On soft-tissue knee injuries: Epidemiology and Outcome

Isacsson, Anders

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LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00

On soft-tissue knee injuries

Epidemiology and Outcome

ANDERS ISACSSON
DEPARTMENT OF CLINICAL SCIENCES, LUND | FACULTY OF MEDICINE | LUND UNIVERSITY

This thesis on soft-tissue knee injuries springs from the clinical work at the Department of Orthopaedics in Helsingborg and the scientific questions that constantly arise in the management of patients with traumatic knee injuries. The general aims were to to investigate the array of injuries as well as long-term outcomes of acute soft-tissue knee trauma as visualised on sub-acute MRI in a large consecutive cohort of knee injured individuals. Further aims were to examine the epidemiology and outcomes of different injury patterns, with an in-depth analysis of first-time lateral patellar dislocations and their concomitant injuries.

ANDERS ISACSSON works as an orthopaedic surgeon at Helsingborg Hospital and received his qualifications as an orthopaedic specialist in 2009. His major clinical fields are sports-related injuries and general orthopaedic trauma. He is also active as the head of the course in orthopaedics at the Helsingborg branch of the faculty of medicine, Lund university.

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DOCTORAL DISSERTATION

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All patients with acute knee trauma and subsequent swelling presenting to the emergency department or orthopaedic out-patient clinic in a defined catchment area between 2002 and 2008 were consecutively enrolled in the study and examined with sub-acute magnetic resonance imaging (MRI). Radiographic signs of osteoarthritis (OA) and subjective outcomes were investigated at a median of twelve years (range, 8-17 years).

In paper one we presented incidences and described clinical data on different knee injuries and concomitant injury patterns, related to age, sex and activity at injury from the cohort of consecutive knee injuries assessed by subacute MRI (n=1145). About half of the knee sprains with hemarthrosis (52%) resulted in an anterior cruciate ligament (ACL) rupture with an incidence of 77 (95% CI 70-85) per 100 000 person-years, followed by meniscal tears (41%), medial collateral ligament injuries (28%), impaction fractures (21%), and lateral patellar dislocations (LPD) (17%). Ten percent of the knees with hemarthrosis did not show any signs of structural injury. Sports activity was the most common trauma mechanism (72%). LPD was the most common injury in children.

To investigate the epidemiology of traumatic first-time LPDs, we extracted these from the cohort (n= 175), in addition to nine patients who went straight to surgery during the study period. **In paper two**, we presented age- and sex-specific incidences of primary LPD and detailed concomitant chondral injuries including osteochondral fractures. Peak incidence of primary LPD was found at the ages of 13-15 years, both for boys (135/100 000) and girls (114/ 100 000), thereafter gradually declining with higher incidences for males than females over all ages except ages 10-12 years. A primary LPD resulted in chondral injuries in 43% of the knees and in 18% of the knees this was an osteochondral fracture.

A follow-up of the baseline cohort including bilateral radiographs and patient-reported outcome measures was conducted (n=814). **In paper three** we investigated the risk of developing radiographic and symptomatic OA after different types of soft-tissue knee injury and related the findings to the individuals' contralateral knee. One in three had radiographic OA in the affected knee whilst one in five in the contralateral knee. One in five was also considered to have symptomatic OA. An ACL rupture with a meniscal tear had the strongest correlation to radiographic OA with an incidence proportion of 50% (95% CI 44-56), three times the risk of the contralateral knee. Compared to patients with hemarthrosis alone, the risk of radiographic OA after an ACL rupture and meniscal tear was increased 10 times and the risk of symptomatic OA eight times.

To conclude, an ACL rupture was the most common injury with traumatic hemarthrosis, rarely isolated, and strongest correlated to both radiographic and symptomatic knee OA at long term follow-up. With a first-time LPD, nearly half will have chondral injury and one in five an osteochondral fracture.

Key words: Soft-tissue knee injuries, epidemiology, osteoarthritis, anterior cruciate ligament, patellar dislocation, meniscus, MRI.

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MADE IN SWEDEN

To my patients, my patient family, and friends

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List of scientific papers

This thesis is based on the following scientific papers

I. Olsson O, Isacsson A, Englund M, Frobell RB. Epidemiology of intra- and peri-articular structural injuries in traumatic knee joint hemarthrosis – data from 1145 consecutive knees with subacute MRI. Osteoarthritis Cartilage. 2016 Nov;24(11):1890-1897.

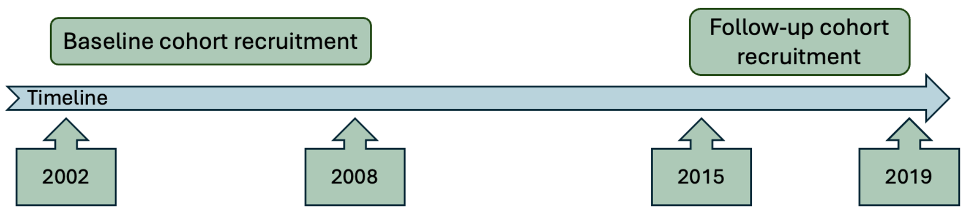
II. Isacsson A, Olsson O, Englund M, Frobell RB. Incidence and concomitant chondral injuries in a consecutive cohort of primary traumatic dislocations examined with sub-acute MRI. Int Orthop. 2023 Apr;47(4):973-981.

III. Isacsson A, Turkiewicz A, Olsson O, Brown JS, Hesslow J, Bengtsson P, Mahmoudian A, Frobell RB, Paemre J, Englund M. Long-term impact of acute knee injury with hemarthrosis: osteoarthritis incidence and patient-reported outcomes over 12 years. (Submitted)

Thesis at a glance

Paper	I	II	III
Aim	To obtain clinical data on different soft-tissue knee injury incidences and concomitant injury patterns related to age, sex, and activity at injury	To present age- and sex-specific incidences of primary patellar dislocations, and data on concomitant chondral injuries and osteochondral fractures	To estimate the 12-year incidence of radiographic and/or symptomatic knee osteoarthritis following MRI-confirmed soft-tissue knee injuries
Study period	2002-2008	2002-2008	2015-2019
Patients	1145 consecutive soft-tissue knee injuries with hemartrhosis in 1129 individuals assessed by subacute MRI	Sub-group analysis of 184 consecutive knees with primary patellar dislocations assessed by subacute MRI or arthroscopy	Long-term follow-up of 814/1129 (72%) consecutive patients at median 12 years after acute knee trauma with hemarthrosis assessed by plain radiographs and PROMs
Design	Prospective cohort study	Prospective cohort study	Prospective cohort study
Results	Serious soft-tissue knee injury occurred in 90% with traumatic hemarthrosis. Most injuries were sports-related (72%) and combination injuries (80%). ACL rupture was the most common (52%) injury, and LPD the most common in children (39%)	Highest incidence was found in those aged 13-15 years at 125 per 100 000 person-years. Higher incidences were seen in males across all age groups, except 10-12 years. 43% of the knees had chondral injuries where 18% were osteochondral fractures	Structural soft-tissue knee injury doubled the risk (RR 1.9 [95% CI 1.5-2.3]) of radiographic OA vs the other knee and depended on injury type. ACL rupture combined with meniscus tear conferred the highest risk and hemarthrosis alone the lowest (RR 10 [95% CI, 4-27])

MRI, magnetic resonance imaging. ACL, anterior cruciate lilgament. LPD, lateral patellar dislocation. OA, osteoarthritis, PROMs, patient-reported outcome measures. RR, risk ratio. CI, confidence interval



Abstract

Knee sprains with hemarthrosis are common, often sports-related, and with the potential to cause disability in the short as well as the long term. Clinical diagnosis of the freshly injured knee is unreliable and therefore the epidemiology in the population is not fully known. The aims of this thesis were to investigate the array of injuries after traumatic hemarthrosis of the knee, their epidemiology and long-term consequences.

All patients with acute knee trauma and subsequent swelling presenting to the emergency department or orthopaedic out-patient clinic in a defined catchment area between 2002 and 2008 were consecutively enrolled in the study and examined with sub-acute magnetic resonance imaging (MRI). Radiographic signs of osteoarthritis (OA) and subjective outcomes were investigated at a median of twelve years (range, 8-17 years).

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contralateral knee. Compared to patients with hemarthrosis alone, the risk of radiographic OA after an ACL rupture and meniscal tear was increased 10 times and the risk of symptomatic OA eight times.

To conclude, an ACL rupture was the most common injury with traumatic hemarthrosis, rarely isolated, and strongest correlated to both radiographic and symptomatic knee OA at long term follow-up. With a first-time LPD, nearly half will have chondral injury and one in five an osteochondral fracture.

Abbreviations

ACL	Anterior Cruciate Ligament
AE	Athletic Exposure
AKP	The Acute Knee Project
BML	Bone Marrow Lesion
ED	Emergency Department
ESSKA	European Society for Sports Traumatology, Knee Surgery and Arthroscopy
ICD-10	International Classification of Diseases, Tenth Revision
KANON	Knee Anterior Cruciate Ligament, Nonsurgical versus Surgical Treatment study
KL	Kellgren and Lawrence
LCL	Lateral Collateral Ligament
LPD	Lateral Patellar Dislocation
MCL	Medial Collateral Ligament
MPFL	Medial Patellofemoral Ligament
MRI	Magnetic Resonance Imaging
OA	Osteoarthritis
OARSI	Osteoarthritis Research Society International
OC	Osteochondral
OCF	Osteochondral fracture
PCL	Posterior Cruciate Ligament
PF	Patellofemoral
PROMs	Patient-Reported Outcome Measures
RCT	Randomised Controlled Trial
RR	Risk ratio
TF	Tibiofemoral
TKA	Total Knee Arthroplasty

Background

> "Because it's there"
>
> George Mallory

The true natural history of soft-tissue knee injuries is elusive with a scarcity of older reports without intervention. Virtually all modern accounts also involve some kind of intervention. That is also the case in our study. In 1938, Ivar Palmer wrote about the natural history of anterior cruciate ligament injuries "The critical study still remains to be done, i.e. a prospective analysis of anterior cruciate insufficiency and its sequelae."[1] and many hold that true still today.[2]

Knee sprains with soft-tissue injury are common, especially in the young and active population,[3] often sports-related, and burdened with substantial disability in the short and long term.[4-14] The presence of knee-effusion in the acute phase after knee trauma is indicative of hemarthrosis and significant intra-articular injury.[15, 16]

Because of the immediate pain and discomfort with acute hemarthrosis and the anxiety over the risk of not being able to return to play, normal life or work, the afflicted person is probable to seek the attention of a health-care facility or the medical team for advice and treatment. Every unique knee problem and unique individual is likely to need different, individualised, therapy. As with all problems, it is imperative to thoroughly know its nature and potential long-term effects to solve it. The solution should normally be as safe, cost-effective and patient-centred as possible. An accurate diagnosis is the foundation for further management of the injury.

Different settings offer different possibilities to examine and diagnose a sprained knee. Clinical diagnosis of the traumatised knee joint is unreliable in the acute setting,[17-19] although it can be improved when performed by a specialist skilled in knee examinations or with arthroscopically assisted diagnostics in combination with examination under anesthesia.[15, 20-22] The use of magnetic resonance imaging (MRI) after a traumatic knee injury can increase the diagnostic precision and may be considered a cost-effective gold standard for assessing intra- and peri-articular knee injuries with the benefits of being non-invasive, re-interpretable and maybe more readily at hand than an orthopedic knee subspecialist.[22-28] Even an expert in the field is likely to refer the patient for an MRI to pinpoint the diagnosis.[29]

Timely treatment of soft-tissue knee injuries should be based on the specific injury and can involve surgery, bracing or good advice, most often accompanied with structured physiotherapy. Top level athletes may have teams of physiotherapists, doctors and other rehabilitation facilities available where resources like MRI or arthroscopy are not far away. The general population may not enjoy the same work up in many societies. In Sweden, the health-care system aims to care for every citizen equally, making it possible to set up a similar clinical routine for knee trauma care as the one provided for the sports community.

Post-traumatic osteoarthritis (OA) is the major concern in the long term after traumatic knee injury. Traumatic knee injury is one of the main predisposing factors for knee OA.[30]

Given the difficulties to correctly determine the specific injury pattern in a freshly sprained knee, many studies of specific injuries can be suspected to be biased by missed and non-representative cases. We examined every patient with a knee sprain and subsequent hemarthrosis, top athlete, child or ordinary person with an MRI was within a couple of days of presentation to the emergency department or the out-patient knee clinic. Team-based advice was given to the patient within another couple of days. The consecutive patients between 2002 and 2008 formed the cohort of this project, the acute knee project (AKP), and were followed up after a median of 12 years. This thesis describes which crucial structures that are likely to be injured with a knee sprain, their respective incidences and the long-term outcomes regarding radiographic and symptomatic knee OA, as well as knee-related health.

Anatomy of the knee

Anatomy follows function and vice versa. The various functions of the knee stem from a complex interaction between its bony and soft-tissue components. The bones involved are the femur, tibia, fibula, and patella. Soft-tissue structures include the cartilage, menisci, ligaments, tendons, muscles, and joint capsule (Figure 1).

The knee is largely a hinge joint allowing the proximal tibia to flex and extend against the distal femur with the patella acting as a fulcrum. It is also referred to as a trochoginglymos, a gliding hinge joint.[31] The femur and tibia articulate against each other in the tibiofemoral (TF) joint consisting of the medial and the lateral TF compartments. The patella articulates against the anterodistal part of the femur, the trochlea, in the patellofemoral (PF) joint. The articulating surfaces are largely covered with cartilage.

The articular cartilage allows painless movement of joints with minimal friction but also load transmission and absorption. It is lubricated and nourished by the synovial fluid. Contrary to most other tissues, there are no blood vessels or nerves in articular cartilage. The hyaline cartilage layer is approximately 2.4 mm thick on the medial femur and is composed of a dense extracellular matrix of mainly water, collagen, and proteoglycans, with a few percent of chondrocytes.[32] The patella has the thickest cartilage in the body of up to 7 mm, with thicker cartilage in males than females.[33] The chondrocytes are essential for the development, maintenance, and repair of the extracellular matrix.[34] Articular cartilage has a limited capacity for intrinsic healing and repair. The preservation and health of articular cartilage are consequently most important for retaining joint function.

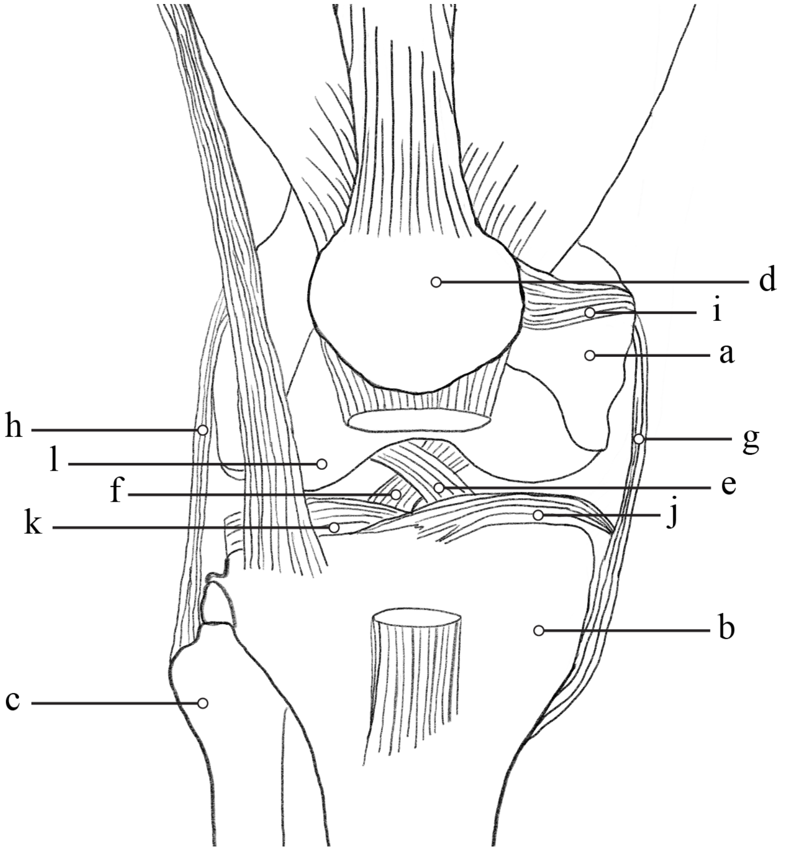

Figure 1. Outlines of the knee anatomy. Right knee.
Osseous components: femur (a), tibia (b), fibula (c), patella (d). Soft-tissue components: anterior cruciate ligament (e), posterior cruciate ligament (f), medial collateral ligament (g), lateral collateral ligament (h), medial patellofemoral ligament (i), medial meniscus (j), lateral meniscus (k), and articular cartilage (l). Illustration by Jens Strandberg.

The patellofemoral joint is inherently congruent, while the morphology of the femur and the tibia forms an incongruent joint. The menisci act as inlays to even the surfaces and transform the flat or somewhat convex surface of the tibia into a concave surface that matches the convex surface of the femur. Their role is to minimise articular cartilage contact pressures with 40-60% of knee joint load being transmitted through the menisci.[35] With increasing flexion, the rate of load through the menisci also increases. In addition, they function as secondary stabilisers of the knee. The menisci, formerly referred to as the semilunate cartilage, are mostly attached to the capsule across the outer rims and at both ends to the anterior and posterior tibia via the roots. [36] The root attachments are essential as they anchor the menisci and convert axial loads into hoop stresses and prevent extrusion while under load. The medial meniscus is larger and covers approximately 60% of the medial tibial cartilage, while the slightly smaller lateral meniscus covers around 80% of the lateral tibial cartilage surface.[37] The menisci are also interconnected with the anterior intermeniscal ligament. The medial meniscus is more firmly connected to the capsule in addition to being connected to the medial collateral ligament (MCL), making it less mobile and more prone to injury than the lateral meniscus. The lateral meniscus is connected to the femur posteriorly through the anterior meniscofemoral ligament of Humphrey and the posterior meniscofemoral ligament of Wrisberg in most individuals.[38, 39] Neural endings have been noted in proximity to the meniscal attachments making it plausible that they contribute to proprioception [40]. Although only the outer 10-25% of the menisci has been reported to have vascular supply, recent findings imply that some vascular vessels also can be found in the inner areas of the tissue.[41, 42]

While the main kinematic principles of the knee joint are rolling, gliding and rotation, it offers in total six degrees of movement. Rotational movement in flexion/extension, varus/valgus and internal/external, translational movement in anterior/posterior and medial/lateral as well as compression/distraction. The ligaments provide passive stability in all directions of movement except compression.[43]

The anterior cruciate ligament (ACL) is the principal passive stabiliser against forward movement of the tibia to the femur and the posterior cruciate ligament (PCL) against backward movement. The ACL runs from the posterior part of the medial side of the lateral femoral condyle to an area in front and lateral to the anterior tibial spine where it blends with fibres from the anterior root of the lateral meniscus.[44] The ACL consists of two bundles, named after their insertion on the tibia: the posterolateral and the anteromedial bundles. The mean length of the ACL is around 27-38 mm.[45] The PCL, which has about the same length as the ACL, origins from the lateral side of the medial femoral condyle and attaches to a depression on the proximal posterior tibia, behind the intraarticular part of the tibia.[44] It is broader and stronger than the ACL and likewise consists of two bundles, the anterolateral and posteromedial.[46] The main passive stabiliser against varus force is the lateral collateral ligament (LCL) and against valgus force the MCL.[47, 48] In addition, stability is delivered from the surrounding envelope of the quadriceps and hamstrings tendons, the capsule, and other ligamentous structures, such as the anterolateral ligament.

Paul Segond reported in 1879 of an avulsion fracture at the anterolateral proximal tibia as a remarkably constant result of forced internal rotation of the tibia and attached to this a "pearly, resistant, fibrous band which invariably showed extreme amounts of tension during forced internal rotation (of the knee)"[49]. This Segond-fracture has later, and after the discovery of X-rays, become regarded as a pathognomonic sign of an ACL rupture.[50-52] The pearly band is now known as the anterolateral ligament, a secondary stabiliser to the ACL which helps resist tibial internal rotation and anterior tibial translation.[53, 54]

The primary dynamic stabiliser of the patella is the vastus medialis obliquus muscle and the main static stabiliser when not engaged in the femoral trochlea, is the medial patellofemoral ligament (MPFL).[55]

Historical background

Hippocrates (460-370 BC) may have been the first to describe soft-tissue knee injuries.[56] In translation by Francis Adams: "Owing to their configuration, the bones at the knee indeed frequently dislocated, but they are easily reduced, for no great inflammation follows, nor any constriction of the joint. They are displaced for the most part to the inside, sometimes to the outside, and occasionally into the ham." Probably, he was referring to a recurrent lateral patellar dislocation (LPD) and continues by describing the management: "Moderate extension for the most part sufficeth, extension being made at the leg, and counter-extension at the thigh."

Claudius Galenus (129-216 AD), also known as Galen of Pergamon, has been credited with the first description of the cruciate ligaments that he named 'ligamenta genu cruciata'.[57] He was a physician and philosopher building on, among others, Hippocrates' works. Galen has been attributed as the first sports physician, serving as the physician to the gladiators in Pergamon, but also as a physiotherapist that described exercise recommendations for rehabilitation.[58] However, there is competition in who is to be called the father of sports medicine and others have also been given this title. Herodicus, a physical educator and physician born in the fifth century BC and one of Hippocrates' teachers, combined sports and medicine at the medical school of Cnidos. He emphasised the relationship between physical activity and good health and is suggested to be the first physician to advice patients to start moving after an injury, much preceding what is common practice today.[59]

There was a long pause in history regarding further written accounts on knee anatomy until the Renaissance, when Andreas Vesalius in 1543 published ‘De Humani Corporis Fabrica’. These were the first results from anatomical dissections since Galen and the ancient Greeks. Another 300 years passed before the next known publication on knee anatomy arrived in 1836, when the Weber brothers described the biomechanics of knee joint motion, the two bundles of the ACL, and demonstrated that through sectioning of these bundles, an abnormal antero-posterior movement was produced.[60] Amédée Bonnet (1809-1858) of Lyon published his ‘Traité des maladies des articulation’ in 1845 where he described the signs of an ACL rupture and suggested conservative management with early rehabilitation to preserve the cartilage. Since then, the interest in the soft tissues of the knee has not failed.

