Clinical and Basic Science of Cartilage Injury and Arthritis in the Football (Soccer) Athlete

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Abstract

Joint injuries are very common in the athletic population, especially professional soccer players, with an incidence of 10 to 35.5 injuries per 1000 hours. Most soccer-related joint injuries occur in the lower extremities, with 16% to 46% occurring in the knee and 17% to 40% occurring in the ankle. Because of the limited healing capacity of cartilage and other intra-articular soft tissue structures, such as anterior cruciate ligament (ACL) and meniscus, joint injuries often lead to the development of early disabling osteoarthritis. Osteoarthritis in soccer players is 5 to 12 times more frequent than in the general population and diagnosed 4 to 5 years earlier. It remains a major cause of disability from this sport. This review focuses on the epidemiology of soccer-related joint injuries and subsequent development of osteoarthritis in the hip, knee, and ankle joints. As well, two different pathways for pathogenesis are described: (1) primary osteoarthritis via direct trauma to the articular cartilage and (2) secondary osteoarthritis that occurs indirectly through injury to the soft tissue structures that subsequently result in articular cartilage degeneration and loss.

Keywords

cartilage injury, osteoarthritis, sports injury, soccer, football

Soccer and Joint Injuries

Soccer is the most popular sport worldwide, with more than 240 million players overall, with 200,000 being professional/elite athletes.[1,2] However, as it is a high-speed contact sport with the highest intensity of joint impact and torsional loading, soccer is a sport that puts maximal demands on the joints.[3] There is an incidence of 10 to 35.5 injuries per 1000 training/playing hours,[2,4-6] with risk of injury during a match greater than that during training.[7-10] This injury incidence is around 1000 times higher than for high-risk industrial occupations.[10-14] Soccer, therefore, is estimated to be responsible for the majority of athletic injuries in Europe.[5] Participation at a professional/elite level and/or more game hours can further increase the risk and severity of injuries.[1,6,7,11,12,15-22] Most soccer injuries occur in the lower extremities (52%-95%), with the knee (16%-46%) and the ankle (17%-40%) being most prevalent.[1,5,9,16,18-20,23-28] The subsequent risk of early development of osteoarthritis (OA) is also expected to be high, and OA has been reported to be the major chronic injury suffered by professional soccer players.[29]

Epidemiology of Soccer and Osteoarthritis

The studies on OA and its relationship to soccer have been variable in terms of joint location, age groups, and outcome measures. Among former professional soccer players, 32% to 49% were medically diagnosed with OA,[14,17] with the knee being affected most frequently, followed by the ankle and hips.[16] The prevalence of OA is greater in players who had retired due to injury than those who had retired for other reasons.[16] Soccer players had an increased odds ratio of 2.10 for hospital admissions, likely for total joint arthroplasty, for OA of the hip, knee, and ankle.[17] Their mean age at first admission was also earlier at 56.2 years versus 61.2 years in controls. When the locations of the OA were separated, 8 incidences were for the hip, 5 for the knee, and 2 for the ankle. Hence, admissions for OA of the hip and knee were more common than for OA of the ankle.

Ankle Osteoarthritis in Soccer

Ankle sprains are common soccer injuries and are generally thought to be benign. However, studies report persistent signs and symptoms for months to years in 59% to 100% of

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injured soccer athletes.[30-32] Soccer was also the sport that most frequently gave rise to ankle injuries that caused post-traumatic ankle arthritis,[33] with one study quoting 33% of injured ankles developing radiographic signs of arthritis after 25 years.[15] This risk of developing arthritis was greater than that compared to the noninjured soccer players and age-matched general population controls. On the other hand, another study reported only a 1.6% overall incidence of ankle osteoarthrosis in soccer players, when the definition of ankle OA was strictly narrowed to be a loss of joint space and formation of new bone at the articular surface via radiography.[32]

Knee Osteoarthritis in Soccer

Among former professional soccer players, the radiographic signs of knee OA in soccer players increased with age in a greater percentage than in the general public.[34] Radiographic signs of knee arthritis were present in 15.5% to 63% of injured knees[11,15,34,35]; however, the rate of clinical symptoms was significantly less. The prevalence of radiographic signs of knee OA was also greater among elite soccer players compared to controls and nonelite players.[11] Thus, the authors concluded that soccer is associated with an increased risk of knee OA, especially at an elite level. Soccer players were also younger when they were diagnosed with knee OA. However, there was no difference in knee OA risk between the nonelite and controls when soccer players with known knee damage were excluded from the analysis.

