in prevention and management of joint injury and OA, and physical activity for general health, exercise should be an integral part of all programs aiming to prevent or manage joint injury and OA.

The best way to deal with any problem is to prevent it; hence, the earlier and more individualized the approach can be to minimize risk factors that compound the risk of PTOA (e.g., adiposity, inactivity, fear of movement, and nutrition) after injury, the better. Finally, it is critical that the practitioner, patient, and all other stakeholders understand that the long-term goal when treating acute sport or exercise-related joint injuries is lifelong musculoskeletal health, mobility, and good health-related quality of life, which may or may not involve return to preinjury sport.

### **Summary**

Sport and exercise-related joint injuries are associated with an elevated risk for PTOA and subsequent disability. OA is amenable to prevention and early-stage treatment. Despite the enormous and

expanding burden of OA, there is a paucity of high-quality evidence to assist clinicians in identifying who is at the greatest risk for PTOA following a sport or exercise-related joint injury or the most effective interventions for delaying or preventing PTOA. In the absence of this information, a thorough understanding of the factors and mechanisms that contribute to the elevated risk of symptomatic PTOA after a sport and exercise-related injury can inform a pragmatic approach to prevention. Based on what is currently known, individuals who suffer a sport or exercise-related joint injury that involves one or more intra-articular structures and present with abnormal joint morphology, elevated adiposity, weak musculature surrounding the injured joint, or have become physically inactive have an elevated risk for PTOA. This risk may be further elevated by insufficient exercise therapy or incomplete rehabilitation, premature return-to-sport and re-injury, unrealistic expectations and beliefs, or unbalanced or inadequate nutrition. Accordingly, delaying surgical interventions in lieu of exercise therapy aimed at optimizing muscle strength and neuromuscular control while addressing fear of movement to guarantee resumption of recommended levels of physical activity, completeness of rehabilitation prior to return-to-sport, education that promotes realistic expectations and self-management, and nutritional counseling are the best approaches for delaying or preventing PTOA.

### Practice points

- It is essential that lifelong musculoskeletal health, mobility, and health-related quality of life be the long-term goal when treating persons with sport or exercise-related joint injuries to curb the expanding individual and societal burden associated with osteoarthritis.
- Individuals who suffer a sport or exercise-related joint injury that involves intra-articular structures and who present with abnormal joint morphology, elevated adiposity, weak musculature or have become physically inactive have an elevated risk for PTOA.
- Insufficient exercise therapy and/or incomplete rehabilitation, premature return to sport, unrealistic expectations and beliefs, or unbalanced/inadequate diet may further elevate the risk for PTOA after a sport or exercise-related joint injury.
- Efforts to delay or prevent the onset of symptomatic PTOA after joint injury should include exercise therapy aimed at optimizing muscle strength and neuromuscular control while reducing fear of movement to guarantee resumption of recommended levels of physical activity.
- Patient education that promotes realistic expectations, importance of exercise and physical activity, self-management, and nutritional counseling will likely enhance prevention efforts.

### Research agenda

- Despite considerable research investigating sport and exercise-related post-traumatic injury populations, few risk factors for PTOA beyond joint injury and adiposity, or interventions aimed at delaying or preventing PTOA have been identified.
- Prior to addressing these knowledge gaps, efforts to seek consensus on the definition of injury and interim or surrogate outcomes, the natural course of potential risk factors in the period between joint injury and PTOA onset, potential treatment targets, and the therapeutic window for various interventions are needed.
- Given the multifactorial nature of PTOA, there is a need to align outcomes and combine datasets across research studies to ensure an adequate sample size to enable multivariable statistical analyses.
- Efforts to understand the implementation context for future PTOA prevention interventions are essential to facilitate uptake and effectiveness in otherwise healthy young populations who may be difficult to engage given the dynamic nature of their life stage.

## Conflict of interest

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