Scottish surgeon Thomas Annandale is credited with the first meniscus repair in 1883, and a few years later with the first meniscectomy. English surgeon Arthur Mayo-Robson performed the first repair of both cruciate ligaments in 1895, more than 2000 years after the description by Galen. A Swedish surgeon, Knut Harald Giertz was the first to reconstruct the collateral ligaments on a 13-year-old girl using a fascia lata autograft. Another big step was taken when Watanabe performed the first arthroscopic meniscectomy in 1962.[61]

Soft tissue knee injuries

The injury mechanisms for different soft-tissue knee injuries are complex and continually debated, even though video analyses and analyses of post-traumatic MRI have added a lot of information to biomechanical studies on cadavers.[62-64] In LPD, movement from extension to flexion is the most common mechanism, but one tenth has been shown to occur in movement from flexion to extension.[65] Athletes, having invested a lot of time and energy into their sport, are at greater risk of knee injury than others.[66]

With the general population being more active and involved in sports activity, the already common soft-tissue knee injuries are increasingly prevalent, especially in females.[3, 67-71] The major structural injuries are cruciate ligament ruptures, collateral ligament ruptures, meniscal tears, articular cartilage injuries, and LPD.[70] All of these can occur in solitude or in combinations. Traumatic knee hemarthrosis is strongly suggestive of significant structural injury.[72] Rotational knee trauma in combination with a rapid effusion is a good indicator of soft-tissue injury even if the clinical examination does not reveal instability, as shown in multiple studies on arthroscopy in conjunction to acute traumatic hemarthrosis of the knee [15, 20, 24, 73].

There is an ongoing discussion whether to aspirate a traumatic hemarthrosis in every case or not. Both knee pain and effusion are correlated to quadriceps arthrogenic muscle inhibition and weakness.[74]. In addition to the short-term discomfort of a traumatic effusion, there may be harmful effects of proinflammatory and paracrine substances associated with chondrotoxicity, that potentially could contribute to posttraumatic OA.[75, 76] However, aspiration may also remove factors that are potentially beneficial to the healing process.[77] In a study on arthrofibrosis after ACL reconstructions, an aspiration post trauma seemed to delay the onset of arthrofibrosis.[78] Given what is known today, to evacuate a traumatic hemarthrosis to ease pain and increase range of motion should be considered but is not regarded as mandatory. However, to identify a potential traumatic hemarthrosis is of great importance.

Epidemiology

Acute knee injuries have been estimated to represent 8% of all injuries presenting to the ED in a Swedish population.[69] Seventy-five percent of these were classified as sprains/strains, dislocations or contusions, and hence with potential soft-tissue injury. Incidences of knee injuries in general, and ACL ruptures as well as LPD in particular, have been studied in a variety of populations [3, 71, 79-89]. However, most of these reports are based on clinically diagnosed injuries rather than MRI-derived diagnostics with the possible risk of under-estimates. There appears to be a need for real-life clinical data from large prospectively collected cohorts with a minimum of diagnostic inaccuracy to confirm previous reports.[19]

Diagnostics

In acute knee injury, plain radiographs are often indicated as the primary imaging evaluation of the knee.[90] Plain radiographs can rule out fractures, identify an intraarticular effusion, evaluate pre-existing OA, and determine the bone morphology. An avulsion of the tibial attachment of the ACL, most common in the paediatric population, can be noted as a tibial eminence fracture, and a Segond fracture as a pathognomonic sign of ACL injury. A posterolateral fracture on tibia can also be a sign of a pivoting trauma and a possible ACL rupture. Other indirect signs of an ACL rupture are the arcuate sign, that represents an avulsion fracture of the arcuate ligament from the proximal fibula, and the deep lateral femoral notch sign, representing an impaction fracture. With an LPD, osteochondral fractures can be present, often in the lateral recess. In most cases, plain radiographs are normal, except for signs of an effusion and an MRI should be indicated.[29] At longer-term, weight-bearing radiographs are needed for the evaluation of post-traumatic radiographic OA.

In soft-tissue knee injuries, MRI is the most helpful non-invasive diagnostic tool. A variability of diagnostic accuracy has been reported, between 80 and 100%, but there is a continuous improvement of MRI protocols and field-strength. With good kappa agreements (0.43 and 0.45) for both menisci and excellent (0.84) for ACL ruptures with routine MRI and arthroscopy, MRI should be considered the primary tool of diagnostics in these injuries.[91] In LPD, MRI seems to be superior to other modalities in detecting osteochondral fractures.[92]

Outcome

The natural history of the injured knee is not yet fully understood. In many cases, an injury will result in a knee that will not allow the same activity level that the individual was used to before the injury and with a decline in perceived quality of life.[93] This can be due to knee joint instability and/or the development of post-traumatic knee OA. Differences in tibia position, bone morphology, presence of effusion and synovitis, and cartilage/subchondral bone composition can be detected as early as 12 months after an ACL injury.[94] A knee injury predisposes the knee to an early onset of OA.[30] However, different injuries come with different risk profiles. An ACL rupture could be a mild injury, and sometimes even heal with excellent results.[95] Other injuries at the time of trauma may confer worse effects for the long-term outcome, ie meniscal tears, cartilage injury or osteochondral fractures.[4, 96]

Altered biomechanics in ACL deficient knees can lead to cartilage wear and possibly OA.[97-99] Longer time from the ACL injury, and delayed ACL reconstructions have been correlated to increased likelihood of cartilage and meniscus damage.[100-102] On the other hand, reconstruction of the ACL has not convincingly been shown to prevent the progression of OA or patient-reported outcomes, suggesting that joint instability alone is not solely responsible for the increased risk of OA.[8, 103-105]

Osteoarthritis

The symptoms of OA are pain, morning stiffness, and loss of function. Osteoarthritis has been around since the dawn of times. Typical joint osteoarthritic joint changes have been identified even in dinosaurs living millions of years ago, as well as in Egyptian mummies.[106, 107] Articular cartilage changes with age, and the hydration of the extracellular matrix decreases which makes the cartilage stiffer and potentially less capable of absorbing compressive forces, which can lead to OA.[34] An injury to the knee joint largely increases the risk of OA.[108] Osteoarthritis is a major global cause of disability and is expected to increase.[109] Paradoxically, two major reasons for this are increased participation in athletic activities as well as increased obesity.[110]

A consolation for younger athletes could be that sustaining a major knee injury at older age is related to more rapid development of OA compared to those having a knee injury at younger age.[97] However, in a study of female elite soccer players a higher prevalence of OA signs (11%) was observed even without known injuries at the mean age of 55 years compared to non-elite players and controls, that had the same prevalence in the absence of former injury. Also, for the nonelite players with a known injury 13% had OA vs 3% without injury.[111]

Rationale

To improve care for the patient with a serious soft-tissue knee injury, it is important to know as much as possible about the injury panorama and the natural course of the injury at hand. A soft-tissue knee injury is potentially disastrous for a young person in the beginning of a sports career but is likely very cumbersome also for an older active individual. The knee injury puts a sudden halt to an active lifestyle and, depending on the injury, it can take a long time before a player is back on the field, or a leisure skier is back on the slopes. The long-term implication of a knee injury is something that is likely to occupy the mind of the carer as well as the patient.

A recent meta-analysis focusing on risk factors for knee OA after traumatic knee injuries stresses the need for studies with a broader perspective on knee-injuries than ACL tears, to better understand which forces are in play.[112] Furthermore, there is a paucity of prospective studies with high follow-up rates studying the outcome after knee injury.[30] One of the most important unanswered questions for future research priorities in soft-tissue knee injuries has been identified as gaining further knowledge regarding outcome after acute knee injury.[113]

This project provided a large funnel for all traumatic knee injuries with hemarthrosis and addresses the limitations of clinical diagnostics in clinical practice. Due to post-traumatic pain and swelling, most individuals are likely to seek medical care after a knee sprain with hemarthrosis. Therefore, there was a good chance of including a representative sample of knee injuries in the broad perspective, which allows for a detailed investigation of the included injuries and their outcomes.

Aims

General aims of the thesis

The aims of this project were to investigate the array of injuries as well as long-term outcome of acute soft-tissue knee trauma as visualised on sub-acute MRI in a large consecutive cohort of knee injured individuals. Further aims were to examine the epidemiology of different injury patterns, with an in-depth analysis of first-time lateral patellar dislocations and their concomitant injuries.

Specific aims

Paper I – To obtain clinical data on different knee injury incidences and concomitant injury patterns related to age, sex, and activity at injury from a consecutive cohort of 1145 knee injuries assessed by subacute MRI.

Paper II – To present age- and sex-specific cumulative annual incidences of primary LPD and to detail patient characteristics together with concomitant chondral injuries including osteochondral fractures, as visualised on MRI.

Paper III - To estimate the risk of developing radiographic and symptomatic knee OA at a median of 12 years after traumatic soft-tissue knee injury with hemarthrosis, to determine the self-reported knee health and patient satisfaction, and further to compare groups with different knee injuries within the cohort as well as relate the findings to the individuals' contralateral knee.

Methods

> "Let us not overlook the further great fact, that not only does science underlie sculpture, painting, music, poetry, but that science is itself poetic."
>
> Herbert Spencer

This study is based on a prospective cohort of consecutive soft-tissue knee injuries from a defined catchment area. The study did not involve any interventions. However, a subset of the subjects (n=107/1145) was also involved in a randomised controlled trial where ACL injured individuals were randomised to either structured rehabilitation and sub-acute ACL reconstruction or structured rehabilitation and optional ACL reconstruction later, the Knee Anterior Cruciate Ligament, Nonsurgical versus Surgical Treatment (KANON) trial.[114, 115] The KANON study recruited patients from two centres, Helsingborg hospital and Lund University hospital and the subjects from the defined region of this trial (n=107/121) were part of this cohort for completeness. The general methodology in this thesis is descriptive with the intention of presenting as comprehensive data as possible.

Design of the cohort

In the year 2000 a new clinical routine, the Acute Knee Project (AKP) was introduced at the Hospital of Helsingborg. A study project with the aim to identify, diagnose with sub-acute MRI and set up a management plan for every significant traumatic soft-tissue knee injury in the population.

This is a general hospital, located in the south of Sweden, that provides medical services to the adult as well as the pediatric population within a well-defined catchment area. The population is mainly urban but also includes rural inhabitants. Official population statistics of Sweden, held by Statistics Sweden, provides detailed retrospective open access population data. At the introduction of the project in 2002, the number of inhabitants in the catchment area was 155 870. As the orthopaedic emergency care at the nearby Ängelholm Hospital, at the time serving a population of 81 549, merged with Helsingborg Hospital in October 2004 the population of the catchment area expanded. By the end of 2007 Helsingborg Hospital served a population of 246 999 inhabitants.

The emergency and orthopaedic departments in Helsingborg were the only facilities handling acute knee trauma, initially in the smaller catchment area and from 2007 in the larger catchment area, during the study period. The orthopaedic out-patient clinic was also the only referral center for adult and pediatric trauma or sports injuries. A specialist knee clinic within the orthopaedic department was set up to manage referrals from primary care as well as patients triaged from the emergency department (ED). Dedicated radiology rounds were also set up to run twice weekly where all knee MR images from the AKP were demonstrated by experienced musculoskeletal radiologists with special interest in knee MRI. The knee specialists and research nurse at the orthopaedic department were scheduled to attend these rounds.

When the routine was introduced, primary care physicians and physiotherapists in the area were informed and encouraged to refer all patients with acute knee trauma to the hospital. Written information about the study project and how referrals should be made was distributed to the relevant primary care physicians, physiotherapists and emergency departments. The radiologists were instructed to note in the report if an effusion was seen on plain radiographs and, when noted, recommend a referral according to the study protocol.

The baseline cohort (Papers I-III)

By January 2002, the routine was considered as fully implemented and well known among healthcare professionals who could get in contact with patients after knee trauma in the area.

Inclusion criteria

All patients with acute knee trauma and hemarthrosis defined as a puncture-verified hemarthrosis or an acute intra-articular swelling within 24 hours who presented to the ED or were referred to the orthopaedic department out-patient clinic between 1 January 2002 and 29 February 2008 were eligible for the study.

Exclusion criteria

Patients were excluded if there was no evidence of acute trauma, no evidence of rapid post-traumatic effusion (<24 hours), clear fluid on aspiration, delayed MRI until after the sub-acute period, defined as six weeks post trauma, lacking medical records, or if they were included with the same knee earlier. Knees with fractures other than osteochondral, avulsion or impaction fractures were also excluded. These were mainly fractures to the femoral or tibial condyles and patella fractures.

The AKP routine

All patients with a verified or suspected traumatic knee hemarthrosis were eligible for a sub-acute knee MRI. Plain radiographs were ordered in cases of clinical suspicion of a fracture but were not mandatory. A clinical examination was performed at presentation and suspected injuries managed accordingly. Generally, collateral ligament injuries grade 2-3 were treated with a hinged brace. The hemarthrosis was recommended to be evacuated if the patient gave consent and if it was practically feasible. Patients were given an information leaflet about the project, and an MRI was ordered. The radiology department scheduled the patient for a sub-acute MRI within 5 working days. Once the MRI was made and assessed by the radiologist, the images were demonstrated at the dedicated knee rounds held twice weekly. The attending nurse had prepared the charts from the initial visit for presentation. Management of the individual cases was determined at the rounds by the attending knee specialists based on the medical records and demonstrated images. Treatment recommendations could involve physiotherapy alone, physiotherapy and a scheduled appointment at the out-patient clinic, sub-acute appointment with a knee specialist, sub-acute surgery, or the fitting, adjustment or discontinuation of a knee brace. Subsequently, the research nurse contacted the patients primarily by telephone forwarding the information about the injuries at hand and planned management strategy. The patients were, at this stage, consecutively included in the study file by the research nurse.

Forming the cohort

A study sample of 1279 consecutive individual knees with acute soft-tissue knee trauma was collected over the study period. In case of multiple MRI of the same knee, only the first one was included. Medical records, radiography reports and MRI reports were mainly reviewed by two investigators (OO and AI) according to the study protocol (Table 1). Baseline data were retrospectively collected and recorded in the study database. This included sex, date of injury, laterality, clinical examiner and level of experience, earlier visits for the same injury, the date of clinical examination at presentation, activity at injury, previous injury to either knee and what type, estimation of Tegner activity score if possible, rapid or slow effusion, aspiration of the knee and result, graded instability in the sagittal and coronal planes, indecisiveness or lack of final diagnosis, clinical diagnosis at the initial visit, diagnosis code according to the International Classification of Diseases tenth version (ICD-10), county of residence at injury, date of MRI, name of MRI reader, results of MRI reading, date and results of plain radiographs, name of radiographer, date of radiography rounds and results thereof regarding further management.

Non-eligible knees were excluded (n=134) according to the exclusion criteria (Figure 2) and the final cohort of 1145 unique knees in 1129 individuals was formed.

Table 1. Protocol for data retrieval from medical records and MRI/radiograph reports
NOS, Not otherwise specified. ACL, anterior cruciate ligament. MCL, medial collateral ligament. LCL, lateral collateral ligament. LPD, lateral patellar dislocation. ICD-10, international classification of diseases tenth revision

Patient demographics
Date of birth, Laterality of injury, Sex, Assessed Tegner Activity Level, Time of injury, Time of clinical examination, Previous knee injury in the injured or contralateral knee and specification of what and when
Activity during injury
Injury during sports, work or other leisure activity. If injured during sports, which sport
Category clinical examiner
Emergency doctor; orthopaedic surgery resident; general orthopedic surgeon; knee specialist team member
Hemarthrosis
Rapid (<8 h) or slower onset of effusion. Aspiration or not. Result of aspiration if performed
Clinical examination
Anteroposterior instability, stable/unstable/pain makes assessment impossible/not noted
Valgus instability, grade 1-3/pain makes assessment impossible/not noted
Varus instability, grade 1-3/pain makes assessment impossible/not noted
General impression of stability examination, Clinical desicion/Unsure assessment
Clinical assessment
Knee sprain NOS/ACL/ACL+MCL/ACL+LCL/ACL+meniscus/meniscus tear/LPD/isolated MCL/isolated LCL/not noted/other injury, if other injury – which, ICD-10 diagnosis
Radiography
Acute plain radiography or not, radiologist, time of examination, laterality, examined under weight-bearing or not, effusion or not, fracture or not, actuality and type of fracture if present, osteophytes and localisation if present, joint space narrowing and localisation if present
MRI
Time of examination, radiologist, laterality, assessment:
ACL, intact/total rupture new or old/partial rupture/graft rupture/reconstructed (intact)
PCL, intact/total rupture new or old/partial rupture/graft rupture/reconstructed (intact)
MCL/LCL, intact/grade 1-3
Meniscus medial/lateral, intact/injured, localisation if injured: anterior horn/posterior horn/body, type if injured: horisontal/vertical/comminute/dislocated/buckethandle/meniscocapsular/ contusion/radial/flap tear
Bone marrow edema, femoral condyles/tibial condyles/patella, none/medial/lateral/not noted
Chondral injuries, Femoral condyles/tibial condyles: none/medial/lateral/not noted, patella/trochlea: none/present/not noted
Fractures, osteochondral fracture or not, impaction fracture or not, other fracture or not, if other fracture – which. Localisation of fracture if present: medial femur/lateral femur/medial tibia/lateral tibia/patella/trochlea
Typical signs of LPD
Baker cyst present or not
Management
Time of presentation at MRI rounds, Decision: no action/schedule for visit/physiotherapy/brace/sub-acute arthroscopy/sub-acute meniscus fixation and ACL reconstruction/other treatment. If other treatment - which

Potential losses of study individuals

There were several potential losses of subjects in the cohort that was not accounted for. First, patients who did not seek medical attention or patients who were cared for by healthcare personnel who were not aware of the project had no possibility to be entered into the study file. Further, there were patients who potentially were scheduled for acute or sub-acute surgery without being subject to an MRI, i.e. locked knees with a suspicion of a bucket handle meniscus tear, LPD with a large osteochondral (OC) fracture seen on plain radiographs, or knee dislocations that sometimes could have been referred to the university hospital prior to MRI.

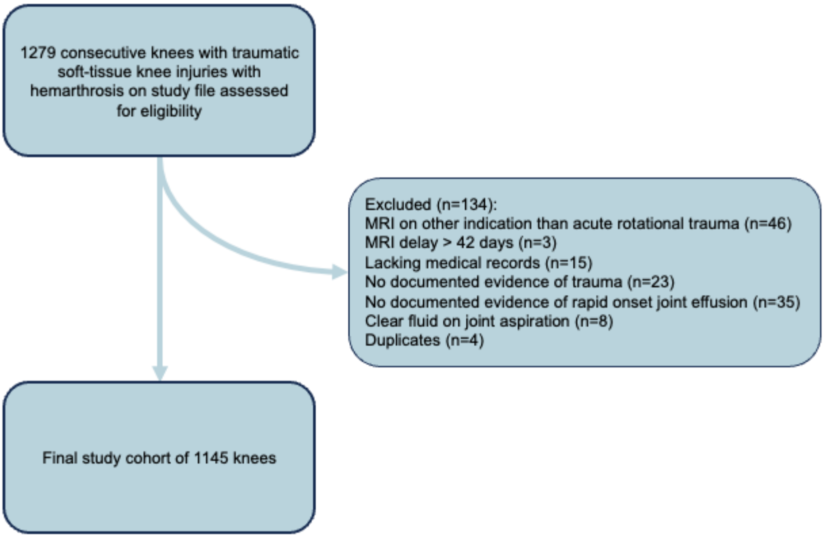

Figure 2. Flowchart of exclusion criteria and the formation of the final study cohort
MRI, magnetic resonance imaging

The primary traumatic patellar dislocation cohort (Paper II)

There were 199 knees with LPD in the baseline cohort, as visualised on MRI. After revision of the medical records, 168 could be identified as first-time traumatic patellar dislocations and included in the cohort. Exclusion criteria were a documented or suspected earlier LPD. Knees with an earlier history of patellofemoral pain or suspected subluxation was accepted for inclusion, under the circumstance that it was obvious in the patients' medical records that there had been no prior dislocation. In the study database, there were additionally seven knees with a documented primary LPD during the study period between 1 January 2002 to 29 February 2008, but the MRI in the study file was from a secondary event. These were included for the epidemiological calculations. In total, 175 knees with a primary traumatic LPD were included from the baseline cohort while 24 knees with a history of dislocation or surgery for patellofemoral instability outside the study period were excluded (Figure 3).

As some patients with a traumatic LPD could be subject to sub-acute arthroscopic surgery due to findings of a suspected large OC fracture on plain radiographs without a prior MRI, we scrutinised the surgical records. At the time, arthroscopic surgery was only offered at two hospitals in the geographical region serving the studied population. All surgical records from the two hospitals during the study period were reviewed for any surgery under the ICD-10 diagnoses of LPD (S83.0), Chondral injury (S83.3) or Loose body (M23.4) and the Nordic Medico-Statistical Committee (NOMESCO) Classification of Surgical Procedures (NCSP-96) surgery codes for arthroscopic or open fixation of articular surface fragments (NGF21, NGF22), excision of articular surface fragments (NGF31) or excision of loose body (NGH41). Since all surgical procedures are noted together with the patients' unique personal identification number, we reviewed the medical records and found another nine knees with primary traumatic patellar dislocation that were not in the baseline cohort. Hence, the total cohort of primary LPD during the study period comprised 184 knees. Among these there were seven individuals included in the study cohort with both their knees due to bilateral LPD. The second knee was excluded from incidence calculations. Furthermore, eight knees had an MRI or surgery (n=4, respectively) more than 42 days (six weeks) after injury, which was stipulated to be the sub-acute period, and were excluded from calculations on concomitant intra-articular injuries since the time lapse potentially could tamper the diagnostic accuracy (Figure 3).