Among patients with radiographic signs of knee OA, soccer was one of the sports that was significantly related to knee OA among men.[36] This increased risk of knee OA with soccer remained after adjusting for body mass index (BMI), heredity, smoking, and occupation but not after adjusting for previous knee injuries. Similarly, when patients undergoing total knee replacement were examined, there was an increased relative risk of severe knee OA among men aged between 55 and 65 years who were highly exposed to soccer.[37]

On the other hand, some studies showed no difference in the frequency of radiograph-confirmed knee OA between former elite soccer players and controls.[28,38] Paradoxically, subjects having symptomatic knee OA were more frequent in the control group than in the former soccer player group, despite the more severe radiological structural damages in the latter.[28]

Hip Osteoarthritis in Soccer

In addition to the ankle and the knee, many studies report a higher incidence of hip OA in former soccer players compared to age-matched controls.[12,38,39] The prevalence of hip OA between these two groups was significantly different, with an odds ratio of 10.2.[39] It was also determined that hip OA in former elite soccer players was three times more common compared to nonelite players.[12] Soccer players were diagnosed with hip OA at a younger age, similar to the knee OA findings. Among men who recently received a prosthesis due to severe idiopathic hip osteoarthrosis, there was an increased risk among men with medium and high exposure to soccer.[40] Nonetheless, soccer was not one of the most hazardous sports, which included track and field sports and racket sports.

Studies have variable findings, primarily due to the different methodologies used; the outcome definition and criteria were rarely consistent across studies. Structural outcomes, such as radiographic changes, do not correlate well with patient-relevant aspects, such as pain and function.[41] However, long-term soccer training/playing seems to increase the risk for early development of OA in the lower extremities, and this risk is higher in professional players.

Pathogenesis of Soccer-Related Osteoarthritis

The most common soccer injuries are sprains and strains affecting mainly the ankle and knee joints. A single game of soccer has not been shown to directly result in increased cartilage breakdown products[42]; however, soccer may increase the risk for OA in two different ways: (1) directly by high loading on the articular cartilage that leads to primary OA and (2) indirectly by damaging the extra-cartilage soft tissue structures within the joint that will subsequently lead to joint instability and secondary OA.[29] The intensity and duration of a sporting activity increase the occurrence of arthritic impairment.[28]

Primary Osteoarthritis

The articular cartilage surface can be damaged through sports participation via a single or repetitive impact or torsional loadings. The hip and the knee are the two main locations that may develop OA due to traumatic joint loading.[29,43] Direct acute trauma with an osteochondral impact may induce progressive cartilage damage and ultimately lead to OA.[28] Some studies that reported an increased risk of former elite soccer players to develop hip OA also postulated that compression of the joint surface and sudden shock to the joints can cause cartilage degeneration.[12,39]

Potential mechanisms for the rapid progression of OA after such an injury are extensively studied in both *in vitro* and *in vivo* settings, and studies have shown that matrix degradation, chondrocyte death, and/or abolishment of anabolic functions occur after impact to articular cartilage. Chondrocyte death leads to biochemical changes of the extracellular matrix and weakening of its biomechanical properties. *In vitro* studies on human cartilage explants have also shown significantly increased glycosaminoglycan (GAG) breakdown and release from the injured versus unloaded intact cartilages during 0 to

3 days postinjury.[44,45] Since the mechanical properties of articular cartilage are largely defined by the GAG content, GAG loss is both a sign and stimulator of deterioration of load-bearing cartilage function. Chondrocyte apoptosis rate in the human explants study also correlated with the GAG findings, with significantly higher apoptosis in the loaded (34%) versus control explants at 96 hours postinjury.[44]

Secondary Osteoarthritis

Indirect cartilage injury and subsequent arthritis development due to joint instability induced by damaging the extra-cartilage soft tissue structures that leads to chronic joint injury is a common soccer-related pathological process for the knee and ankle joints. Pathologic changes in all joint components, not just the cartilage, are an integral part of OA.