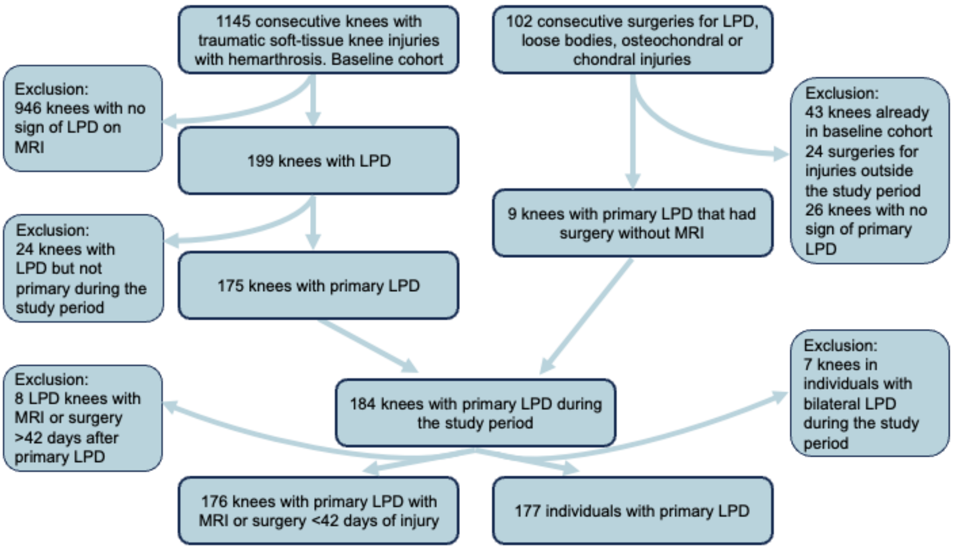

Figure 3. Flowchart of the formation of primary patellar dislocation cohort and exclusions
LPD, lateral patellar dislocation. MRI, magnetic resonance imaging

In summary, from the study period, we identified 184 knees with traumatic primary LPD in 177 individuals. Of the 184 knees, 176 were examined with MRI or arthroscopy within 42 days.

The follow-up cohort (Paper III)

The research subjects from the baseline cohort were contacted for long-term follow-up at a median of 12 years (IQR, 11-14; range 8-18). At least three attempts were made by telephone and one per mail. Reimbursement for travel costs was offered. Patients who could not be reached or did not show up twice for a scheduled appointment were considered lost to follow-up (Figure 4).

Follow-up was staged for practical reasons according to sub-groups and the follow-up protocols were slightly different depending on the main diagnosis at index injury and a concomitant RCT (KANON). The follow-up groups were LPD, Collateral (collateral ligament injuries grades 2-3), PCL, KANON, ACL+ (patients excluded from KANON), and Total (all remaining patients from the baseline cohort).

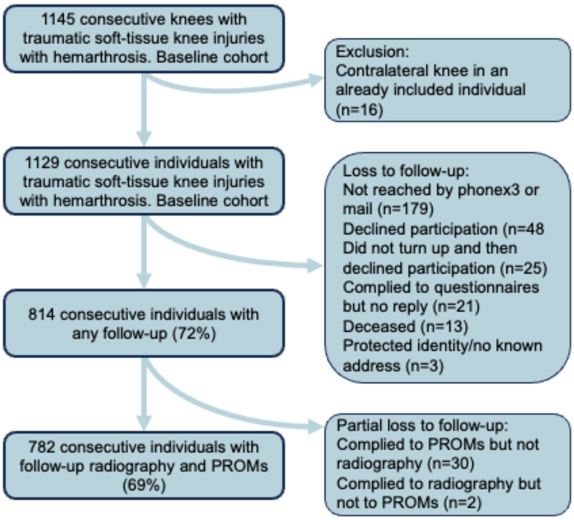

Figure 4. Flowchart of study cohort and loss to follow-up by reason
PROMs, patient-reported outcome measures

In total, 814 (72%) out of 1129 patients in the cohort (1145 knees) went through a follow-up protocol across the groups: Total (n=314), Collateral (n=161), LPD (n=128), KANON (n=95), ACL+ (n=93), and PCL (n=23).

Out of these, 782 consecutive individuals (69%) had a full follow-up with bilateral radiography and patient-reported outcome measures (PROMs). This main cohort was used for all descriptive purposes (Figure 4).

Follow-up visit

The visit was scheduled to the radiology department and included radiographs of both knees, weight, height, a semi-structured interview, and PROMs in one 45-minute session. Written informed consent was obtained together with PROMs by the research nurse. Plain radiographs were taken by a radiography nurse while the clinical examination and a semi-structured interview were performed by an orthopedic specialist or a medical student who had been trained by an orthopedic specialist involved in the study.

Diagnostics

Clinical examination

Clinical examination at baseline

If presenting to the ED, the patients were examined by the attending doctor, generally an orthopaedic trainee or emergency doctor. Fifty-nine percent of the patient in the baseline cohort were examined by a doctor of one of these categories. Patients triaged or referred to the orthopaedic department were most often examined by a knee sub-specialist or a specialist in orthopaedics at a dedicated acute knee clinic. The median time from injury to clinical examination was 2 days (IQR, 1-7). Hemarthrosis was confirmed by knee joint aspiration in 53% of the cases.

The sub cohort of primary traumatic patellar dislocations had an initial clinical evaluation at a median of 1 day (range 0-31) from injury.

Clinical examination at follow-up

Clinical examination according to protocol was performed in 786 patients (69%) at the follow-up visit including length and weight. The examinations differed slightly between groups but consisted for all parts of a thorough examination of both knees. Intra-articular swelling was evaluated through the ballottement test that has been found associated with joint effusion.[116] Range of motion was measured with a goniometer.

Valgus and varus instability was assessed according to Fetto and Marshall and graded into grade 1 (stable at 0 and 30 degrees of flexion), grade 2 (stable at 0 degrees but unstable at 30 degrees of flexion), and grade 3 (unstable at 0 and 30 degrees of flexion).[117] Further, the LCL was palpated with Cabot's manoeuvre in the figure of four position.[118]

The ACL was evaluated through the Lachman test and graded as normal, grade 1 (3-5 mm translation), grade 2 (5-10 mm), and grade 3 (>10 mm) and noted with a firm or soft endpoint.[119] The pivot shift was graded according to Hefti et al as normal, grade 1 (glide), grade 2 (clunk), or grade 3 (gross).[119]

The posterior cruciate ligament was evaluated through observation in 70 degrees of flexion and the posterior drawer step off-test as grade 1 (increased laxity, but anterior tibia not parallel with femoral condyles), grade 2 (increased laxity and anterior tibia parallel with femoral condyles), and grade 3 (increased laxity and anterior tibia posterior to femoral condyles).[120]

Location of tenderness at palpation was noted. Quadriceps hypotrophy was measured through the thigh diameter seven centimetres above the upper limit of the patella.[121]

Lateral patellar dislocation cases were evaluated for the J-sign,[122-124] the Q-angle,[125, 126] and patellar mobility.[125, 127] Apprehension was evaluated with Fairbanks test[128] and the moving patellar apprehension test.[129]. Additional palpation was made along the patella[130, 131] together with the patellar compression test.[132-134] In these patients the Beighton score was examined as well.[135, 136]

Semi-structured interview at follow-up

In preparation for the visit, the examiner made a review of the medical records according to protocol in the sub-groups of LPD, Collateral, PCL and Total (n=607). A semi-structured interview was held during the visit according to the same protocol to stipulate the initial trauma, initial treatment, feeling of instability/give away/redislocations, compliance to bracing if initiated, surgeries, additional knee injuries, pain and other knee-related problems.

Magnetic resonance imaging

The median time from injury to MRI was 8 days (IQR, 5-15, range 0-42). Two different MRI machines were used during the study.

Magnetic resonance images were examined by experienced radiologists according to an analysis protocol for later recording in the study file (Table 1). Two senior radiologists (TB and HdV) with several years of special interest in knee radiographs and knee MRI read 93% of the examinations and the remaining were examined by a handful of other dedicated musculoskeletal radiologists trained by them. Findings were classified according to the recommendations by Khanna et al.[137]

The MRI scanners were mainly a 1.5 Tesla (T) imager (Gyroscan, Intera, Philips, Eindhoven, the Netherlands) but also a 1.0 T imager (Impact, Siemens, Erlangen, Germany) both used with a circular polarized surface coil. The patients were examined with a T2-weighted turbo spin-echo sequence (tSEPdT2) and a T2-weighted turbo short tau inversion recovery sequence (tSTIRT2) on the 1.5 T imager and a proton density- and T2-weighted turbo spin-echo sequence (tSE PdT2) on the 1.0 T imager in the coronal and sagittal views. The sagittal sequence was perpendicular to a line connecting the dorsal aspects of the femoral condyles and the coronal sequence was parallel to that line.

The two senior radiologists did not report any relevant differences in the analysis of anatomical lesions between the machines. Studies have shown comparable results to usually observed intra- and inter-reader reliability with a moderate to high level of agreement and accuracy between 1.0 T and 1.5 T systems[138] and comparable accuracy between 1.5 T and 3 T MRI,[139-141] although modest increased performance for 3.0 T MRI as compared to 1.5 T has been noted for articular surface pathology.[142-145]

Classification of injuries in the baseline cohort (Paper I)

Any derangement of the ACL as seen on MRI was classified as an injury irrespective if the rupture showed signs of being acute (n=527), chronic (n=33), partial (n=30) or a graft rupture (n=9). The PCL was classified in the same way. The collateral ligaments were graded according to Khanna as grade 1 (periligamentous edema without signs of discontinuity), grade 2 (partial tear), and grade 3 (complete tear).[137] Tears in the medial or lateral meniscus were classified as present or absent, irrespective of type and location.[146] Signs on the MRI of post-traumatic bone marrow lesions (BML), impaction fractures and osteochondral fractures were classified as present or absent in the lateral and medial femoral and tibial condyles, respectively.[147] The injury was classified as a lateral patellar dislocation if there were characteristic post-traumatic BML patterns in the medial patella and/or the anterolateral portion of the lateral femoral condyle.[25, 148-152]

All injuries to the ACL, PCL, MCL, LCL, or meniscal tears as well as typical signs of LPD and fractures were considered primary acute traumatic structural injuries in the analyses and were presented as isolated entities or combinations.

Classification of injuries in the primary patellar dislocation cohort (Paper II)

A total of 171 knees from the baseline cohort with typical signs of LPD on sub-acute MRI (Figure 5) and history of a first-time traumatic dislocation were reviewed for evidence on the MRI of concomitant injuries to the cartilage surfaces together with the surgical reports of five knees that were scheduled for surgery due to an acute LPD without a prior MRI. The cohort of primary patellar dislocations had MRI within a median of 6 (range, 1-39) days of injury.

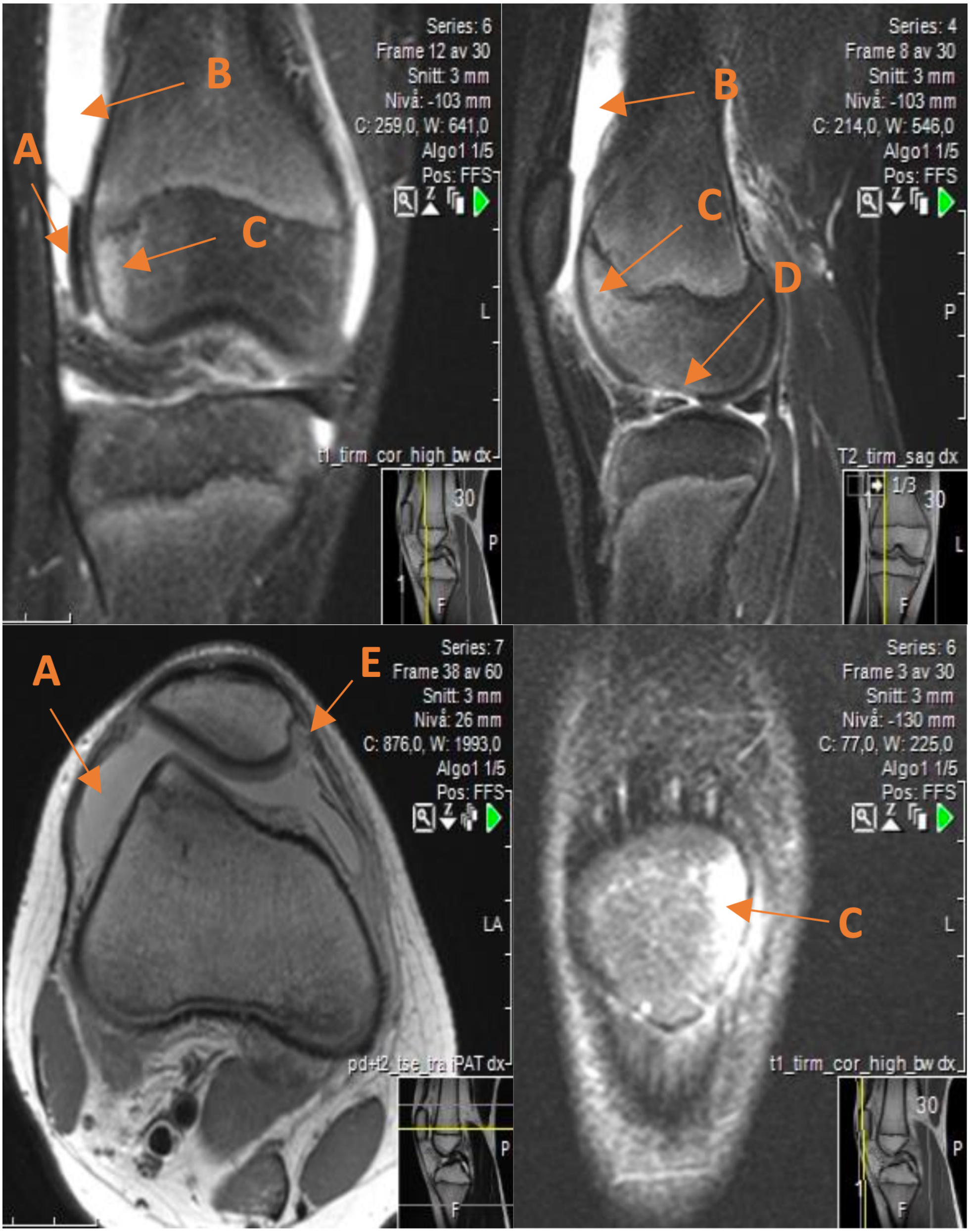

Figure 5. Typical MRI signs of LPD with an osteochondral fracture
A loose chondral fragment (A) in the lateral recess of a 13-year-old boy following lateral patellar dislocation sustained during leisure activity with large effusion (B) and typical bone marrow edema (C). The injury site is on the lateral femoral condyle (D) and there is a small avulsion fracture on the medial border of the patella (E).

Classification of injuries in the follow-up cohort (Paper III)

The follow-up cohort was divided into groups depending on the initial injury as seen on MRI at baseline to compare outcomes between different injury types.

The six main injury types are depicted below.

Categorisation of injuries into six main injury types

- **Hemarthrosis alone** - No structural injury except MCL or LCL grade 1 (edema only), i.e. only hemarthrosis as the intra-articular observation with or without BML.
- **LPD** - Lateral patellar dislocation with or without concomitant cartilage injury potentially combined with other injuries but no ACL rupture.
- **Other injury** - Structural injury in any combination of MCL or LCL grade 2-3, PCL any grade, cartilage injuries, osteochondral or compression fractures but no ACL rupture, LPD or meniscus tear.
- **Meniscus/not ACL** - Meniscus tear but no ACL rupture (potentially combined with other injuries).
- **ACL/not meniscus** - ACL rupture but no meniscus tear (potentially combined with other injuries).
- **ACL and meniscus** - ACL rupture and meniscus tear (potentially combined with other injuries).

The injuries were, hence, often in combinations. Details of included injuries in the follow-up cohort of the 782 injuries with a comprehensive follow-up are outlined in Figure 6.

The follow-up cohort included 139 knees with multi-ligament knee injuries, defined as at least two of the following injuries in combination: ACL, PCL, MCL and/or LCL grade 2 or 3, as visualised on MRI. This group consisted of six knees with concomitant injuries to ACL, PCL and MCL, one knee with ACL, PCL, MCL and LCL, one knee with ACL, PCL and LCL, four knees with ACL and PCL. 110 knees with ACL and MCL, six knees with ACL, MCL and LCL, and 11 knees with ACL and LCL. Furthermore, three knees had concomitant ACL rupture and typical signs of LPD.

Hemarthrosis alone (n=70):	1 MCL* + LCL* + BML, 4 MCL* + BML, 2 isolated MCL*, 1 LCL* + BML, 36 isolated BML, and 26 with no findings
LPD (n=124):	7 MCL, 1 LCL, 4 MM, 7 LM and, 1 MM + LM, but excludes 3 with typical signs of LPD on MRI with concomitant ACL rupture as this was considered the main injury
Other injury (n=67):	4 isolated PCL, 1 PCL + MCL + comp, 1 PCL + MCL, 20 isolated MCL, 5 MCL + cart, 3 MCL + comp, 1 LCL + cart + OCF, 2 cart + OCF, 4 cart + comp, 12 isolated cart, 14 isolated comp
Meniscus/not ACL (n=70):	37 MM, 25 LM, 8 MM + LM. In combination with 4 PCL, 3 PCL + MCL, 9 MCL, 1 LCL
ACL/not meniscus (n=197):	3 PCL, 4 PCL + MCL, 1 PCL + MCL + LCL, 52 MCL, 3 MCL + LCL, 4 LCL, 2 LPD
ACL and meniscus (n=254):	114 MM, 69 LM, 71 MM + LM. These occurred in combination with 4 PCL (1 PCL + MCL + cart + comp, 1 PCL + MCL, 1 PCL + LCL, 1 isolated PCL), 62 MCL (1 MCL + LCL, 1 MCL + LCL + cart, 1 MCL + LCL + comp, 4 MCL + cart + imp, 6 MCL + cart, 25 MCL + comp, 24 isolated MCL), 7 LCL (1 LCL + comp, 1 LCL + cart, 5 isolated LCL), and 1 LPD

Figure 6. Details of included injuries in each subgroup of injury type after classification
MRI, magnetic resonance imaging. BML, bone marrow lesion. MCL, medial collateral ligament injury. LCL, lateral collateral ligament injury. LPD, lateral patellar dislocation. MM, medial meniscus tear. LM, lateral meniscus tear. ACL, anterior cruciate ligament rupture. PCL, posterior cruciate ligament injury. OCF, osteochondral fracture. Cart, cartilage injury. Comp, compression fracture.* MCL/LCL Grade 1, all other collateral ligament injuries (MCL/LCL) were grade 2 or 3.

Plain radiography

Knee radiography at baseline

Plain radiographs were not mandatory but sometimes obtained as part of the initial clinical visit at the ED and were in these instances noted in the study file. In the baseline cohort, 652 out of the 1145 knees (57%) had plain radiographs at a median of 1 day after trauma (IQR, 0-3). Of these, 149 were taken under weight-bearing.

Among the 184 knees with primary acute LPD, plain radiographs were obtained as part of the initial clinical routine in 124 (67%) cases.

The radiographs at baseline were not examined according to protocol and were not part of the research project per se. The radiologist at duty, who was not necessarily specialised in musculoskeletal radiology, made the report as part of normal clinical work.

Knee radiography at follow-up (Paper III)

Standardised plain weight-bearing bilateral radiographs were obtained in three views: posteroanterior frontal plane (Schuss view) and sagittal plane in semi-flexion of the tibiofemoral (TF) compartment and axial plane of the patellofemoral (PF) compartment.[153] In total, 784 out 1129 (69%) patients from the baseline cohort were examined with bilateral plain radiography during follow-up.

The specific instructions for the frontal view were: posteroanterior beams, patient in standing, equal weight-bearing on both legs with big toe, patella and hips bilaterally against the detector and the detector plane with vertical femora, meaning a 30–40-degree flexion in the knees. The medial borders of the feet directed orthogonally against the detector plane. The beam directed 10 degrees caudal with the beams centred in the popliteal fossa and parallel to the tibial joint surface. Criteria for an acceptable film was free projection of the joint and the intercondylar notch. In cases with different slopes on the lateral and medial tibia, two projections were made. For the lateral view, the patient was standing with 20 degrees of flexion in the knee and the relevant knee against the detector. The beam was directed 5 degrees cranial and centred on the femoral condyles. Criterium for an acceptable film was perfect alignment of the anterior femoral condyles. Radiographs in the axial plane were taken standing in a stand called "the horse" with 40 degrees of flexion in the knee and the foot directed straight forward and the beam centred through the patella's anterior border. Criterium for an acceptable film was free projection of the patellofemoral joint.

Analysis of follow-up radiography (Paper III)

Radiographs were analysed by a specialist in musculoskeletal radiography (JP) who was blinded to clinical data. The images were graded according to the Osteoarthritis Research Society International (OARSI) atlas as well as Kellgren and Lawrence (KL).[154-156] Intra-observer correlation was performed for 40 readings.

The severity of OA was assessed by evaluating joint space narrowing (JSN) in the medial TF, lateral TF, medial PF and lateral PF joints together with osteophytes on the medial tibia or femur, lateral tibia or femur, and medial or lateral patella according to Altman et al, using a categorical scale: 0 = normal, 1 = mild change, 2 = moderate change and 3 = severe change.[155]

Patient-reported outcome measures

At the time of the follow-up visit, different PROMs were obtained. Some patients who could not make it to the visit answered the questionnaires by mail (n=30). The follow-up sub-groups had slightly varied protocols due to the nature of the injury and for practical reasons. However, there was a core of questionnaires that was included in the protocol for all patients.

Knee injury and osteoarthritis outcome score

All follow-up groups were administered the Knee Injury and Osteoarthritis Outcome Score (KOOS), which was obtained from 72% (n=811/1129) of the baseline cohort.[157-159]

Knee-related health is commonly measured with KOOS, a 42-item self-administered knee-specific questionnaire that divides into five subscales: knee-related symptoms, pain, function in activities of daily living (ADL), function in sports and recreational activities and, quality of life (QoL). Each question is answered on a 5-point Likert scale.

Tegner Activity scale

All follow-up groups were also administered the Tegner Activity Scale (TAS), which was obtained from 72% (n=812/1129) of the subjects in the baseline cohort.[160]

The general level of sports was evaluated in the TAS questionnaire, where the patient is asked to grade their activity level on a 10-point scale ranging from walking on plain ground (1) to elite competitive sports on a national level (10).

Knee satisfaction questionnaire

All follow-up groups were further administered a knee satisfaction questionnaire with validated measurements previously used in musculoskeletal disorder research, and was obtained from 72% (812/1129) of the individuals in the baseline cohort.[161-163]

The knee satisfaction score validated for outcome after ACL reconstruction consists of four questions:[163]

(1) "Are you satisfied with your present knee function? Yes/No"

(2) "If not, do you consider your treatment as a failure? Yes/No"

(3) "Are you satisfied with your current knee function?" answered on a 10-point Likert scale from 1 "Not satisfied at all" to 10 "Very satisfied"

(4) "If you were to spend the rest of your life with your knee function just the way it has been over the last week, would you feel …?" with the alternatives of "Happy", "Satisfied", "Mostly satisfied", "Mixed feelings", "Mostly dissatisfied", "Dissatisfied" and "Unhappy"

The variables were grouped into good outcome: "Satisfied" (Happy, Satisfied, and Mostly satisfied) and bad outcome: "Dissatisfied" (Mixed feelings, Mostly dissatisfied, Dissatisfied and Unhappy).

Additionally, the questionnaire included questions on general illness since the injury, hospitalisations and further knee injuries in the injured or contralateral knee.

Osteoarthritis

Definition and reliability of radiographic osteoarthritis

Radiographic knee OA was defined as the presence of at least one osteophyte grade ≥2, or JSN grade ≥2, or a sum score of JSN and osteophyte grades ≥2 within at least one of the three compartments (medial TF, lateral TF and/or PF).[146] Tibiofemoral OA was present if the definition of OA was fulfilled in either the medial or lateral compartment. Patellofemoral OA was present if the definition of OA was fulfilled within the patellofemoral joint.

Intra-observer variability was calculated from repeated readings of 40 individuals (80 knees) with a Cohen's Kappa (bootstrap 95% confidence intervals) of 0.89 (0.76-1.00) for TF OA and 0.81 (0.63-0.98) for PF OA. The definition approximates Kellgren and Lawrence grade 2 or higher for the TF joint with a Cohen's kappa of 0.90 (95% CI 0.87-0.93), sensitivity of 94%, and specificity of 98% in our material.[156] Total knee arthroplasty (n=2) was considered as both TF and PF OA.

Definition of symptomatic knee osteoarthritis

An individual was considered to have symptomatic knee OA if they met the criteria for radiographic OA as outlined above, and had symptoms based on KOOS values as previously defined by Englund et al.[146]

A knee was classified as symptomatic if

1. the KOOS subscale for knee-related Quality of Life (QoL) and
2. at least two of the remaining four subscales
3. had scores equal to or less than the threshold obtained when at least half of the questions in a subscale were answered with one or more steps decrease from the best response on the 5-point Likert scale.

This calculates into scores indicating a symptomatic knee, after conversion to a 0-100 scale (worst to best outcome), in the KOOS subscales for knee-related

- symptoms ≤85.7
- pain ≤86.1
- ADL ≤86.8
- sports/recreation ≤85.0
- QoL ≤87.5

Epidemiology

The baseline cohort, population at risk and incidences

The population in the catchment area at different time points and the populations at risk were extracted from the open archives of Official population statistics of Sweden, Statistics Sweden.

To account for seasonal variations of injury patterns, only complete years within the study period from 1 January 2002 to 29 February 2008 were included. Hence, the years of 2004 and 2008 were excluded from incidence calculations and the analysis of injury distribution over the year. In 2004 there was a change in the catchment area population due to a merge between the Helsingborg and Ängelholm hospitals and 2008 only consisted of January and February. This resulted in the exclusion of 216 knees, leaving 929 knee injuries during the full study years of 2002-2003 and 2005- 2007.

Repeated sensitivity analyses were made to define the populations at risk of knee injury, in general and for specific sex and age groups, as well as confirming consistency in the calculated incidences. Estimates of mid-year population from end-year population of consecutive years were used.

Incidences were calculated for different types of injuries for the whole cohort, and for males and females separately, within the age groups 10-19 years to 50-59 years and expressed as the yearly number of injuries per 100 000 inhabitants. Incidence for patients with LPD were also calculated in the age group 10-16 years.[80] To evaluate how age and sex affect injury patterns, relative incidences of specific injuries were calculated. These were expressed as the fraction of the overall incidence of traumatic hemarthrosis for each separate age and sex group.

The primary patellar dislocation cohort, population at risk and incidences

During the study period, 184 primary traumatic patellar dislocations were identified in 177 individuals. The second dislocation was excluded for individuals with bilateral LPD (n=7). Incidences were calculated as the number of primary dislocations per 100 000 person-years for those at risk annually. Population at risk was extracted from the same source as above (Statistics Sweden) per 1-year age groups and calculated per month for the divided years 2004 and 2008. Peak incidences were calculated in 3-year age groups 10-12 years to 25-27 years as well as the incidence in the total population, overall and by sex.

Statistical methods

Baseline cohort (Papers I-II)

Since the population was not normally distributed, continuous variables were expressed as medians, interquartile range and range. Non-parametric tests were used for between-groups comparisons, the Mann-Whitney U-test for continuous variables and Chi-square test for binomial data. For incidences, 95% confidence intervals were determined using the Clopper-Pearson exact method and comparisons between proportions were made using the Fisher's exact test. A two-tailed P value < 0.05 was considered to determine statistical significance.

In paper I, Analyse-it Standard Edition 3.20 (Analyse-it Software, Ltd. Leeds) was used for statistical analyses.

In paper II, SPSS version 28 for Mac (IBM Corp, Armonk, NY, USA) was used for descriptive statistics and between groups comparisons, Epitools (Sergeant, ESG, 2018. Epitools Epidemiological Calculators. Ausvet) for determination of confidence intervals, and MedCalc (MedCalc Software, Ostend, Belgium) for comparisons between proportions.

Follow-up cohort (Paper III)

In calculations of radiographic and symptomatic knee OA we assumed that the presence of pre-existing OA in either knee at the time of injury would be negligible, given that the median age was 26 (IQR, 17-35) when injured, and only 8% (n=88/1129) were 45 years or older.

Cumulative incidences of radiographic knee OA, tibiofemoral OA and patellofemoral OA as well as symptomatic knee OA, were calculated as incidence proportions with 95% logit transformed confidence intervals (CIs).

The risk of radiographic OA in the injured compared to the contralateral knee was analysed with the two knees as a matched pair by a fixed effects Poisson regression model, including only knees with at least one outcome (OA). This in-person analysis accounts for multiple measurable and un-measurable person-level confounders such as age, sex, body mass, physical activity level, risk propensity, etc. Risk ratios (RR) with 95% CIs were calculated with the contralateral knee as the reference category. Additionally, adjustments were made for earlier knee injuries as collected from the medical records at baseline, and new knee injuries since the initial trauma as reported by the patients at follow-up.

Between-persons comparisons of risk of radiographic and symptomatic OA in the injured knees between different injury types were made with a Poisson regression model with robust standard errors. Risk ratios were calculated with 95% CIs using the group of hemarthrosis alone as reference. However, due to the low incidence of OA in this group, it was not possible to do comparisons to other injury groups with an adjusted regression model. Thus, crude estimates with adjustments only for time to follow-up were calculated. To control for sensitivity, comparisons between different injury groups and the miscellaneous group of other injury, i.e. other than ACL, meniscus or LPD, were made with additional adjustments for age, sex, sports-related injury, earlier or new knee injury, and body mass index (BMI).

The patient-reported outcome measures that were analysed included KOOS, TAS and knee satisfaction score, which all exhibit ceiling effects. Therefore, a Tobit model (linear regression for censored data) was applied with the results reported as mean differences with 95% CIs. Further adjustments were made for the same confounders as in the between-persons analysis of knee OA risk.

Confidence intervals were provided as inferential measures, due to the observational nature of the study with no a priori sample size calculation. This observational study was designed to include all eligible patients presenting with traumatic knee hemarthrosis between the years 2002 and 2008, when standardised MRI and clinical routines were used per protocol. No sample size calculations with the current outcomes in mind were made.

Stata 18 (StataCorp. 2023. Stata Statistical Software: Release 18. College Station, TX: StataCorp LLC) was used for all calculations in paper III.

Ethical considerations

This project was conducted in accordance with the ethical standards of the Helsinki Declaration. Approval was obtained from the local institutional ethics board at Lund University, Sweden, with registration numbers Dnr 2009/237 and 2015/250.

All components of the baseline projects (Papers I-II) were elements in a new clinical routine that was implemented at the start of the project and did not involve any interventions. The ethics board concluded that there was no need for ethical approval but delivered an advisory opinion that the project was of value and that no ethical risks could be detected (Dnr 2009/237). However, information regarding the project was disseminated to the public through the three major newspapers in the area. The notice included details on whom to contact for individuals who wished to exclude their information from the scientific component of the project.

There were ethical considerations involved in the follow-up project (Paper III). Ethical approval was granted for all parts of the project (Dnr 2015/250). All subjects voluntarily consented to take part after receiving thorough information. The visit was not part of standard care and involved coming to the hospital for a 45-minute session, including additional radiographs of both knees in three views, a clinical examination, and questionnaires. Regarding the additional ionising radiation, a statement was obtained from the hospital physicist, being responsible for radiation safety. It declared that the radiographs corresponded to one day's background radiation (0.002 mSv), which was considered insignificant. The follow-up group PCL had a new MRI in close temporal vicinity of the visit, which involved additional time consumed. In contrast to plain radiography, MRI does not include ionising radiation associated with the risk of DNA damage and carcinogenic mutations. No current scientific evidence is available that suggests that MRI is associated with an any increased risk of health issues. There were plans to take urine and blood samples at follow-up, which later was excluded from the protocol. Written consent was obtained from all subjects.

The follow-up visit did not involve any cost for the research subjects. Only reimbursements for travel cost were made and the participants were offered a scheduled visit with a knee specialist if needed.

Results

"A rigid plaster or leather case to be worn for a year, followed by a hinged apparatus represents the generally accepted method"

- Ernest William Hey Groves on the treatment of knee ligament ruptures, 1917

Distribution and incidences of soft-tissue knee injuries (Paper I)

Most traumatic knee injuries with hemarthrosis occurred in males (64%). The median age at injury was 24 years for both males and females (IQR, 17-33; range, 6-64 and IQR, 16-38; range, 6-64, respectively) and knee injury was most frequent in the ages 10-19 years for both, with a bimodal distribution for females (Figure 7). There was no difference in the rate of significant structural injuries on MRI between knees that had hemarthrosis confirmed by aspiration and others (p=0.94).

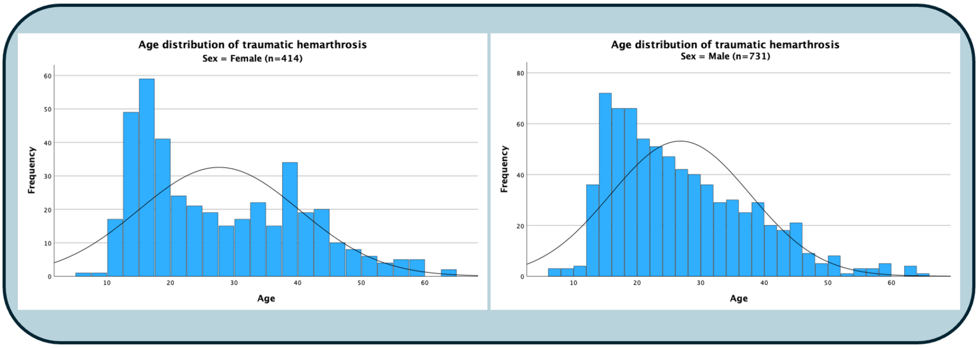

Figure 7. Distribution of knee injuries with traumatic hemarthrosis over age, by sex

Structural soft-tissue knee injuries

An anterior cruciate ligament rupture was the single most common injury (599/1145, 52%), followed by meniscal tear (474/1145, 41%). Twenty-nine percent of the knees had a medial meniscus tear (n=327), 22% a lateral meniscus tear (n=253), and 9% both medial and lateral tears (n=106). MCL injury grades 1-3 occurred in 28%, impaction fracture in 21% (n=240), LPD in 17% (n=199), and an LCL injury grades 1-3 in 6% of the knees (n=33). Least commonly, a PCL rupture was present in 3% of the knees (n=33), roughly one per 20 ACL ruptures.

An MCL rupture grade 2 (n=130) or grade 3 (n=114) occurred in 21% of the knees (244/1145) and an LCL rupture grade 2 (n=23) or grade 3 (n=6) in 3% of the knees (29/1145).

A lateral patellar dislocation was the most common injury for both girls (42%), and boys (39%) in children under the age of 16. Males injured their ACL most frequently between 20-29 years of age, while females most frequently injured their ACL in the ages 10-19.

Except for LPD, which generally occurred in isolation, most injuries were in combinations. Injuries to the ACL were combined with other ligament injuries, meniscal tears, compression or osteochondral fractures in 88% of cases. Lateral collateral ligament injuries were also seldom in isolation, most often associated with an ACL rupture and in 68% in combination with at least two more injuries (Table 2).

Table 2. Number of injuries on MRI, in isolation and combinations

The number of index injuries and combined injury types and the proportion of the combination to the index injury is given. NB, since injuries occur in combinations, the total number of diagnoses exceeds the total number of knees with traumatic hemarthrosis (n=1145)

ACL, anterior cruciate ligament. PCL, posterior cruciate ligament. MCL, medial collateral ligament. LCL, lateral collateral ligament. LPD, lateral patellar dislocation. MT, meniscus tear. Associated, more than one associated injury including compression and osteochondral fractures.

MAIN INJURY	Isolated	In combination with						
		ACL	PCL	MCL	LCL	LPD	MT	>1 associated
ACL (n=599)	72 (12%)	-	16 (3%)	231 (39%)	58 (10%)	2 (0%)	331 (55%)	240 (40%)
PCL (n=33)	6 (18%)	16 (48%)	-	13 (39%)	3 (9%)	0	14 (42%)	17 (52%)
MCL (n=323)	53 (16%)	230 (71%)	13 (4%)	-	11 (3%)	13 (4%)	107 (33%)	147 (46%)
LCL (n=65)	2 (3%)	58 (89%)	3 (5%)	11 (17%)	-	2 (3%)	30 (46%)	44 (68%)
LPD (n=199)	130 (65%)	2 (1%)	0	13 (7%)	2 (1%)	-	18 (9%)	11 (6%)
MT (n=474)	94 (20%)	331 (70%)	14 (3%)	107 (23%)	30 (6%)	18 (4%)	-	192 (41%)

Medial meniscus tears were noted in 29% of the knees and lateral meniscus tears in 22%, whilst 9% had tears in both menisci. A concomitant ACL injury was seen in 72% of the knees with a medial meniscus tear and in 75% of the knees with a lateral meniscus tear.

As the footprint of injury, post-traumatic BMLs were noted in 84% of all knee injuries. Knees with an ACL injury had BMLs in 92% of the cases and knees with an intact ACL in 76%. One or more compression fractures were present in 21% of the knees and more often in ACL injured knees than others ($p<0.0001$).

A total of 112 knees (10%) were without noticeable structural injuries on MRI, 63 of these with BMLs without signs of LPD or other structural injury, 40 without any visible pathology in the knee, and 9 with cartilage defects but without BMLs and, hence, considered unrelated to the trauma.

Annual incidences

Traumatic hemarthrosis occurred with an annual incidence of 151 (95% CI, 140-162) per 100 000 inhabitants aged 10-59 years in the studied population with higher numbers for males, 189/100 000 (95% CI, 173-206), than for females, 111/100 000 (95% CI, 98-125). In the ages of 10-16 years, the annual incidence was at its peak at 261/100 000 (95% CI, 224-302), with 305 for boys (95% CI, 250-368), and 214 for girls (95% CI, 168-270), per 100 000. Incidences for traumatic hemarthrosis among males peaked, however, between the ages of 20-29 years with 334/100 000 (95% CI, 284-390).

The incidence of ACL injury was 77/100 000 (95% CI, 70-85), in the ages 10-59 years, 91/100 000 for males (95% CI, 80-103), and 63/100 000 for females (95% CI, 53-73) and peaked in the ages 20-29 years for males, 182/100 000 (95% CI, 145-224), and between 10-19 for females, 89/100 000 (95% CI, 65-120). Lateral patellar dislocations occurred with an annual incidence of 88/100 000 (95% CI, 68-113) in the ages 10-16 years (113 for boys [95% CI, 81-154], and 62 for girls [95% CI, 39-95]).

The overall incidences of traumatic hemarthrosis as well as the incidences for females in ACL injury, meniscal tears, and LPD gradually decreased from 10-19 years to 50-59 years. Medial collateral ligament injuries in females showed a bimodal pattern with highest incidences in the age groups of 10-19 and 40-49 years. In males, incidences peaked in 20-29 years for ACL injury, meniscal tears and MCL injuries, while LPD incidences peaked in early ages. Males displayed higher peak incidences for all major injury groups; ACL, MCL, LPD and meniscal tears.

The relative incidence of injury type compared to hemarthrosis revealed higher fractions for females than males regarding ACL injuries in all age groups (Figure 8), while the relative incidence of meniscal tears was higher for males in all age groups. For both females and males, the relative incidences of LPD gradually decreased, while relative incidences for MCL injuries and meniscal tears gradually increased with age.

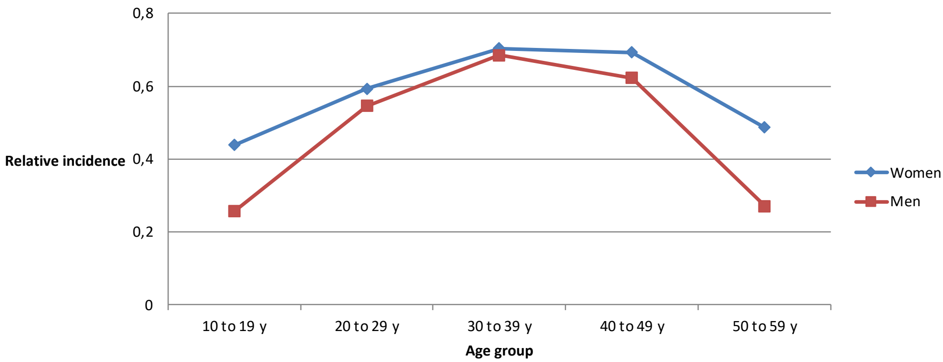

Figure 8. The relative incidence of ACL injury compared to the incidence of traumatic hemarthrosis per age groups and sex.
ACL, anterior cruciate ligament rupture

Sports-related soft-tissue knee injuries

Sports was the main culprit in eliciting a soft-tissue knee injury, standing for 72% of the traumatic knee injuries (n=828). Half of the sports injuries (n=410) were related to football and 41% of all ACL injuries. Alpine skiing (15%), floorball (6%), and team handball (5%) were the other main sports performed while injuring the ACL. A lateral patellar dislocation was less likely to happen during sports (53%), but it was three times more likely to happen during gymnastics compared to ACL or MCL injuries (p=0.013). More than 80% of all ACL and MCL injuries occurred during sports, most often football, but 22% of MCL injuries occurred during alpine skiing compared to 15% of the ACL injuries (p=0.009).

The activity level could be estimated from medical records for 70% of the baseline cohort with a median TAS score of 8 (IQR, 7-9). There were no differences between the ACL injured population and others.

Distribution of injuries over the year

Knee injuries with traumatic hemarthrosis peaked in April and dipped in July and December (Figure 9). Both ACL injuries and LPD followed this seasonal trend. MCL injuries were more frequent in February and March, particularly among women over 30, with 55% occurring during alpine skiing compared to 17% for those in younger ages (p<0.0001). Males did not exhibit this pattern. In sports like football, team handball, floorball, and alpine skiing, ACL injury distribution matched the competitive season.