Ankle. When the mechanism of injury was investigated, 49.3% to 55% of all ankle sprains occurred during athletic activity, with soccer being one of the most common athletic activities.[33] Most ankle sprains involve the lateral ligament complex of the ankle joint.[15,30] Posttraumatic OA of the ankle accounts for more than 70% of ankle arthritis.[33] The overall mean latency time for the development of ligamentous posttraumatic ankle OA is 34.3 years, with a range of 6 to 57 years.[33] There was a significant difference between the patients with a single severe ankle sprain and with recurrent ankle sprains, with longer latency time with the latter group.

Knee. The knee anterior cruciate ligament (ACL) and menisci injuries are common in the general population and in soccer players. Studies report 24% to 40% and 21% to 31% of soccer injuries in the knee joint were ligamentous and meniscal injuries, respectively.[5,13,17,19,23] Injuries to the ACL and menisci are believed to be the main reason for the increased risk of knee OA in soccer players.[46] Both ACL and menisci injuries have been shown to be followed by arthritic changes of the knees.[17] In addition to the initial high force that damages intra-articular structures involved in the trauma,[11] the ACL and menisci disruptions initiate a cascade of pathogenic processes in the acute phase that can lead to the development of OA in itself.[41] There are also findings of greatly increased concentrations of markers of cartilage matrix metabolism in joint fluid in connection with trauma.[47] In addition to acute effects, the lack of a functionally normal ACL or meniscus leads to chronic changes in the static and dynamic loading of the knee and increased forces on the cartilage and other joint structures.

ACL injury and osteoarthritis in soccer players. The ACL is the most commonly disrupted knee ligament.[41,47] Nearly 40% of all soccer-related knee injuries are ACL injuries,[47] and a risk increase has been estimated at 100 to 1000 in professional soccer.[16,48] A complete ACL tear usually causes a long layoff from soccer and may even end the athletic career.[49] Soccer athletes sustain more ACL injuries than basketball players when compared by sex.[50] Nonetheless, isolated ACL ruptures are uncommon due to the high impact of the trauma, and it is often associated with injuries to other intra-articular structures. A meniscal injury most frequently accompanies an ACL injury: it is associated with 50% to 75% of all acute ACL cases.[3]

The mechanism responsible for cartilage degeneration following ACL injury is unclear. Biomechanical instability of the joint does not appear to be the only factor, and additional biochemical factors, such as inflammatory cytokines from synovitis, are also important. Dogs that underwent surgical transection of the ACL had significant elevation of cartilage breakdown products, collagenase-generated cleavage epitope of type II collagen, and cross-linked peptides from the C-telopeptide domain of type II collagen in their joint fluid at 3 and 12 weeks postoperatively.[51] The long-term clinical consequences of ACL rupture in soccer players have also been well studied: soccer players with an ACL tear are also more likely to develop knee OA than those players with intact ACL.[3] At 12 to 14 years after an ACL tear, 75% of soccer athletes had significant symptoms, and 41% to 77% had radiographic knee OA.[46,52] Additional meniscus injury requiring surgery was the most significant factor that may strongly influence the long-term symptoms and OA prevalence after ACL injury.[52]

The effect of ACL reconstruction on the subsequent development of knee OA has been controversial. Most studies report that the increased risk of development of OA following ACL injury remains unchanged regardless of the surgical ACL repair status.[41,46] When the clinical and radiographic outcomes of surgical repair or nonsurgical ACL treatments were compared, it was reported that an ACL reconstruction itself did not reduce the risk of OA.[53] ACL repair also did not increase the subjective symptomatic scores; however, it did decrease instability problems. In conclusion, although ACL reconstruction may facilitate restoring the kinematics of the joint, it does not make the knee normal.[41] There is a lack of evidence to support a protective role of ACL reconstruction surgery against OA, both in athletes and in the general population. Hence, the ACL-injured knee, whether it is repaired or not, will be subjected to abnormal loading over time, significantly increasing the risk of OA.