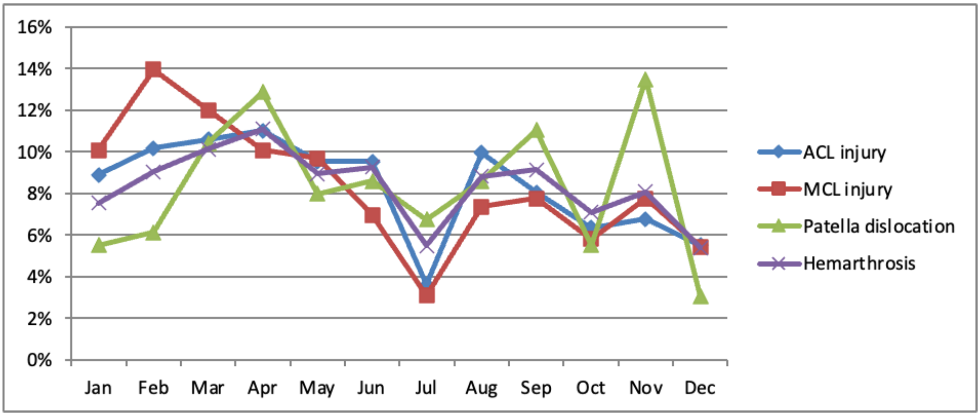

Figure 9. Distribution of traumatic hemarthrosis, ACL, MCL and LPD over the year
ACL, anterior cruciate ligament rupture, MCL, medial collateral ligament injury. LPD, latera patellar dislocation

Incidences and concomitant injuries in primary traumatic patellar dislocations (Paper II)

A first-time patellar dislocation was also more common in males, as compared to females (59% vs. 41%, p=0.015). The median age was 16 years (IQR, 14-21), but males were older than females (17 [IQR, 15-22] and 15 [IQR, 13-20] years respectively, p = 0.021). Practically all primary LPD occurred before the age of 40 (99%) and 55% before the age of 17 (Figure 10).

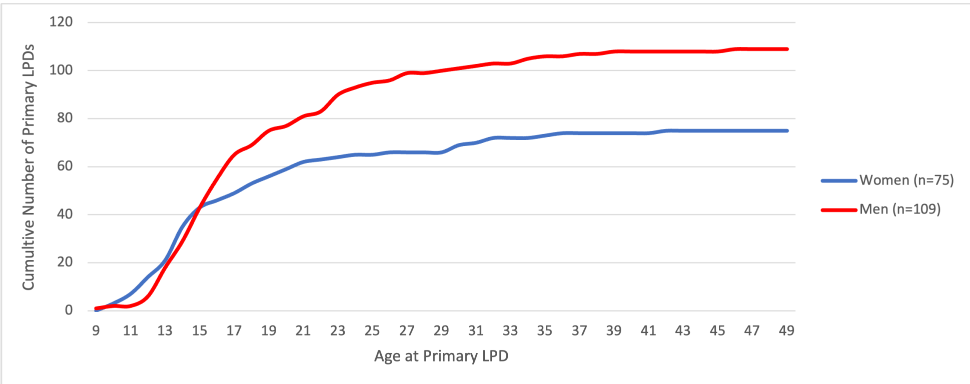

Figure 10. Cumulative numbers of primary LPD, by sex.
LPD, lateral patellar dislocation

Seven individuals (4%) had a contralateral first-time LPD during the study period. All but one of these were under the age of 16 at the first LPD, with an absolute risk of 7% for those under 16 (n=6/83) as compared to 1% (n=1/94) for those 16 years or older.

Incidences

The annual incidence of LPD in the population was 14 (95% CI, 12-16) per 100 000 person-years, or 11 (9-14) for females and 17 (14-20) and males respectively (p = 0.010). A peak of annual incidences for both females and males occurred between the ages of 13 and 15 years (114 [95% CI, 76-165] and 135 [95% CI, 94-187] per 100 000 person-years respectively) and declined gradually over older ages (Figure 11).

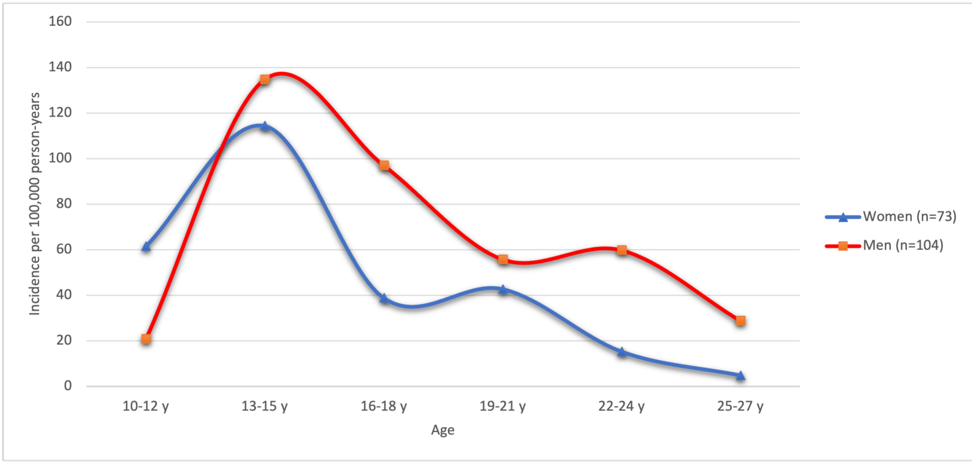

Figure 11. Primary LPD incidences per 3-year age group and sex.
LPD, lateral patellar dislocation

Activity at primary LPD

Sports was the overall most common activity during primary LPD. Fifty-five percent of the injuries were sustained during sporting activity but more often in males compared to females (65% vs. 40%, p<0.001). For girls, however, the majority of the primary LPDs occurred during leisure time and not during sports. Soccer was the most common sport at injury for both sexes, followed by gymnastics, martial arts and alpine skiing.

Concomitant chondral or osteochondral injuries

A total of 176 knees with primary LPD had either an MRI (n=171) or arthroscopic surgery without prior MRI (n=5) in the sub-acute phase (i.e. within 42 days). In these knees there were 113 concomitant lesions to the cartilage surfaces observed in 75 of the 176 knees (43%). These were located on the patella (n=56), the lateral femoral condyle (n=49), or trochlea femur (n=8) and consisted of a total of 69 purely chondral lesions, 34 osteochondral fractures and 10 impaction fractures as visualised on MRI or during surgery (Figure 12).

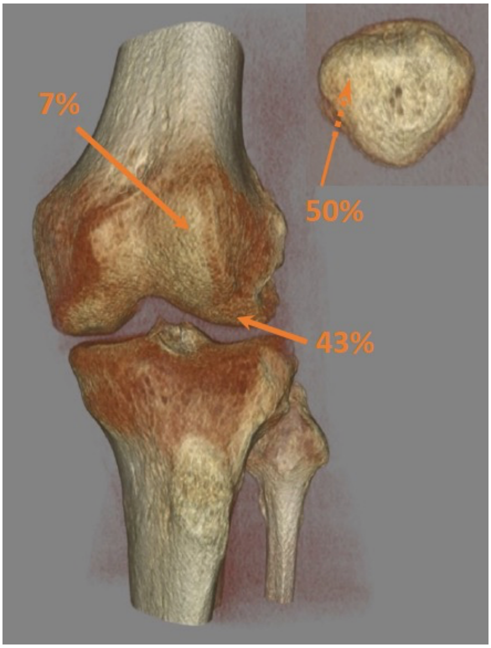

Figure 12. Distribution of 113 concomitant injuries to articular surfaces. 3D-CT of left knee. Pure chondral lesions (n=69), osteochondral fractures (n=34), and impaction fractures (n=10) in 75 (43%) knees with concomitant articular surface injuries.

The risk of injury to the articular surfaces was not greater (p=0.231) during sports (39%) than during other activities (48%). No difference was seen in the risk of concomitant injury to chondral surfaces between males (44%) and females (41%, p=0.696), but males seemed to sustain such injuries during sporting activities more often than females (61% vs 31%, p=0.012).

Individuals with cartilage injuries during LPD were older (median 18 years [IQR, 15-25]) than those without any concomitant injuries to the articular surfaces (median 16 years [IQR, 14-19], p=0.004).

Osteochondral fractures were present in 18% of the knees with LPD (n=32). These were mostly located on the patella (n=17) alone, while 13 were on the lateral femoral condyle and in two knees on both the patella and the lateral femoral condyle. These fractures showed essentially the same age distribution between the sexes (median age females 16 years [IQR, 13-22] and males 18 years [IQR 15-23], p=0.478). The risk of an osteochondral fracture was not statistically significantly different for LPD as a sports injury compared to during other activities (14% vs. 24%, p=0.080), nor between males and females (16% vs 21%, p=0.405). Males' osteochondral fractures occurred during sports in 53% of the cases, while females' osteochondral fractures in 27%, a difference that was not statistically significant (p=0.131).

Plain radiography and osteochondral injuries

Twenty-two knees with osteochondral injury were subject to plain radiography prior to MRI. Skeletal injury was suspected in 13 of these (59%).

Clinical diagnosis

Of the 184 knees with LPD, 60% were correctly diagnosed at the initial visit. Twenty-two percent were diagnosed as either cruciate ligament, collateral ligament, or meniscus injuries, separately or in combination. The remaining were diagnosed as either distortions or contusions.

Consequences of soft-tissue knee injuries (Paper III)

Median age at follow-up was 39 years (IQR, 31-50; range, 19-78). No major differences were observed between the baseline cohort and the follow-up cohort regarding sex distribution, age at injury, injury during sports, or noted earlier injury in either knee. New injuries were most frequently reported after LPD and least frequently after meniscus tear/not ACL (Table 3).

Table 3. Descriptive data of the baseline cohort, the follow-up cohort and the subgroups by injury type.

Injury types: Hemarthrosis alone, no structural injury. LPD, LPD but no ACL rupture. Other injury, other structural injuries including medial or lateral collateral ligament grade 2-3, posterior cruciate ligament any grade, cartilage injuries or fractures but no ACL rupture, meniscus tear or LPD. Meniscus/not ACL, meniscus tear but no ACL rupture. ACL/not meniscus, ACL rupture but no meniscus tear. ACL and meniscus, ACL rupture in combination with meniscus tear.

Earlier injury refers to injury before index injury at baseline according to retrospective data from charts collected per protocol. New injury refers to new injury collected as self-reported data at follow-up visit. IQR, interquartile range. Contralat., contralateral knee. Hema alone, hemarthrosis alone. LPD, lateral patellar dislocation. ACL, anterior cruciate ligament rupture. BMI, body mass index.

	Baseline cohort	**Follow-up cohort**	**Follow-up cohort, injury types**					
			Hema alone	LPD	Other injury	Meniscus not ACL	ACL not meniscus	ACL and meniscus
Individuals, n (% of group baseline)	1129	782 (69%)	70 (56%)	124 (66%)	67 (70%)	70 59%)	197 (74%)	254 (76%)
Median age at injury, years (IQR; range)	26 (17-35; 6-64)	26 (18-37; 6-64)	17 (14-24; 6-58)	18 (15-24; 10-47)	31 (21-40; 12-63)	34 (22-43; 13-63)	30 (22-39; 14-62)	29 (21-38; 12-64)
Female sex	36%	39%	37%	44%	37%	19%	47%	36%
Right knee	52%	51%	41%	56%	43%	50%	49%	55%
Injury during sports	72%	72%	63%	52%	61%	60%	85%	82%
Earlier injury index knee	24%	24%	19%	27%	24%	26%	18%	29%
Earlier injury contralat.	14%	16%	7%	16%	12%	19%	18%	16%
New injury index knee,	n/a	30%	23%	54%	28%	15%	24%	32%
New injury contralat.	n/a	23%	24%	34%	19%	37%	14%	22%
Median time to follow-up, years (IQR; range)	n/a	12.1 (10.9-14,0; 7.9-17.5)	13.9 (12.1-15.5; 10.5-17.5)	10.2 (8.8-11.9; 7.9-17.2)	12.3 (11.2-14.6; 8.9-16.8)	14.1 (11.8-15.5; 10.5-17.2)	12.0 (11.0-13.5; 8.8-17.4)	12.3 (11.0-14.1; 8.9-17.2)
Median BMI at follow-up (IQR, range)	n/a	26 (24-29; 16-47)	25 (22-28; 20-40)	26 (23-31; 18-47)	27 (25-30; 18-44)	27 (24-29; 18-40)	26 (24-29; 18-41)	26 (24-29; 16-43)

Cumulative incidences of radiographic and symptomatic knee osteoarthritis

At a median of 12 years after traumatic soft-tissue knee injury with hemarthrosis, 34% (95% CI, 30-37) of the injured knees and 18% (95% CI, 15-20) of the contralateral knees showed radiographic signs of OA. Twenty percent (95% CI, 17-23) of the cohort had symptomatic OA with symptoms as reported in KOOS and radiographic signs of OA in the injured knee. No sex differences were observed in the development of radiographic or symptomatic OA (Table 4).

Table 4. Incidence proportions of radiographic knee osteoarthritis (OA) and symptomatic knee OA, respectively*.

*There were two persons with missing KOOS data and one who opted to have radiography of the injured knee only. TF, tibiofemoral. PF, patellofemoral. N/a, not applicable. KOOS, Knee injury and Osteoarthritis Outcome Score.

	Injured knee		Contralateral knee	
	n	%, (95% CI)	n	%, (95% CI)
Males				
Radiographic knee OA	170/483	35 (31, 40)	87/483	18 (15, 22)
Radiographic TF OA	122/483	25 (22, 29)	53/483	11 (8, 14)
Radiographic PF OA	131/483	27 (23, 31)	74/483	15 (12, 19)
Symptomatic knee OA	99/481	21 (17, 24)	n/a	n/a
Females				
Radiographic knee OA	93/301	31 (26, 36)	51/300	17 (13, 22)
Radiographic TF OA	73/301	24 (20, 29)	36/300	12 (9, 16)
Radiographic PF OA	68/301	23 (18, 28)	39/300	13 (10, 17)
Symptomatic knee OA	56/301	19 (15, 23)	n/a	n/a

An ACL rupture in combination with a meniscal tear inferred the worst outcome regarding radiographic OA compared to the contralateral knee with an incidence proportion of 50% (95% CI, 44-56) in the injured knee and 20% (95% CI, 16-26) in the contralateral knee. Slightly lower incidence proportions of knee OA were seen after an ACL rupture without concomitant meniscus tear (32% (95% CI, 26-39) vs 18% (95% CI, 13-24)), while all other injury types resulted in low risk differences between the injured and the contralateral knees (Figure 12).

Any structural knee injury as compared to hemarthrosis alone without any relevant structural injuries resulted in higher incidence proportions of radiographic knee OA, (36%; 95% CI, 33-40) versus (6%; 95% CI, 2-14) (Appendix, Suppl Table 1). Tibiofemoral and patellofemoral compartments seemed to be equally affected by OA development in the injured knee, as well as in the contralateral knee. Contrary to what could be expected, LPD patients had less PF OA development at follow-up, 15% (95% CI, 10-23), than all other injury types except hemarthrosis alone. An ACL injury combined with meniscus tear had the highest incidence proportion of PF OA at 36% (95% CI, 30-42).

Radiographic tibiofemoral knee OA was only seen in 2 of 70 individuals or 3% (95% CI, 0-10) with hemarthrosis alone compared 103 of 256 or 40% (95% CI, 34-46) after ACL injury with meniscus tear (Figure 13).

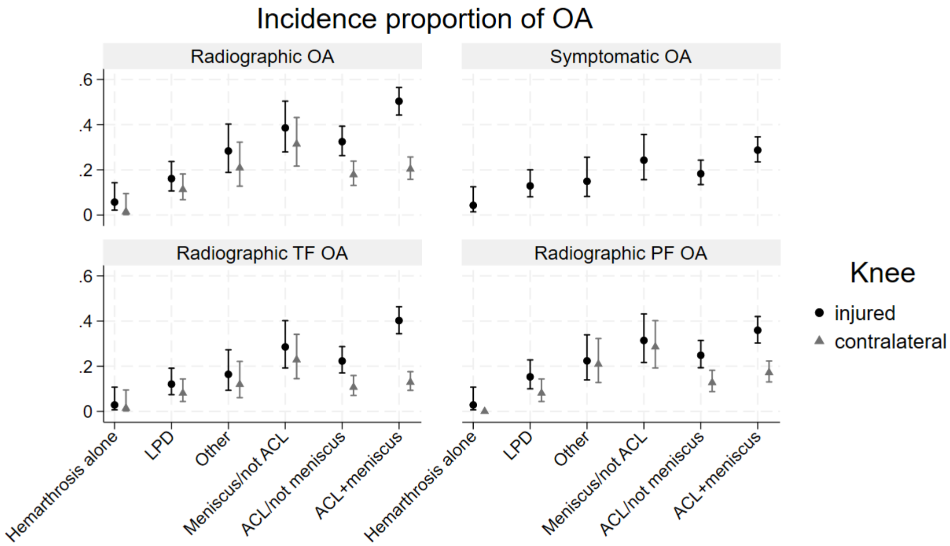

Figure 13. Incidence proportions of radiographic and symptomatic knee OA by injury type with 95% confidence intervals and comparisons between index and contralateral knees.
Radiographic OA, OA in any compartment. Symptomatic OA, OA in combination with KOOS scores equivalent with a knee considered symptomatic. KOOS, Knee injury and Osteoarthritis Outcome Score. PF, patellofemoral. TF, tibiofemoral. Please see Table 3 for description of injury types and other abbreviations.

Symptomatic OA was rare in the group with hemarthrosis alone (4% (95% CI, 2-14)). However more prevalent in all other injury groups, especially after ACL rupture and/or meniscal tear, it was most notably recorded after an ACL rupture with concomitant meniscal tear. This injury combination had an incidence proportion of 29% (95% CI, 24-35) symptomatic knee OA (Figure 12).

Risk ratios of knee osteoarthritis compared to the other knee (within-person) and between injury types (between-persons)

The risk ratio of radiographic knee OA in the injured compared to the contralateral knee was 1.9 (95% CI, 1.5-2.3) in the matched pair analysis. The elevated risk was injury dependent with highest risk after ACL injury with meniscal tear of 2.48 (95% CI, 1.80-3.42) and lowest after meniscal tear without concomitant ACL injury, 1.23 (95% CI, 0.70-2.15). However, that is excluding hemarthrosis where there was virtually no radiographic OA (n=4), and no analysis could be made (Table 5). Adjustments for earlier knee injuries before inclusion or new knee injuries during the time to follow-up as reported by the patients did not change the results but marginally (Appendix Suppl Table 2).

Table 5. Within-person comparisons of injured versus contralateral knee and between-persons analyses comparing the specific injury types to hemarthrosis alone. Matched pair analysis in a fixed effects Poisson regression model and crude risk ratios from a Poisson regression model with robust standard errors adjusted for the logarithm of follow-up time, respectively.
*Hemarthrosis alone not counted as a structural soft-tissue injury. TF, tibiofemoral, PF, patellofemoral. OA, osteoarthritis, RR, risk ratio. CI, confidence interval.

	Within person versus contralateral knee		Between persons versus hemarthrosis alone	
	Radiographic knee OA			
	RR crude	95% CI	RR crude	95% CI
Any structural injury*	1.9	1.5, 2.3	7.8	2.9, 20.9
LPD	1.4	0.7, 2.8	4.1	1.4, 12.2
Other injury	1.4	0.7, 2.7	5.7	2.0, 16.1
Meniscus/not ACL	1.2	0.7, 2.2	6.8	2.5, 18.7
ACL/not meniscus	1.8	1.2, 2.7	6.7	2.5, 18.2
ACL and meniscus	2.5	1.8, 3.4	10.1	3.8, 27.0
	Radiographic TF OA			
Any structural injury*	2.2	1.7, 2.8	11.7	2.9, 47.3
LPD	1.5	0.7, 3.3	6.3	1.4, 28.2
Other injury	1.4	0.6, 3.4	6.6	1.5, 29.2
Meniscus/not ACL	1.3	0.7, 2.4	10.1	2.4, 42.0
ACL/not meniscus	2.1	1.3, 3.5	9.3	2.3, 38.2
ACL and meniscus	3.1	2.1, 4.6	16.2	4.0, 65.5
	Radiographic PF OA			
Any structural injury*	1.7	1.4, 2.2	12.2	3.0, 49.0
LPD	1.9	0.9, 4.1	8.6	2.0, 37.6
Other injury	1.1	0.5, 2.2	9.2	2.2, 39.5
Meniscus/not ACL	1.1	0.6, 2.0	11.2	2.7, 46.1
ACL/not meniscus	1.9	1.2, 3.1	10.7	2.6, 43.8
ACL and meniscus	2.1	1.5, 3.0	14.9	3.7, 60.1
	Radiographic and symptomatic OA			
Any structural injury*	n/a		6.4	2.0, 20.2
LPD	n/a		5.2	1.5, 18.6
Other injury	n/a		4.2	1.2, 15.0
Meniscus/not ACL	n/a		5.8	1.7, 19.0
ACL/not meniscus	n/a		5.4	1.7, 17.6
ACL and meniscus	n/a		8.1	2.6, 25.8

Comparing individuals with different structural injuries to those with hemarthrosis alone, the RR for radiographic OA was 7.8 (95% CI, 2.9-20.9) in the between-persons analysis (Table 5). Risk ratios were highest for a combined ACL and meniscal injury at 10.2 (95% CI, 3.8-27.0). Also, this combination gave the highest RR of symptomatic OA at 8.1 (95% CI, 2.6-25.8) compared to hemarthrosis alone.

Analyses of OA development after any of the injuries LPD, ACL rupture and/or meniscus tear compared to other injuries were made with adjustments for all measured confounders (Appendix Suppl Table 3). Only knees with an ACL rupture in combination with a meniscal tear were at increased risk of knee OA with an RR of 1.9 (95% CI, 1.3-2.7), and symptomatic OA with RR 2.2 (95% CI, 1.3-3.6).

Patient-reported outcome and satisfaction

A lateral patellar dislocation was associated with the lowest scores in KOOS, TAS and knee satisfaction, but by small numbers (Figure 14). As compared to hemarthrosis alone, lower scores were seen after LPD in the KOOS subscale for knee-related function in sports and recreational activities (-13.3 (95% CI -24.4, -2.2)) and QoL (-14.8 (95% CI -23.8, -5.8)) as well as Tegner activity scale (-1.0 (95% CI -1.6, -0.39)) and knee satisfaction score (-0.90 (95% CI, -1.75, -0.05) after adjustments for age, sex, if injury during sports participation and logarithm of follow-up time (Appendix Suppl Tables 4a and 4b).

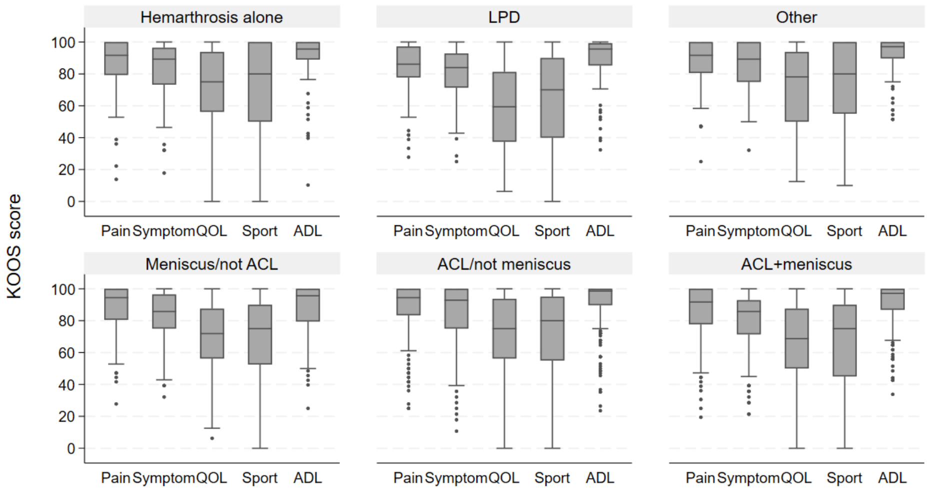

Figure 14. KOOS subscales by injury type. Means and standard deviations.
KOOS, Knee injury and Osteoarthritis Outcome Score. ADL, activities of daily living. QOL, quality of life.

Individuals with ACL rupture combined with meniscus tear also scored worse than hemarthrosis alone for the KOOS subscale of QoL with a mean difference of -10.2 points (95% CI, -18.1, - 2.3), whilst no essential differences were observed for other injury groups. All observed KOOS scores had CIs that excluded differences larger than 10 points and virtually no differences remained after additional adjustment for earlier or new injuries and BMI (Appendix Suppl Tables 4a and 4b).

As for knee satisfaction, four out of five individuals answered that they were happy, satisfied or mostly satisfied with their knee function if they were to spend to rest of their lives just the way it had been over the last week. Lowest scores were seen in the LPD group, 72% (95% CI, 63-79) and the highest in the other injuries group, 91% (95% CI, 82-97). The groups with ACL, meniscus or LPD injuries together with hemarthrosis alone had similar scores (Table 6).

Table 6. Satisfaction with knee function at a median of 12 years after different knee injuries as answered by the question "If you were to spend the rest of your life with your knee function just the way it has been over the last week, would you feel …?"

"Satisfied" includes answering: "Happy", "Satisfied" or "Mostly satisfied". Dissatisfied includes answering: "Mixed feelings", "Mostly dissatisfied", "Dissatisfied" or "Unhappy". CI, confidence interval. See Table 3 for description of injury types and other abbreviations.

	Satisfied (n)	Dissatisfied (n)	Satisfied (%)	95% CI
Hemarthrosis alone (n=70)	56	14	80	69-89
LPD (n=124)	89	35	72	63-79
Other injury (n=67)	61	6	91	82-97
Meniscus/not ACL (n=70)	57	13	81	70-90
ACL/not meniscus (n=197)	163	34	83	77-88
ACL and meniscus (n=254)	206	48	81	76-86
All (n=782)	632	150	81	78-84

Discussion

> “-She tore up her knee in her junior year and tried to play another two games on it.
> -But then didn't she have, um, career-ending surgery?”
>
> Jonathan Franzen, Freedom

This thesis describes the panorama of soft-tissue knee injuries in the general population after a knee sprain with hemarthrosis, as visualised on MRI within a median of 8 days. The incidences, sex differences and seasonal variations of different injuries are explored in detail and the outcomes regarding knee-related health and development of radiographic as well as symptomatic osteoarthritis are evaluated.

Who gets a soft-tissue knee injury, when, and how to find them

Significant structural soft-tissue knee injuries were found on MR images in 90% of the 1145 consecutive cases of knee sprains with hemarthrosis in this study. These results confirm the strong relationship between traumatic knee hemarthrosis and significant structural injury, in both adult and paediatric populations. Mariani et al made a 5-10-year follow-up of 88 out of 92 young athletes who all suffered a traumatic knee hemarthrosis during sports between 1970 and 1975. The clinical examination was negative for serious ligament injury with the anterior drawer test in neutral and external rotation, and varus and valgus laxity at 30° of flexion, as compared to the contralateral knee. They were all treated conservatively with joint aspiration and casting, as was their clinical routine in treating non-serious knee injuries. Plain radiographs were negative, and the examinations were made before the introduction of arthroscopy at the study site. At follow-up, only 9% were without complaints and 80% had either had surgery or experienced dissatisfaction due to residual grave instability. The study high-lights that traumatic knee hemarthrosis often comes with serious structural injuries, even in the absence of initial clinical signs.[164] With the introduction of arthroscopy, multiple diagnostic arthroscopy studies of traumatic knee hemarthrosis have findings of serious structural injuries in 50 to 99%.[15, 16, 18, 20, 24, 72, 73, 165, 166] Still, knee arthroscopy is a surgical intervention with potential risks that should be avoided if there are other means of reaching the same goal. In a study by Sarimo et al of 320 traumatic hemarthroses examined arthroscopically, only 35% had an immediate therapeutic procedure performed in conjunction with the diagnostic arthroscopic examination.[16] The advent of MRI has offered a cost-effective and safe means to an accurate diagnosis of the injured knee with hemarthrosis and the rationality of systematic use of MRI in this setting should be gold standard.[17, 21, 23, 25-28, 85, 92, 167-170]

Traumatic knee hemarthrosis

The occurrence of traumatic hemarthrosis in the knee joint was sex, age, season, and activity dependent in this cohort. The annual incidence of traumatic hemarthrosis in the defined ages at risk between 10 and 59 years was 151 per 100 000 inhabitants with higher numbers for males than females in all age groups. Males were most at risk of a traumatic knee injury with hemarthrosis in their third decade, while females were most at risk in young adolescence between 10-16 years of age. Knee injuries followed the sporting seasons and were most prevalent in the spring months of March and April, while there were less injuries in the holiday seasons of July and December. These results largely followed the seasonal variation shown by Peat et al of clinically diagnosed soft-tissue knee injuries presenting to healthcare facilities in southern Sweden between 2004 and 2012.[71] About three quarters of the knee injuries with hemarthrosis in our study were sports related.

In a study of the epidemiology of knee injuries in northern Sweden by Ferry et al, the incidence of knee injuries was 600 per 100 000 inhabitants including all complaints of knee pain whereof 48% were classified as sprains, strains or dislocations.[69] Knee problems accounted for 8% of all injury-related visits to the ED. In an older study by Nielsen and Yde of all injuries treated at a single ED during a one-year period, 6% involved the knee with an incidence of 120 per 100 000 inhabitants.[68] Peat et al found an annual incidence of 720 clinically diagnosed soft-tissue knee injuries per 100 000 inhabitants at any health care facility including primary care in southern Sweden.[71] In the light of the large numbers of patients with knee problems that seek medical attention, different triage systems have been developed. A widely used system to rule out fractures around the knee is the Ottawa knee rule, that has been proven to lower the number of plain radiographs taken without missing fractures with good diagnostic accuracy.[171-174] However, fractures are only a small part of the knee injuries and most relevant injuries are soft-tissue derangements.[3] In a study of fracture incidences from the Swedish Fracture Register by Bergh et al, the annual incidence of proximal tibia fractures in a Swedish population between 2015 and 2018 has been estimated to 25 (21 for males and 28 for females) per 100 000 in the adult population 16 years or older, and of the distal femur to 9 (5 for males and 14 for females) per 100 000 inhabitants.[175] In similar Swedish populations, comparing the findings by Peat et al and Bergh et al, soft-tissue injuries seems to be 20 times more frequent than fractures around the knee. A significant soft-tissue knee injury, as demonstrated by traumatic hemarthrosis, is likely to be at least five times more common in the general population than fractures around the knee comparing the supposedly similar Swedish populations described by Bergh et al and the one forming the cohort described in this thesis.

Soft-tissue knee injuries have been shown to be an increasing problem by Maniar et al, with a significant growth in annual knee injury incidence for both males and females in Australia between 1998 and 2018.[70] This is explained by general increases in ACL ruptures and knee contusions, as well as PCL ruptures and knee dislocations in females. However, most new, isolated, acute soft tissue knee injuries in the general population are not significant and do not require orthopaedic or surgical attention as shown by Yawn et al, who found 400 acute knee injuries per 100 000 adult inhabitants in Olmsted county, USA.[67] In their study period of 1993-1996, 37% eventually came into an orthopaedic surgeon's care, increasingly so with injury severity and age of the patient. Overall, surgical treatment was recommended for 12% of the subjects.

A triage system for identifying acute soft tissue knee injuries in need of specialist care has been proposed by Molloy et al, The Cambridge Knee Injury Tool (Cam KIT) after assessing the diagnostic challenges in acute soft tissue knee injuries.[176] This tool has 12 variables of yes/no questions rendering a maximum of one point per question and was recently tested with a sensitivity of 100%, and a specificity of 89% where 117 of 229 knee injuries that presented to the ED were classified as low risk that could be managed by advice and education only.[177] In their cohort, 30% of the presentations to the ED had a clinically significant injury. Of the patients referred for specialist consultation, 56% were confirmed to have a significant injury and 34% required surgical intervention. The identification of traumatic hemarthrosis was not an item in their tool, but virtually all the items represent typical signs thereof including: global swelling, rapid swelling, inability to bear weight, reduced range of motion, reported instability (giving way, shifting), reported twisting (or pivoting mechanism), reported popping (cracking or tearing), reported hyperextension, high risk activity, noncontact mechanism, and previous soft-tissue knee injury.

In our setting, identification of a traumatic knee hemarthrosis as a qualifier for a subsequent sub-acute knee MRI and multi-disciplinary conference where further management is decided by sub-specialists, could be a cost-effective and safe way to find the significant soft-tissue knee injuries in need of further care. Regardless of whether further care may be orthopaedic surgery, specialised physiotherapy or patient education.

Soft-tissue knee injuries in males and females

Up to the age of 40, there was a male predominance for all injury types in this cohort. In older ages, injury rates were similar between males and females except for meniscal tears where higher incidences were seen in males. This finding may, however, be open for discussion since some of the meniscal tears found on MRI could be non-traumatic or chronic by nature and therefore represent a prevalence of earlier meniscal injury, rather than an incidence.[178] For clinically diagnosed soft-tissue knee injuries, Peat et al found a shift from higher incidences for males to higher incidences for females around the age of 45 years.[71]

Similar patterns were seen in the Australian longitudinal study by Maniar et al, but with a later shift around 60 years of age.[70] They also noted that males had greater incidences of acute knee injury than females, but with a closing gap over time. ACL injuries seemed to have the fastest annual growth, especially among young females (5-14 years). This is in line with a report from Finland, where they noted increasing annual incidences of paediatric ACL injuries between 1997 and 2014, with a two-fold increase the last 10 years of the study period and a significant increase of annual incidences in girls aged 13-17 years.[179] A similar finding was made by Beck et al in a study on insurance billing data from Minnesota of a total 3303 ACL injuries in patients aged between 6 and 18 years from 1994 to 2013.[180] In these young ages they noted a higher incidence of 129 ACL injuries per 100 000 person-years in females compared to 114 in males. In their study, the peak incidence for females was found at the age of 16 with 392 ACL injuries per 100 000 person-years, while males peaked at the age of 17 with 422 ACL injuries per 100 000 person-years. Lastly, a Swedish study from the national patient registry of 56 659 patients with cruciate ligament injuries between 2002 and 2009 also noted peak incidences for females in the second decade of life at 144 per 100 000 inhabitants and in the third decade for males with an incidence of 225 per 100 000.[88]

In a Salt Lake City retrospective cohort study of cumulative all-cause knee injuries in 3964 cisgender and 1971 transgender patients, Miro et al found a higher incidence of knee injuries among both cisgender men (5.6%) and transgender women who had received gender-affirming hormone therapy (6.6%) compared to cisgender women (4.1%).[181]

Similarly, the overall incidence of ACL injuries is higher for males in this study of both paediatric and adult patients in line with other reports.[68, 71, 88, 89] Consistent with other studies of young patients though, females had a higher annual incidence of ACL injury between 10 and 19 years of age (89/100 000 person-years) than males in the same age-span (79/100 000).

Soft-tissue knee injuries are sports-related

In an injury surveillance study of 498 Spanish athletes over one year, 40% sustained an injury and the most common anatomical injury sites were ankles (36%) and knees (19%), with highest injury rate during football (soccer).[182] Considering that most of the knee injuries in our study (72%) were related to sports activities and half of these being football which was associated with 36% of all injuries, an individual's risk of a knee injury is closely linked to sporting habits. The exception was LPD, with only around half (53%) of the injuries occurring during sports, reflecting the sometimes more atraumatic nature of LPD, while more than 80% of ACL and MCL injuries were sports related. Annual ACL injury incidence is higher among athletes than in the general population, and substantially higher in professional sport.[183] Medial collateral ligament injuries, however, have been shown to be the most common ligament injury in male high-level football players.[184]

We found that medial collateral ligament injuries were more frequent during alpine skiing, accounting for 22% of these injuries compared to 15% of the ACL injuries and only 5% of the LPD. Especially so in females over 30 years of age, with 55% of all MCL injuries in that group being sustained during alpine skiing compared to 17% for females in younger ages. There was no similar pattern in males. Even though alpine skiing is the second most common sports activity leading to a traumatic hemarthrosis in this study (11%), the study site is far away from any ski resort and most alpine skiing is traditionally a recreational sport, exercised one week yearly. One can speculate that some of these individuals are mothers on holiday for recreational skiing with their family, perhaps distracted from their own skiing with focus being on their off-spring learning to ski. Injuries to the MCL have earlier been proposed to be more common during skiing than other sports.[185] In a series of 100 consecutive ACL ruptures, Fridén et al also noted a high prevalence of concomitant MCL injuries in ski accidents (22/30) as compared to contact sports (23/59), but with very few bicompartmental meniscus injuries with skiing. This led to the hypothesis that ski injuries mostly were distraction injuries while contact sport ACL injuries more often was a compression injury.[186] However, since the introduction of carving skis, there seems to be a decline in MCL injuries with the majority of recreational alpine ski injuries now involving the ACL.[187, 188]

Sex-related soft-tissue knee injuries in sports

Female elite athletes have been proposed to have an up to eight times elevated risk of ACL injury compared to male athletes practicing the same sport.[189, 190] Prodromos et al reported in a meta-analysis about 3 times greater incidence of ACL injuries among female athletes in football and basketball as compared to male athletes.[191] Recreational alpine skiers had the highest incidence of ACL injuries, while expert skiers had the lowest. However, they did not find any sex differences for ACL injuries among alpine skiers.

To investigate the risk of injury related to exposure of the activity inflicting the risk, the concept of athlete-exposure (AE) is commonly used. An athlete-exposure is usually defined as one athlete participating in a practice or game in which there is a risk of sustaining an injury or the actual number of hours that an individual participates in practice or game. The concept is naturally a bit vague and can under- or overestimate exposure time when comparing injury rates between different sports.[192] It has recently been pointed out that the concept of AEs can be treacherous in structurally overestimating the rate of injury per AE in females.[193]

There were no available data on AE in the present cohort, but there was a Swedish national survey on sports activity in the general population between 7 and 70 years available from the time (FoU-rapport 2004:5; The Swedish Sports Confederation; Sweden). Approximately 20% of males and 10% of females reported competitive or recreational participation in one of the three main ballgames involved in traumatic hemarthrosis in this study, namely football, floorball and team handball. Given that males seemed to have a larger exposure, it can be suspected that young female athletes have a higher risk of ACL injury during sports than male. Waldén noted higher incidences by two to three times for females when related to time of exposure to football in a study of female and male Swedish and male European top football clubs, as well as in a review of the literature.[194, 195] They also found that females were significantly younger at injury than their male counterparts. A similar finding was seen for cruciate ligament injuries in a large Finnish cohort of 46 500 adolescents and young adults reported by Parkkari et al, with twice the risk of injury for female athletes compared to male athletes.[196]

A meta-analysis on studies that control for athletic exposure by Montalvo et al in 2019 revealed that one in 29 female athletes and one in 50 male athletes injured their ACL over 25 years.[197] The relative risk of an ACL injury for a female athlete was 1.5 as compared to male athletes. They found 1.9 ACL injuries in 10 000 female AEs while the male athlete incidence was 0.9 per 10 000 AEs. Overall, female athletes had a 1.7-fold increase in incidence rate of ACL injury. The differences in rates of ACL injury between sex were independent of level of sports participation.

To further investigate the sex- and sports-related risk of ACL injuries in young athletes specifically, Bram et al conducted a systematic review and meta-analysis on studies from 1990 to 2019 reporting on ACL injuries per athlete-exposure or sport exposure time (hours) in athletes between the ages of 12 and 19 years.[198] They evaluated more than 1 200 ACL injuries in almost 18 million athlete-exposures (AE) and found a significantly higher rate of ACL injuries in girls, with an RR of 1.4 (95% CI 1.2-1.6) as compared to boys. The RR for an ACL injury during match relative to practice was 8.5 for girls and 6.9 for boys. Between different sports the ACL injury risk per season ranged from 0.11% for female track and field to 1.21% for female football contrasted to 0.03% for male track and field, 0.39% for male football, and 0.82% for male American football. The sports with highest risk regarding incidence of ACL injury per AE for girls were football, gymnastics, lacrosse and basketball while American football, lacrosse, football and wrestling inferred the greatest risk for boys. Comparable to the above mentioned meta-analysis by Prodromos et al, Bram et al found RRs of ACL injury for female versus male athletes of 3.1 in soccer and 4.1 in basketball.[191] They concluded that a multi-sport female would have a nearly 10% risk of ACL injury over the high school and secondary school career or a risk of 1 ACL injury per 10 000 AEs. A 2025 systematic review and meta-analysis by Childers et al on the same topic and within the same age-span (12-19 years of age) but more focused on sport affiliation, concluded that the risk of ACL injury was 1.56-fold greater in adolescent female athletes compared to male athletes and the sport with the highest risk for females in football.[199]

In a 2023 publication, Mancino et al pointed out that female participation in sport has grown 11-fold over the last 50 years.[189] The number of women and girls playing football are continually rising.[200] In women's elite football, thigh muscle injuries have been found to be the most common, but ACL injuries result in the highest injury burden with lost days in their sport with a median of 292 days.[201] Montalvo et al reviewed the literature and conducted a meta-analysis regarding ACL injuries in football players.[202] The incidence of ACL injury among all football players was 1.7 per 10 000 AEs, with a 2.2-fold increase in incidence rate for female as compared to male players (2.2 and 0.9 ACL injuries per 10 000 AEs, respectively) regardless of participation level. A study among German football players have shown a reduced athletic performance at return to play after ACL injury for all level of play and longer time to return to competition especially in lower playing levels and after re-ruptures.[203] The career of professional football players has also been reported to be shorter, and with less play time after ACL reconstruction compared to matched uninjured controls.[204, 205]

Modifiable and unmodifiable risk factors of knee injury

It seems clear that female athletes have an increased risk of ACL injury compared to male athletes playing the same sport, especially in contact sports like basketball and football.[206] Return to the previous level of sport has also been shown to be less frequent for female athletes compared to their male counterparts (OR=1.4).[207] Factors leading to greater susceptibility for females of ACL injury that have been studied include anatomical, physiological and biomechanical differences to males.[208]

Some possible risk factors of knee injury, where ACL ruptures and re-ruptures are most studied, are naturally unmodifiable or questionable to modify. These include intrinsic elements, inherent to the individual, such as hormonal influences, anatomical variations of bony or ligamentous structures in the knee, and generalised joint hypermobility. Other intrinsic risk factors are possibly modifiable, such as landing, pivoting biomechanics and neuromuscular control. Extrinsic risk factors of knee injury are also more accessible to modification and include factors like shoe design, playing surface, the weather conditions that the athlete acts in, and the sport that is being played including the amount it is being exercised.[209]

Unmodifiable intrinsic risk factors of knee injury

Our genetic composition is naturally unmodifiable. Twin studies by Magnusson et al have shown approximately 40% heritability for meniscal tears and an even higher genetic contribution to the risk of ACL rupture during the lifespan, with around 69% heritability.[210, 211] In the world's largest twin registry, 1395 ACL ruptures in 88 414 monozygotic and dizygotic twins over the age of 17 were investigated over 1-30 years. The familial risk was higher in monozygotic twins than dizygotic twins and the findings implied an increased risk by 20-140% of ACL rupture compared to the population if a twin sibling has had one.[211] Bram et al studied familial ACL injuries among 450 paediatric patients that underwent ACL reconstruction and found a nearly 3 times higher odds of having a first-degree relative with an ACL tear for the ACL injured patients compared to uninjured controls.[212]

Less modifiable intrinsic risk factors of knee injury

Variations in sex hormones have been attributed to the risk of ACL injury.[213] Two systematic reviews of the literature on the effect of the menstrual cycle phase on the risk of ACL injury have concluded that although there seems to be an alteration in knee laxity during the menstrual cycle phases associated with changes in knee joint loading, there is no evidence of a particular phase in the menstrual cycle predisposing eumenorrheic and naturally menstruating females to greater risk of noncontact ACL rupture.[214, 215] Hence, they recommend caution in manipulation of physical preparation, injury mitigation, or screening practises related to the menstrual cycle based on existing evidence. However, a 2025 study from Colorado comparing the incidences of ACL reconstructions in two million females using systemic hormonal contraceptives with 12 million non-contraceptive using females aged 15-35 years, found lower incidences among contraceptive users with incidences of 79/100 000 and 120/100 000, respectively.[216] The association was stronger with progestin-only oral contraceptives. Surprisingly, after age-stratification of the data, no association was found between the use of oral contraceptives and an elevated incidence of ACL reconstructions for females between the ages of 15 and 19 years.