Meniscus injury and osteoarthritis in soccer players. Sports-related acute injuries to the menisci are common; however, the incidence of meniscus injuries in soccer is not as clearly described as the incidence of ACL injuries.[29] ACL injuries, which are common in soccer, are associated with a meniscus tear in up to 75% of cases.[3] As well, some investigators say that soccer is first and foremost a hazard to the menisci since a blow to the knee while weightbearing is more likely to cause damage to the menisci than to the ligaments.[19] In a study on former elite soccer players compared to age-matched controls, 14% of the former players had meniscal injuries resulting in meniscectomy versus 2% in the control group.[11] In another study, of the 33% of sports-related

meniscal injuries resulting in meniscectomy, 5% were soccer related.[54] Finally, in a study comparing meniscal tear patients to controls, meniscal tear and knee cartilage injuries were strongly associated with participation in sports, especially soccer, during the 12 months preceding the onset of symptoms.[55]

One of the treatments available for meniscal injury is total meniscectomy. However, this results in increased load stresses on the underlying articular cartilage and development of knee OA. The degree of OA has also been shown to be directly proportional to the amount of meniscus removed.[56] Hence, meniscal tears are a potent risk factor for the development of knee OA, regardless of the surgical status.[13] However, as stressed above, meniscus lesions combined with ACL tear are associated with the greatest risk of OA development.[15] In comparison to ACL patients, patients with isolated meniscus injury were about 10 years older when they had a comparable stage of OA.[47]

About 50% of meniscal injury patients with a partial or total meniscectomy have both symptomatic and radiographic signs of OA of the knee 5 to 20 years after injury.[47,56-59] This represents an odds ratio of about 10[41] and relative risk of 14.0[59] for the presence of the more advanced radiographic changes compared with an age- and sex-matched control group without known knee injury. Among former soccer players, all players who had a meniscectomy presented radiological signs of OA 10 to 20 years after surgery, compared to 40% in players who had not undergone meniscectomy.[34]

Conclusion

Joint injuries, including ligament, meniscal, and cartilage injuries, are common in sports, especially in soccer. These soccer-related injuries are most common in the lower extremities, involving the knee, hip, and ankle. Subsequent cartilage damage of the affected joints is due to primary joint impact and/or secondary to the extra-cartilage soft tissue injuries that lead to joint instability and degeneration of articular cartilage. Although primary osteoarthritis from joint impact is frequently responsible for the development of osteoarthritis in the hip and knee, secondary arthritis associated with ligamentous injury occurs primarily in the ankle and knee. The anterior cruciate ligament and menisci are the two intra-articular soft tissues that most often get injured during soccer and also play a critical role in OA development. Unfortunately, currently available treatment strategies, surgical or conservative, are inadequate to prevent the development of OA. Hence, there is a strong need for the development of molecular, biological, and mechanical interventions to delay or prevent the onset of postinjury OA.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interests with respect to the authorship and/or publication of this article.

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References

1. Engström B, Johansson C, Tornkvist H. Soccer injuries among elite female players. Am J Sports Med. 1991;19(4):372-5.
2. Junge A, Dvorak J. Soccer injuries: a review on incidence and prevention. Sports Med. 2004;34(13):929-38.
3. Neyret P, Donell ST, Dejour D, Dejour H. Partial meniscectomy and anterior cruciate ligament rupture in soccer players. Am J Sports Med. 1993;21(3):455-60.
4. Dvorak J, Junge A. Football injuries and physical symptoms. Am J Sports Med. 2000;28(Suppl 5):S3-S9.
5. Nielsen AB, Yde J. Epidemiology and traumatology of injuries in soccer. Am J Sports Med. 1989;17(6):803-7.
6. Keller CS, Noyes FR, Buncher CR. The medical aspects of soccer injury epidemiology. Am J Sports Med. 1987;15(3):230-7.
7. Ekstrand J, Waldén M, Hägglund M. Risk for injury when playing in a national football team. Scand J Med Sci Sports. 2004;14(1):34-8.
8. Waldén M, Hägglund M, Ekstrand J. Injuries in Swedish elite football—a prospective study on injury definitions, risk for injury and injury pattern during 2001. Scand J Med Sci Sports. 2005;15(2):118-25.
9. Morgan BE, Oberlander MA. An examination of injuries in major league soccer. Am J Sports Med. 2001;29(4):426-30.
10. Hawkins RD, Hulse MA, Wilkinson C, Hodson A, Gibson M. The association football medical research programme: an audit of injuries in professional football. Br J Sports Med. 2001;35(1):43-7.
11. Roos H, Lindberg H, Gärdsell 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):219-22.
12. Lindberg H, Roos H, Gärdsell P. Prevalence of coxarthrosis in former soccer players: 286 players compared with matched controls. Acta Orthopaedica. 1993;64(2):165-7.
13. Turner AP, Barlow JH, Heathcote-Elliott C. Long term health impact of playing professional football in the United Kingdom. Br J Sports Med. 2000;34(5):332-6.
14. Krajnc Z, Vogrin M, Rečnik G, Crnjac A, Drobnič M, Antolič V. Increased risk of knee injuries and osteoarthritis in the non-dominant leg of former professional football players. Wiener Klinische Wochenschrift. 2010;122:40-3.
15. Larsen E, Jensen PK, Jensen PR. Long-term outcome of knee and ankle injuries in elite football. Scand J Med Sci Sports. 1999;9(5):285-9.
16. Drawer S, Fuller CW. Propensity for osteoarthritis and lower limb joint pain in retired professional soccer players. Br J Sports Med. 2001;35(6):402-8.
17. Kujala UM, Kaprio J, Sarno S. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. BMJ. 1994;308(6923):231-4.