A variety of anatomical variations within and around the knee have been proposed as risk factors for ACL injury. The most studied bony variation is the sagittal slope of the proximal tibia, referred to as the posterior-inferior tibial slope (PITS).[217, 218] In a study of 100 mainly non-operatively managed ACL injuries that were only reconstructed in case of repeated giving-away episodes, the PITS angle was greater in individuals who had undergone reconstruction than others.[219] High tibial osteotomy aiming to reduce the tibial slope has been proposed in patients with a graft rupture and a slope above 12 degrees with good results and no re-tears at long-term follow-up of nine patients.[220] A matched case control study of 90 ACL injured American football players with measurements of medial and lateral condylar width, medial and lateral plateau width, notch width, bicondylar width, notch width index and medial and lateral tibial slopes revealed increased lateral tibial slope as the sole independent predictor of ACL injury in the multivariable analysis with 32% increase in risk for every degree of increased slope.[221] Another comparative study performed in Turkey of 352 male patients going through ACL reconstruction matched with 350 noninjured individuals investigated 32 previously determined measurements and 7 calculations, with contrasting results. They found significant differences between the groups regarding a wide range of parameters.[222]

A narrow intercondylar notch has also been associated with increased risk of ACL injury.[223-225] A systematic review of the literature by Bayer et al, found intercondylar notch stenosis, variations in sagittal shape of the femoral condyles, increased tibial slope, reduced tibial eminence size, poor tibiofemoral congruence, and small ACL size to be substantial risk factors for ACL injury.[226] Factors not linked to ACL injury were notch height, condylar and plateau width measurements, quadriceps (Q) angle and ACL length. However, the clinical implications of this knowledge are not obvious. As pointed out in a commentary to the article by Davila, if a tibial slope of >12° is associated with a significantly greater risk of ACL rupture and the highest reported mean slope is 10.9° there is a very narrow gap between normal and when to recommend a deflexion osteotomy.[227] The mean medial posterior tibial slope in a German cohort of 327 ACL reconstructed knees with 52 graft failures after a mean of 17 months was 10.6° ±3.2° in patients with graft failures and 11.2° ±2.8° in patients without failures.[228] There was no difference in the frequency of graft failures between patients with PITS <12° and those with PITS ≥12°. A review from 2024 of the literature on ACL reconstruction and deflexion osteotomy included six studies with a total of 133 patients and concluded vaguely that this combined approach may be effective in decreasing the PITS as well as improving knee function and stability.[229]

Another recent systematic review found evidence for increased risk of both ACL injury and ACL graft failure with increased PITS.[230] This was true for both the medial and lateral tibial slope. Voskuilen et al made a similar review of the literature of morphological risk factors for ACL injuries in skeletally immature patients and also found smaller notch width and tibial slope to be relevant.[231]

Conversely, a decreased tibial slope has been associated with primary PCL ruptures in both paediatric and adult populations as well as PCL reconstruction failures.[232, 233]

The muscle force vector of the quadriceps muscle over the knee in the frontal plane is thought to play a role in knee injury and is represented by the Q angle, the angle between a line from the anterior superior iliac spine to the centre of the patella and a line from the centre of the patella to the tibial tuberosity.[234] The Q angle is related to femoral anteversion and knee valgus angles in healthy controls.[235]

Alpay et al found that ACL injured patients had a greater anteversion of the femur than non-ACL injured controls and that this torsion was infratrochanteric rather than supratrochanteric.[236] There were only 5% females in their study, and they could not identify any differences related to sex. The hypothesis is that an increased femoral anteversion leads to a compensatory internal rotation of the hip and a valgus movement in the knee joint, also shown by Kaneko et al.[237] Amraee et al found, in a retrospective study of 53 male athletes with a history of ACL injury compared to accepted standard values, that increased femoral anteversion and decreased active hip internal rotation and ankle dorsiflexion correlated to ACL injury.[238]

However, to investigate whether anteroposterior knee laxity, knee hyperextension, generalised joint hypermobility, femoral anteversion, hamstring extensibility, or navicular drop were associated with noncontact ACL rupture in female team sport athletes, Pasanen et al examined 287 individuals at baseline and screened for injury during 2-4 years.[239] No significant differences in the measured variables at baseline were detected in the 23 individuals that injured their ACLs during the study time, compared to the uninjured individuals. Generalised joint hypermobility, defined as a Beighton score ≥5 out of a 9, has been associated with slightly lower rates of return to sports.[240]

Kamatsuki et al studied a cohort of 870 elite female handball and football players prospectively and found 64 noncontact ACL ruptures.[241] They could not find any correlation between most of the anthropometric measurements they made of limb alignment, joint laxity, leg length, knee alignment, knee anteroposterior laxity, generalised joint hypermobility, genu recurvatum, hip anteversion and the risk of ACL injury. However, increased static knee valgus was associated with greater risk of noncontact ACL injury, especially for a secondary injury. Additionally, hyperextension was a risk factor for secondary injury. Generalised joint hypermobility has earlier been shown to increase the risk of a second ipsilateral or contralateral ACL injury within 12 months after return to sport after ACL reconstruction by more than five times compared to patients without generalised joint hypermobility.[242]

The game of football seems to increase the varus angle in male footballers' knees, an observation not made in females or other high activity sports.[243, 244]. A study on adolescent male football players showed a marked increase in genu varum from the age of 14 compared to healthy controls.[245] Knee varus is not a known risk factor of knee injury but is implicated as a risk factor for knee OA.[246-248]

In summary, studies on hormonal and anatomical risk factors in soft-tissue knee injury are conflicting and the use of any modification strategies are questionable.

Knee injury prevention – Modifiable intrinsic risk factors

Despite extensive research on sex-differences in ACL injury and prevention programs, the incidence rates of ACL injuries in females do not seem to lower over time.[70, 89, 179, 180, 249-251] Concomitant injuries with an ACL rupture are very common and have been associated with younger ages.[252] However, multiple studies have shown protective effects of 10-15 minute prevention programs on the risk of knee injury in both male and female athletes and a 2018 meta-analysis of overlapping meta-analyses evaluating the effect of prevention training programmes summarised the risk reduction of noncontact ACL injury overall to 50% for all athletes and to 67% for female athletes.[253-258] The Swedish knee control programme has shown a 64% risk reduction of ACL injuries in female youth football players, a 45% risk reduction of lower limb injuries in male and female youth floorball players, and 31% reduction of both acute and gradual onset self-reported knee injuries in handball.[259-261]

Noncontact ACL injuries have been shown to stand for about 70% of all ACL injuries, but the rate is activity dependent. In male basketball, up to 100% are primarily noncontact injuries, about 85% in male football and less than 50% in rugby.[62, 262, 263] With a majority of ACL ruptures being noncontact or indirect contact injuries, they are potentially avoidable with prevention programs.[264, 265] Studies on mechanisms of injury, particularly video analyses, are the foundation in developing strategies of injury prevention.[62, 263, 266-273] Injuries have been found to usually occur during a cutting manoeuvre (change of direction) or one-leg landing manoeuvres.[267, 269]

An umbrella review from 2022 evaluating the effectiveness of ACL injury prevention programmes in young female athletes confirmed a reduction of ACL injuries by 64% and suggested that a multi-faceted program from the pre-season period with at least three different exercises may reduce the risk of ACL injury.[274] Preventive programmes in female athletes have been found to have greater effect in younger ages.[275]

Sports with high risk of knee injuries involve natural elements of direction change in order to create space, evade an opponent and score goals.[276] Changes of direction have been noted to occur every four to six seconds in sports like netball, football, and land hockey.[277-279] Up to 700 of these movements have been observed during a 90-minute game of football.[280-282] Donelon et al reviewed the literature and made a meta-analysis of 17 studies on differences of knee joint load between males and females without prior history of ACL injury during change of direction. Females had significantly less peak knee flexion during the stance phase in changes of direction and greater knee abduction at initial contact, which has been associated as visual characteristics of noncontact ACL injury in video analyses. However, no differences in knee joint loads were observed between males and females.[283] The meta-analysis also revealed less hip internal rotation, and less hip abduction in females during change of direction (cutting) tasks.

Additional evidence for the benefit of prevention practice was presented in a 2024 study of 376 adolescent female football players, without prior significant knee injuries, assessed at baseline regarding one-leg jump tests, range of motion and strength in the trunk, hip, and ankle. The athletes were followed weekly for one year with observations of new knee injuries and the volume of matches and training. Nine-teen percent reported at least one new substantial knee injury. The strongest association to injury was seen with low knee extension strength with a hazard rate ratio of 2.3 for knee injury, compared to the strongest athletes.[284] Also, strong evidence was found for higher peak knee abduction angles in post-pubertal compared to pre-pubertal female athletes in a 2024 review of the current evidence on the changes in kinematic and kinetic risk factors associated with ACL injuries during jump landing in female athletes at different stages of maturity.[285] Lower relative peak vertical ground reactions force, higher external knee abduction moment and higher internal rotation moment was also seen in post-pubertal female athletes compared to pre-pubertal female athletes.

An other example of modifiable risk factors of soft-tissue knee injury that has been studied is dietary habits, that was shown to affect the incidence of knee and shoulder injury in adolescent female but not male handball players.[286] Psychological competitiveness has also been shown to increase the risk of knee injury in a Japanese study of 300 female high-school athletes.[287] Thus, there is continually more evidence on potentially modifiable risk factors being published but so far injury prevention programs with neuromuscular and proprioceptive training have shown the most promising results.[288]

Knee injury prevention – Modifiable extrinsic risk factors

The rotational traction (grip) in the shoe-surface interface varies depending on the shoe model and higher rotational traction has been implied to double the risk of lower extremity injuries in American football.[289, 290] A study on the risk of ACL injury in football players in America related to the turf played on showed a significantly larger risk on natural grass (1.16/10 000 AEs) versus artificial surface (0.92/10 000 AEs).[291] Female players had an 11 times greater and males 3 times greater risk of ACL injury during practice on natural grass compared to practice on artificial turf but they did not find any difference in risk during match. However, recent systematic reviews on the topic show conflicting results, both suggesting that the risk is similar and that there is a lower risk of knee injury while playing on artificial surface compared to natural grass.[292-294]

The amount of practice and the ratio between practice and match are other potentially modifiable risk factors of knee injury. In a study of 679 adolescent handball players who reported any new knee injuries in addition to the volume of practice and competition, female athletes had a 30% increased handball-related knee injury risk the week after large increases (>60%) in handball volume as compared to female athletes with a <20% increase or decrease over the last four weeks, while the increased risk in matched male athletes was more modest (10%).[295] More time engaged in vigorous sports activity per week has been associated to increased risk of self-reported ACL ruptures in a longitudinal study of boys and girls aged 9-15 years, where 2.4% and 2.0% sustained an injury, respectively.[296] In boys, overweight or obesity was also a significant predictor of ACL injury.

In a 2024 review of the literature regarding extrinsic risk factors of primary noncontact ACL injury in adolescents between the ages 14 and 18 including type of sport, amount of sport exposure, sport level, sport season, environment, and equipment, Crotti et al concluded that it was not possible to perform a meta-analysis due to the low number of studies per extrinsic risk factor and the discrepancies in definition of injury.[297] They did not find any conclusive evidence that adolescents who participate in different sports have greater or lesser risk of ACL injury. The same conclusion applied to if the amount of sport exposure, the sport level, sport equipment or the phase of sport season were associated with the risk of ACL injury. Early sports specialisation in adolescent athletes has also been suggested as a potential risk factor of injury.[298]

One can conclude that the road to knee injury is paved with multifactorial and complex intrinsic and extrinsic risk factors.[299]

What do they get

Traumatic bone marrow lesions

Traumatic bone marrow lesions (bone bruises) were found on sub-acute MRI in 84% of the 1145 knees with traumatic hemarthrosis in the baseline cohort. Impaction fractures and BMLs can be considered a footprint of the injury and a tell-tale of what happened.[147, 300-303] Bipolar bone contusion BMLs on the central part of the lateral distal femur and the posterior lateral proximal tibia are considered classical signs of ACL injury and lateral-sided BMLs on either the femur or tibia have been reported in more than 80% of patients with complete ACL ruptures.[304-306] Injuries to the ACL sustained during alpine skiing has shown a slightly different pattern, with less BMLs on the lateral femoral condyle, compared to injuries in other sports with a possible higher impact load in non-skiing injuries.[307]

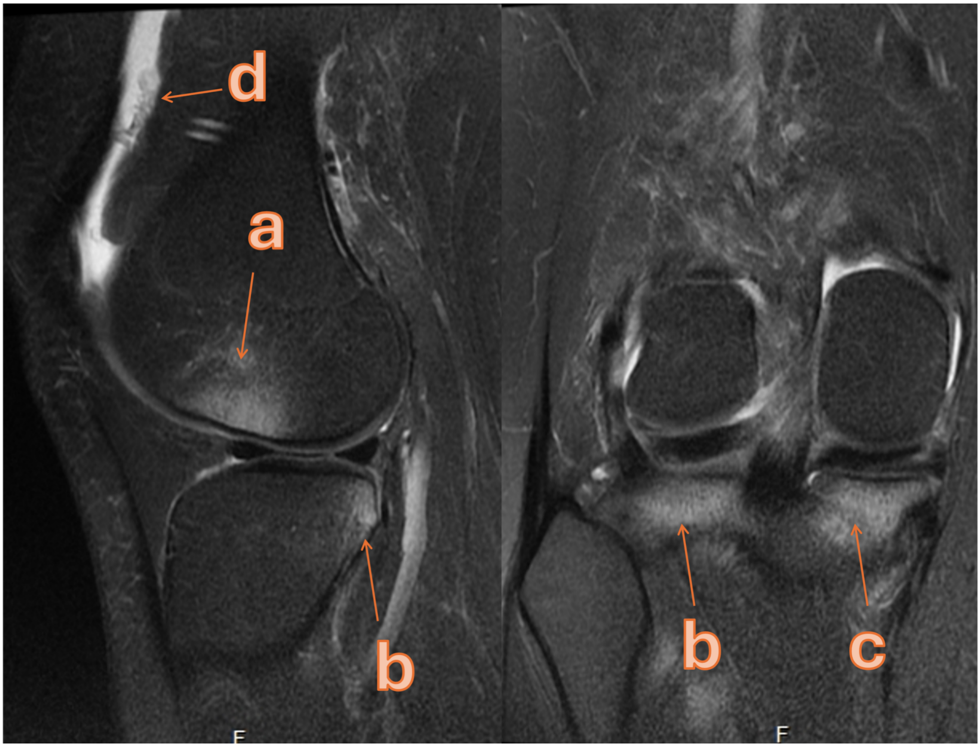

Figure 15. Bone marrow lesions (BMLs)
Traumatic BMLs on MRI after ACL rupture in the lateral femoral condyle (a), the posterolateral tibial condyle (b) and posteromedial tibial condyle (c). Note also the hemarthrosis (d).

The BMLs in our study were noted according to their location but not quantified. Bone marrow lesions and impaction fractures were frequent findings in the ACL injured knees (92%) and 90 % had BMLs on either the lateral tibial or femoral condyles (Figure 15). Classic bipolar BMLs, on both the lateral femoral and tibial condyles, were seen in 61% of ACL injured knees. However, 45% of the knees with intact ACL and no sign of LPD also had BMLs in the lateral compartment, either on the tibial or femoral side. This subgroup was similar in age and rates of meniscal injuries to the group with ACL rupture and could be suspected to have had similar trauma mechanisms as the ACL injured knees, but the ligaments stayed intact. Even though not noted in our study, Baessler et al has suggested that this is more common in skeletally immature patients.[308] They found 269 patients with classical bipolar lateral BMLs after exclusion of individuals with prior ACL reconstruction, fractures, and contusion patterns consistent with LPD in a retrospective MRI database study. Among patients with an open distal femoral physis, this pattern was only associated with an ACL rupture in 89% of the cases, as compared to 99% in patients with a closed physis. Anterior translation has been shown to decrease with age in childhood and adolescence and to be associated with greater global joint laxity and female sex.[309] Hence, individuals with greater ligamentous laxity can experience a pivoting of the knee with an anterior translation of the tibia and typical bone contusions without tearing the ACL, as visualised on MRI.

Traumatic BMLs, also called bone marrow edema-like lesions or bone bruises, are usually defined as a change of the signal intensity of the bone marrow due to trauma as seen by MRI on T1- and T2-weighted images, preferably with fat suppression and short tau inversion recovery (STIR) sequences and were first described in the late 1980's.[310] These are differentiated from bone marrow lesions caused by stress concentration in osteoarthritic knees.[311, 312] The main cause of traumatic BMLs is the direct impact of the joint surfaces in conjunction with shear stress in dislocation of the tibia relative to the femur during ligament rupture or an external contusion.[301]

The impact causes a disruption of subcortical bone trabeculae with accumulation of interstitial fluid and haemorrhage and occur on a spectrum from bone bruising to impaction fractures with displacement of cortical and subchondral bone as well as chondral or osteochondral fractures (Figure 16).[303, 313-315]

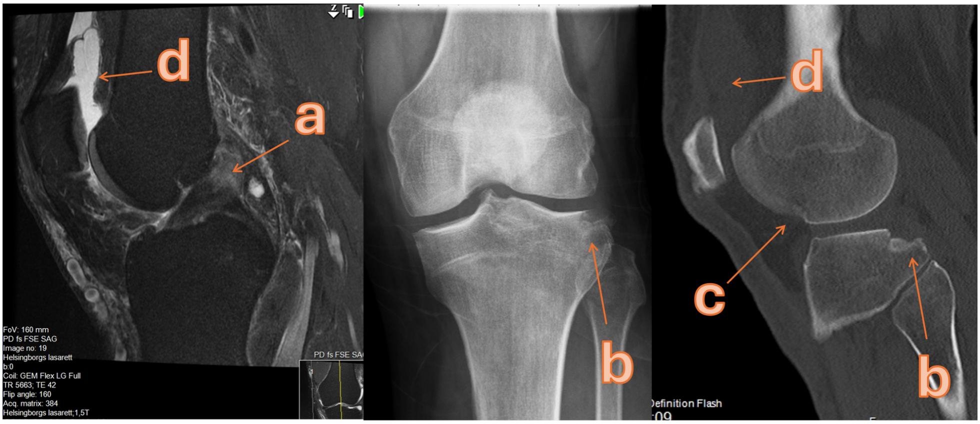

Figure 16. Impaction fractures in an ACL injured knee.
A knee with a proximal ACL injury (a) on MRI and hemarthrosis (d). Plain anteroposterior radiograph showed an impaction fracture in the lateral tibial condyle (b). A computer tomography revealed an osteochondral impaction fracture with cortical disruption (b) and a soft impaction fracture without chondral or cortical disruption in the lateral femoral condyle (c).

Impaction fractures were noted in 240 of the 1145 knees (21%) in the baseline cohort of this study. The ACL injured knees had an impaction fracture in 31% of the cases as compared to 10% in the knees with intact ACL. Overall, the impaction fractures were found in the lateral compartment in 92% of the cases (Figure 16). Posterolateral tibial plateau impaction fractures, as noted on MRI, was reported by Bernholt et al in 49% of patients with primary ACL ruptures in a study of 825 knees and were increasingly associated with lateral meniscus posterior horn root tears related to the size of impaction.[316] They further noted a lateral femoral condylar impaction fracture in 26% of the knees. Traumatic bone marrow lesions were present in the lateral tibial plateau in 77% of the knees and in the lateral femoral condyle in 49%.

There is a bone bruise – so what?

The question on how to treat BMLs in clinical practice has been under discussion for a long time. In a 2014 review of the natural history of bone bruises, Nakamae et al argues for delaying the return to full weightbearing when post-traumatic MRI reveals a severe bone bruise.[317] Filardo et al found a high prevalence of bone bruising after ACL injury in the literature, around 78% in most recent studies, with a distinct pattern related to the mechanism of injury but conclude that prospective long-term studies are needed to better understand the nature and impact of BML.[318] Further, Ward et al did not see any convincing evidence to correlate BML to functional outcomes or the development of OA in their 2022 review of the literature regarding BMLs in the setting of an ACL injury.[319] They speculate on whether the best clinical use of the presence of bone bruises in ACL injured knees may be as a marker of other injuries, such as ramp lesions in the medial meniscus or anterolateral ligament ruptures, that are not always visualised on MRI, rather than advising on weight-bearing or not. In fact, Familiari et al recently found a correlation between medial sided BMLs and ramp lesions, a meniscus tear not necessarily visualised during standard arthroscopy or MRI.[320] Ramp lesions have been reported to occur in 25-50% of all ACL injuries.[321] Injuries to the anterolateral ligament have also been linked to typical BML patterns.[322, 323]

However, Kia et al found a significant relationship between the size of initial BML at the time of injury and the predicted further development of cartilage injury in a retrospective medical chart review of 40 patients with an acute ACL injury and pre- and post-operative MRIs.[324] No BML was the strongest predictor of a grade 0 Outerbridge score while a large BML in the lateral femoral condyle conferred a 74% risk of a grade 3 or 4 Outerbridge score at five years. On the other hand, Galloway et al could not see any correlation at medium-term between BML volume and self-reported return to play, International Knee Documentation Committee (IKDC) score or a battery of objective functional performance tests in 60 patients at two years.[325] These findings were basically in line with previous work by Latterman et al who related preoperative BML volume and severity with KOOS and IKDC scores after 2 and 6 years in 81 patients undergoing ACL reconstruction.[326] They did not find any clear correlation between BML severity and inferior outcomes, even though a subgroup of 17 patients with bone bruises in combination with articular cartilage injury seemed to have worse symptoms than the ones without such damage at six years.