18. Poulsen TD, Freund KG, Madsen F, Sandvej K. Injuries in high-skilled and low-skilled soccer: a prospective study. Br J Sports Med. 1991;25(3):151-3.
19. Sandelin J, Santavirta S, Kiviluoto O. Acute soccer injuries in Finland in 1980. Br J Sports Med. 1985;19(1):30-3.
20. Roaas A, Nilsson S. Major injuries in Norwegian football. Br J Sports Med. 1979;13(1):3-5.
21. Peterson L, Junge A, Chomiak J, Graf-Baumann T, Dvorak J. Incidence of football injuries and complaints in different age groups and skill-level groups. Am J Sports Med. 2000;28(Suppl 5):S51-7.
22. Saxon L, Finch C, Bass S. Sports participation, sports injuries and osteoarthritis: implications for prevention. Sports Med. 1999;28(2):123-35.
23. Chomiak J, Junge A, Peterson L, Dvorak J. Severe injuries in football players. Am J Sports Med. 2000;28(suppl 5): S58-S68.
24. Maehlum S, Daljord OA. Football injuries in Oslo: a one-year study. Br J Sports Med. 1984;18(3):186-90.
25. McMaster WC, Walter M. Injuries in soccer. Am J Sports Med. 1978;6(6):354-7.
26. Giza E, Mithöfer K, Farrell L, Zarins B, Gill T. Injuries in women's professional soccer. Br J Sports Med. 2005;39(4):212-6.
27. Ekstrand J, Gillquist J. The avoidability of soccer injuries. Int J Sports Med. 1983;4(2):124-8.
28. Elleuch MH, Guermazi M, Mezghanni M, Ghroubi S, Fki H, Mefteh S, *et al*. Knee osteoarthritis in 50 former top-level footballers: a comparative (control group) study. Ann Readapt Med Phys. 2008;51(3):174-8.
29. Roos H. Are there long-term sequelae from soccer? Clin Sports Med. 1998;17(4):819-31.
30. Anandacoomarasamy A, Barnsley L. Long term outcomes of inversion ankle injuries. Br J Sports Med. 2005;39(3):e14.
31. Yeung MS, Chan KM, So CH, Yuan WY. An epidemiological survey on ankle sprain. Br J Sports Med. 1994;28(2):112-6.
32. Adams ID. Osteoarthrosis and sport. J R Soc Med. 1979;72(3): 185-7.
33. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous post-traumatic ankle osteoarthritis. Am J Sports Med. 2006;34(4):612-20.
34. Chantraine A. Knee joint in soccer players: osteoarthritis and axis deviation. Med Sci Sports Exerc. 1985;17(4):434-9.
35. Kujala UM, Kettunen J, Paananen H, Aalto T, Battié MC, Impivaara O, *et al*. Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. Arthritis Rheum. 1995;38(4):539-46.
36. Thelin N, Holmberg S, Thelin A. Knee injuries account for the sports-related increased risk of knee osteoarthritis. Scand J Med Sci Sports. 2006;16(5):329-33.
37. Sandmark H, Vingård E. Sports and risk for severe osteoarthrosis of the knee. Scand J Med Sci Sports. 1999;9(5):279-84.
38. Klünder KB, Rud B, Hansen J. Osteoarthritis of the hip and knee joint in retired football players. Acta Orthopaedica. 1980;51(1-6):925-7.
39. Shepard GJ, Banks AJ, Ryan WG. Ex-professional association footballers have an increased prevalence of osteoarthritis of the hip compared with age matched controls despite not having sustained notable hip injuries. Br J Sports Med. 2003;37(1):80-1.
40. Vingård E, Alfredsson L, Goldie I, Hogstedt C. Sports and osteoarthrosis of the hip. Am J Sports Med. 1993;21(2):195-200.
41. Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries. Am J Sports Med. 2007;35(10):1756-69.
42. Roos H, Dahlberg L, Hoerrner LA, Lark MW, Thonar EJMA, Shinmei M, *et al*. Markers of cartilage matrix metabolism in human joint fluid and serum: the effect of exercise. Osteoarthritis Cartilage. 1995;3(1):7-14.
43. Kettunen JA, Kujala UM, Räty H, Videman T, Sarna S, Impivaara O, *et al*. Factors associated with hip joint rotation in former elite athletes. Br J Sports Med. 2000;34(1):44-8.
44. D'Lima DD, Hashimoto S, Chen PC, Colwell CW, Lotz MK. Human chondrocyte apoptosis in response to mechanical injury. Osteoarthritis Cartilage. 2001;9(8):712-9.
45. D'Lima DD, Hashimoto S, Chen PC, Colwell CW, Lotz MK. Impact of mechanical trauma on matrix and cells. Clin Orthop Relat Res. 2001;391(Suppl):S90-9.
46. von Porat A, Roos EM, Roos H. High prevalence of osteoarthritis 14 years after an anterior cruciate ligament tear in male soccer players: a study of radiographic and patient relevant outcomes. Ann Rheum Dis. 2004;63(3):269-73.
47. 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):261-7.
48. Hawkins RD, Fuller CW. A prospective epidemiological study of injuries in four English professional football clubs. Br J Sports Med. 1999;33(3):196-203.
49. Waldén M, Hägglund M, Magnusson H, Ekstrand J. Anterior cruciate ligament injury in elite football: a prospective three-cohort study. Knee Surg Sports Traumatol Arthrosc. 2011;19(1):11-9.
50. Agel J, Arendt EA, Bershadsky B. Anterior cruciate ligament injury in National Collegiate Athletic Association basketball and soccer. Am J Sports Med. 2005;33(4):524-31.
51. Matyas JR, Atley L, Ionescu M, Eyre DR, Poole AR. Analysis of cartilage biomarkers in the early phases of canine experimental osteoarthritis. Arthritis Rheum. 2004;50(2):543-52.
52. Lohmander LS, Östenberg A, Englund M, Roos H. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. Arthritis Rheum. 2004;50(10):3145-52.
53. Meunier A, Odensten M, Good L. Long-term results after primary repair or non-surgical treatment of anterior cruciate ligament rupture: a randomized study with a 15-year follow-up. Scand J Med Sci Sports. 2007;17(3):230-7.
54. Baker BE, Peckham AC, Pupparo F, Sanborn JC. Review of meniscal injury and associated sports. Am J Sports Med. 1985;13(1):1-4.

55. Baker P, Coggon D, Reading I, Barrett D, McLaren M, Cooper C. Sports injury, occupational physical activity, joint laxity, and meniscal damage. J Rheumatol. 2002;29(3):557-63.
56. Englund M, Roos EM, Roos HP, Lohmander LS. Patient-relevant outcomes fourteen years after meniscectomy: influence of type of meniscal tear and size of resection. Rheumatology. 2001;40(6):631-9.
57. Englund M, Lohmander LS. Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy. Arthritis Rheum. 2004;50(9):2811-9.
58. Englund M, Roos EM, Lohmander LS. Impact of type of meniscal tear on radiographic and symptomatic knee osteoarthritis: a sixteen-year followup of meniscectomy with matched controls. Arthritis Rheum. 2003;48(8): 2178-87.
59. Roos H, Laurén M, Adalberth T, Roos EM, Jonsson K, Lohmander LS. Knee osteoarthritis after meniscectomy: prevalence of radiographic changes after twenty-one years, compared with matched controls. Arthritis Rheum. 1998;41(4):687-93.