Traumatic BMLs tend to be present for a long time. The natural history of traumatic BMLs as visualised on MRI has been studied by Boks et al who followed 80 patients with post-traumatic BMLs with MRI until they could no longer be discerned or one year and found a median healing time of 42 weeks.[327] Roemer and Bohndorf found that six of 80 post-traumatic BMLs in 49 patients with acute knee trauma persisted after a minimum of two years.[315] In a study of nine ACL reconstructed patients with MRI within 8 weeks of injury, followed by additional MRI scans at 2 weeks, 6 months and 12 months post-operatively, Theologis et al found that around 50% of the traumatic lesions had resolved over one year but the overlying cartilage exhibited persistent changes.[328] In a review of the literature from 2015, Papalia et al concluded that BMLs yield higher pain levels but could not state that they had an impact on functional outcomes.[329]

The severity of the BML is correlated to the severity of the soft-tissue knee injury. In a study of post-traumatic BMLs on MRI, pre-operative PROMs, and intra-operative findings including a complete kinematical assessment with a surgical navigation system in 29 patients, Agostinone et al found that severe bicompartmental BMLs were associated with greater intraoperative knee laxity.[330] They also found a direct connection between the severity of BML on the medial tibia and the degree of pivot shift as well as the severity of BML on the lateral femoral condyle and the KOOS subscale for knee-related symptoms.

The degree of a posterolateral impaction fracture has also been associated with an increased likelihood of having a high-grade pivot shift laxity at the time of ACL reconstruction.[331] In the same study of 825 ACL injured knees undergoing reconstruction who had available MRIs, worse postoperative outcomes in the Lysholm score after a minimum of two years were related to the severity of the posterolateral tibial plateau impaction fracture. Impaction fractures on the articular surfaces, especially with cortical disruption has been shown by Swärd et al to induce an increased inflammatory response in the knee.[332]

The level of bony impact related to soft-tissue knee injuries with hemarthrosis seems to matter. Both as an indicator of the severity of the injury and subsequently the severity of future impairment, but to date, there is no clear evidence to guide management of BMLs or impaction fractures.

Structural knee injuries in hemarthrosis

Our demonstrated injury distribution could be regarded as a fairly accurate panorama of which significant soft-tissue injuries to be expected in the general population after knee trauma with hemarthrosis. At the time, virtually no private health care alternatives were available and the project, that aimed to include all paediatric or adult knee-injured patients in a well-defined catchment area, was well known in primary health-care facilities and among physiotherapists. To get an MRI in due time, a resource that was otherwise not readily available and hence could be considered a strong incentive, the patient either had to present at the ED or be referred to the orthopaedic out-patient clinic. Further, with peak incidences of soft-tissue knee injuries overlapping paediatric and adult health care, not discriminating on age is another circumstance that adds to the validity of these findings.

In an early Swedish study of 84 knees in 79 patients admitted to the ED with hemarthrosis or a locked knee, Gillquist et al found during arthroscopy and examination under anaesthesia a total or partial ACL rupture in 59% of the knees, a PCL rupture in 3%, a total or partial MCL ruptures in 59%, meniscus tears in 23 %, osteochondral fractures in 10%, and an LPD in 3%. They also found a rupture of the plica synovialis in 10% of the knees, which was presented as a novel finding in 1977.[73] In the contemporary and similar works by Dehaven and Noyes et al, but with the exclusion of knees with clinical instability at first examination, both groups found 72% total or partial ACL injuries at arthroscopy and lesions of surgical significance in up to 90% of the knees with a large proportion of combination injuries.[15, 24]. Hardaker et al also evaluated traumatic hemarthrosis in patients with minimal clinical instability and found partial or complete ACL ruptures in 77% of the knees and osteochondral fractures in 8% of the knees. Similar to the above-mentioned studies, the ACL rupture was highly associated with concomitant injuries, e.g. a meniscal tear in 61% of the cases.[166]

A later Finnish study, questioning the need for routine arthroscopy in every traumatic knee hemarthrosis, evaluated 320 consecutive patients between 1994 and 1996 in one hospital. As they pointed out, this was in a time when the availability of MRI generally was low and, at least in their setting, the availability to diagnostic arthroscopy was good. They found an ACL rupture in 45% of the knees, which was associated with an MCL rupture in 29% and with a meniscus tear in 28% of the cases. In 23% of the patients the hemarthrosis was caused by an LPD. In total 81% of the patients had an ACL rupture, LPD, MCL rupture, or a meniscal tear, in isolation or combination. Loose intra-articular bodies were found in another 3% of the knees.[16] They found similar incidences of injuries to those in an earlier Finnish study with the same design but came to a different conclusion.[333] Since only 35% of the knees had a therapeutic intervention (loose bodies and meniscal tears) at the time of the diagnostic arthroscopy, they advocated that there is no need for a purely diagnostic arthroscopy but rather that the patient is diagnosed via non-invasive means and followed by an orthopaedic surgeon.

The early studies of diagnostic arthroscopy in traumatic hemarthrosis aimed at sparing the patients the larger intervention of an arthrotomy, if the arthroscopy could discern only the patients in need of open surgical intervention. Gillquist et al found in their study that they could avoid an arthrotomy in 31% of the cases, changed their planned surgical procedure in 37% (for example from open ACL surgery to open meniscus surgery), and that the arthroscopy was only unnecessary in the 32% of the cases when it did not change the surgical plan.[73] As mentioned, several of these studies in the 1970’s and 1980’s were carried out on patients with no or negligible ligament instability where the clinical tests at initial examination did not reveal any significant structural injury.[15, 24, 166] Evolving from diagnostic arthroscopy being a safer way to diagnose a knee sprain with traumatic hemarthrosis than both a clinical examination and an arthrotomy, an MRI of the knee with a traumatic hemarthrosis seems to be even safer with the same diagnostic outcome, but without the need for follow-up by an orthopaedic surgeon of every patient.

The relative incidence of different knee injuries in hemarthrosis

A traumatic hemarthrosis is an accurate indicator of a high probability of a significant structural intraarticular injury and especially ACL rupture. The annual incidence of a traumatic hemarthrosis was higher for males than females across all ages in the baseline cohort of this study, but the relative incidence of an ACL rupture in the instance of a traumatic hemarthrosis was higher for females than males in all age groups. In females, 57% of all knees with a traumatic hemarthrosis involved an ACL rupture as compared to 50% in males. The highest relative incidence for an ACL rupture was seen in females between 30-39 years of age (73%). The greatest sex-related difference was seen in ages between 16 and 19, where females had a relative ACL rupture incidence of 61% (n=55/90) with traumatic hemarthrosis as compared to 39% (n=62/161) for males. The relative incidence of an MCL injury grades 1-3 was roughly similar between males and females across ages with an overall occurrence in 28% of all knees with hemarthrosis but steadily increasing over ages from 13% to around 50%. Meniscal tears, on the other hand, were more common in males with a traumatic hemarthrosis from the age of 20. In individuals under the age of 16, LPD was the most common injury with a relative incidence of 43% for girls and 39% for boys.

The findings confirm that young females have a higher risk of significant ligament injury in the event of a traumatic knee hemarthrosis.

It’s the ACL

If a traumatic hemarthrosis is identified in the ED, about 50% is likely to be an ACL rupture. Hence, a guess on an ACL rupture in all soft-tissue knee trauma with hemarthrosis at the first visit would render much better diagnostic accuracy than the results that can be found in the literature. In our cohort, ACL ruptures that were first examined by an emergency doctor only had a 20% chance of getting a correct diagnosis as compared to 95% if examined by a knee sub-specialist.[22] In a British study published in 1996, less than 10% of the patients with an ACL rupture were diagnosed at the first visit, the mean delay to diagnosis was 21 months and 30% of the patients had seen an orthopaedic surgeon without the diagnosis being made prior to presenting to a knee specialist.[334] A similar British study published in 2016 found little progress over the 20 years that had passed with 14% diagnosed at the first visit.[335] The median number of visits before correct diagnosis was 3 (range, 1-10) and median delay from injury to diagnosis 13 weeks (range, 0-926). In Brest, France, Guillodo et al studied the results of a standardised knee examination of 79 consecutive patients aged 15 to 55 years conducted by an emergency physician and compared the findings with the examination by a sports medicine specialist 5±2 days later. The emergency physician noted a suspected rupture of the ACL in seven of the 27 cases noted by the sports medicine specialist (26%). All 27 ruptures were later confirmed by MRI.[336]

In a recent review of the literature on the initial assessment and diagnostic pathways for patients with suspected ACL injuries, Allott et al included six studies with MRI or arthroscopy as gold standard and found that using imaging modalities took less time to reach diagnosis (two appointments) and that clinical tests are highly subjective in assessing acute knee injury.[337] For some reason, the authors conclude that early radiological verification is unattainable for this patient group. Unsurprisingly, the introduction of a specialised acute knee clinic improves accuracy of clinical tests and the time to diagnosis compared to before such a routine.[338, 339] However, it is doubtful that this set-up is more economical and effective than sub-acute MRI since the injured patient most likely will be diagnosed via an MRI ultimately.[29] Aspiration of a traumatic hemarthrosis in the ED has shown pain relief, ROM restoration, and higher sensitivity of the physical examination at follow-up in a retrospective study of reconstructed ACL injuries.[340] As shown by diagnostic arthroscopy and also in this study, very few traumatic hemarthroses will be absent of clinically significant injuries (<10%) even in the absence of clinical instability.[15, 24, 165, 166]

The incidence of Anterior Cruciate Ligament rupture

The annual incidence of ACL injuries in this cohort was 77 (95% CI, 70-85) per 100 000 inhabitants in the defined ages of risk between 10 and 59 years. This is in line with other reports on the general population (Table 7).[68, 71, 88, 89, 196, 341]

The incidence of ACL injuries has been suggested to be rising in younger individuals in the US and in the general population, but especially young females, in Australia over time.[70, 180] These trends have been attributed to multiple factors such as increased participation in high-demand year-round sports at an early age, increased female athletic participation, more single sport athletes in young ages, better clinician awareness, and the expanded role of MRI in the diagnosis of intra-articular injuries.[342] Conflicting trends have also been reported from the US by Bergstein et al with a substantial decline in ACL injury incidence from 90 ACL tears per 100 000 person-years in 2010 to 53/100 000 in 2020.[341] Sanders et al also noted a decline in ACL incidence, but only for males, between 1990 to 2010.[89] Maybe peak-ACL has been reached, at least in the USA.

Table 7. Reported annual incidences of ACL injuries in the general population. ACL injuries per 100 000 person-years (95% CI)

STUDY	Country	Cohort	Overall	Males	Females
Nielsen and Yde 1991	Denmark	Hospital-based cohort. 1986, n=28	30	40	20
Parkkari et al 2008	Finland	Population-based cohort 1987-1997, n=256	61 (54-68)	97 (83-110)	30 (23-37)
Nordenvall et al 2012*	Sweden	Public Health-care register. 2002-2009, n=56 659	78 (77-78)	-	-
Peat et al 2014*	Sweden	Public Health-care register. 2004-2012	71 (61-80)	86 (74-98)	56 (49-63)
Sanders et al 2016**	USA	Historical cohort. 1990-2010, n=1841	69 (65-72)	82 (77-87)	55 (51-59)
Paper I 2016	Sweden	Prospective clinical MRI cohort ages 10-59. 2002-2008. n=599	77 (70-85)	91 (80-103)	63 (53-73)
Bergstein et al 2025	USA	PearlDiver insurance register. 2010-2020, n=931,186	75	-	-

*Cruciate ligament injuries, including PCL. **Only isolated and complete ACL tears (but ACLs with concomitant meniscus tears, articular cartilage injury and non-operated collateral ligament tears inlcuded).

The choice to manage an ACL tear operatively or non-operatively depends largely on the individual patient's preferences and goals for postinjury activity level, but also the demographic situation of the patient. For highly active patients in jumping, cutting, and pivoting sports, early anatomical ACL reconstruction is recommended to avoid secondary meniscus and cartilage injuries according to an international consensus group of ACL experts.[343] However, a period of progressive rehabilitation to address impairments and improve neuromuscular function is advised. The rate of operative treatment for ACL injuries differs remarkably. One 2013 study from Boston, USA, reported that about one quarter of all ACL injuries underwent a reconstruction while a 2016 study from Minnesota reported 62% reconstructed within the first year of ACL injury and 32% of the non-operated within the following nine years.[344, 345] Nordenvall et al described the rate of reconstructed ACL injuries in Sweden to 30% within one year and another 10% during the following eight years.[88] The probability of being operated on was slightly higher for females than males. Several studies have noted that the incidence of ACL reconstructions seems to be rising over time.[89, 251, 341, 346, 347]

It's a combination

It is likely beneficiary to identify an MCL rupture expediently after trauma in order to treat it with a brace, either initially or as the final treatment. This should be especially important in grade 3 injuries.[348] A guess on an ACL rupture and to make sure that the knee is stable in valgus and varus and, if not, treat it with a brace, is a safe way to get by until a subacute MRI can verify the diagnosis and further diagnose any potential meniscal or osteochondral injuries in need of acute surgery. Bearing in mind that most soft-tissue knee injuries are a combination of injuries, a suspected ACL injury can hide anything from an incarcerated bucket handle meniscus tear, a meniscus root avulsion or a deep radial tear to an osteochondral fracture after a misdiagnosed LPD in need of early surgery.

In our baseline cohort, 55% of the ACL ruptures were combined with a meniscal tear, 39% had a concomitant MCL injury, and 40% had more than one associated injury. Over 80% of all significant injuries (ligament injuries and meniscal tears) had concomitant injuries.

Counting only major ligament injuries, i.e. ACL, PCL, MCL grades 2-3 and LCL grades 2-3, 28% (n=195/692) were combination injuries (Table 8). Fetto and Marshal found in 1978 that 53% of MCL grade 2 ruptures and 78% of grade 3 ruptures were combination injuries and that 74% of the grade 3 ruptures were associated with an ACL rupture.[117] In this cohort, 69% and 68% of the MCL injuries grades 2 and 3, respectively, were in combination with an ACL rupture.

Table 8. Major ligament injuries, isolated and in combinations. Number of injuries and proportion of index injury.

MAIN	**Isolated**	**In combination with**							
INJURY		ACL	MCL 2-3	LCL 2-3	PCL	MCL+ PCL	MCL+ LCL	LCL+ PCL	MCL+ LCL+ PCL
ACL (n=599)	409 (68%)	-	151 (25%)	15 (3%)	7 (1%)	7 (1%)	8 (1%)	1 (0%)	1 (0%)
MCL 2-3 (n=244)	72 (30%)	151 (61%)	-	0 (0%)	5 (3%)	-	-	0 (0%)	-
LCL 2-3 (n=29)	4 (14%)	15 (52%)	0 (0%)	-	0 (0%)	0 (0%)	-	-	-
PCL (n=33)	12 (36%)	7 (21%)	5 (15%)	0 (0%)	-	-	0 (0%)	-	-

The treatment of combined ACL and MCL injuries has been debated. Studies have shown both more and less ACL revisions if the MCL is treated non-operatively.[349, 350] A review of the current literature on the topic from 2024 could not find any difference in outcomes between non-operative treatment, ligament repair or ligament reconstruction of the MCL in the conjunction with an ACL reconstruction.[351] Hence, they recommended a delayed decision in the management of these combination injuries to allow for the majority of MCL tears to heal but stressed early repair for avulsion injuries or Stener-type lesions. The mainstay of treatment for isolated MCL injuries of higher grades is range of motion bracing, however the current evidence for non-operative treatment is low and there is no evidence in how to brace patients according to a recent review of the literature by Svantesson et al.[352] On the other hand, the same review concludes that the evidence on when surgery is needed also is of low quality and does not offer guidance to clinicians. Generally, bracing is recommended for grade 3 injuries for six weeks, especially proximal ruptures that have been shown to confer good healing potential.[353] Grade 1 and most grade 2 ruptures can be treated without a brace, where immediate weight bearing and ambulation is allowed as tolerated. Some injuries may be recommended for acute surgery, such as a the mentioned Stener-like lesion with the MCL superficial to the pes anserinus, a positive dial test with anteromedial rotatory instability and bony avulsions.[352-354] The adherence to knee bracing has recently been investigated in grade 2 and 3 MCL injuries with or without a concomitant ACL injury and found to be 75% in the first two weeks, 64% in the two weeks after and 59% in the last two weeks in a six week protocol.[355]

A retrospective study from Singapore reviewing the post-traumatic (within 6 weeks) MRI in 304 patients who had ACL reconstruction in a single unit found no differences in associated intra- or extra-articular injuries between males and females at the time of ACL injury.[356]

Can it be a lateral patellar dislocation

The most common soft-tissue knee injury in children up to the age of 15 years is an LPD.[17, 18, 85] This has been suspected to be the most commonly neglected major acute soft-tissue knee injury. An early study on patients from the cohort in this material revealed that the diagnosis was overlooked in 50% of the cases.[19] However this improved over time, probably with greater awareness of the injury, to 62% equalling the diagnostic accuracy of diagnosing an ACL rupture.[22] A reason for this could be that traumatic hemarthrosis is less frequent in paediatric patients and that clinical examination has been suggested to be more challenging in paediatric patients with knee sprains than others.[357, 358] In a study on traumatic hemarthrosis in a paediatric population under the age of 15 years in Sweden by Askenberger et al, 27% of the LPD patients were not aware of their patella dislocating and 56% of the patients with a major injury had a clear diagnosis after clinical examination and plain radiographs.[85] The historical gold standard of diagnosing soft-tissue knee injuries is arthroscopy. However, the signs of an LPD with BMLs in the anterolateral portion of the lateral femoral condyle and medial patella together with a rupture of the medial patellofemoral ligament are characteristic but easily missed during arthroscopy, especially when the history from the patient is unclear.[25, 148-152, 359]

With LPD potentially being difficult to distinguish from other acute knee injuries with traumatic hemarthrosis, and arthroscopy not always diagnostic, the use of MRI in the acute phase after knee injury may increase diagnostic precision. MRI findings are easily discernible and can identify LPDs that might otherwise be overlooked, as well as detect injuries needing early surgical intervention.[28] Patellar dislocations are traditionally claimed to represent 3% of all knee injuries.[360] This is consistent with data reported in studies from both a large American population by Gage et al, a Swiss athletic population by Majewski et al, and Scandinavian populations by Ferry et al and Nielsen and Yde.[3, 68, 69, 361] Although they report on 'dislocations' most of these should represent LPDs.

In this study, 17% of the 1145 traumatic hemarthroses were traumatic LPDs (n=199) in the baseline cohort, which align with other studies on knee sprains with hemarthrosis.[16] These were re-evaluated to identify the first-time dislocations. Age and sex specific annual incidence of primary LPD and concomitant chondral and osteochondral injuries were analysed.

The epidemiology of first-time LPD

An overall annual cumulative incidence of 14 primary LPD per 100 000 person-years with significantly higher incidences in all ages among males compared to females was noted in this cohort. The incidence was highest at ages 13-15 years for both boys and girls (135 and 114 per 100 000 person-years, respectively).

A first-time (primary) LPD, also called a first-time patellar dislocation (FTPD) has recently been defined by the European Society of Sports Traumatology, Knee Surgery and Arthroscopy (ESSKA) in a formal consensus statement as follows:[362]

"FTPD is the first time event when the patella completely leaves the trochlear groove. The event has to be confirmed clinically and/or radiologically."

The incidence of LPD has been studied in several different populations and age-groups (Table 9).[71, 79-87, 363] However, most of these reports are based on clinically diagnosed injuries rather than MRI findings and reported incidences may be underestimates. Incidence estimates from a geographically well-defined population where all identified traumatic knee injuries (i.e. possible LPDs) underwent an early MRI assessment could represent a more accurate estimate.

Incidences were higher for males except in the youngest ages (10-12 years) in this study. Earlier reports have consistently reported higher incidences for females but a Finnish study on male military conscripts by Sillanpää et al noted one of the highest numbers so far, 77/100 000 person-years as opposed to 58/100 000 in the same male age group (18-23 years) in our material.[82] Not counting studies of military populations[82, 83] we report the largest fraction of male dislocators (59%), particularly among boys in ages between 16 and 18 years (73%). However, a large study of an estimated more than 200 000 cases of patellar instability from the National

Electronic Injury Surveillance System (NEISS) in the USA published by Kell et al in 2025, also found that males accounted for 59% of patellar dislocations between 2001 and 2020.[364] They, among others, have noted increasing numbers of admissions for LPD over the first two decades of this millennium.[363, 365, 366]

For completeness regarding incidence numbers on LPD reported in this thesis in relation to other publications (Table 9), some additional calculations are given. Fithian et al and Gravesen et al reported incidences in the age group 10-17 years.[86, 367] The corresponding incidences in this material was 75 (95% CI, 55-99) per 100 000 person-years in females, 90 (95% CI, 68-115) for males, and 82 (95% CI, 68-100) for both. The corresponding incidences for the groups reported by Askenberger et al was, 66 (95% CI, 51-85) per 100 000 person-years in ages 9-14 and 108 (95% CI, 81-141) in ages 12-14 years.[85] The age group of 14-18 years had the highest incidences as reported by Sanders et al.[87] In this cohort, that age group conferred an incidence of 76 (95% CI, 51-108) per 100 000 person-years for females, 111 (95% CI, 82-148) for males and 94 (95% CI, 74-118) for both. Finally, Lyons et al reported recently NEISS register incidence data from EDs in around 100 hospitals in the USA selected as a probability sample of all EDs.[363] Highest incidences rate was noted for females aged 10-14 years and for males 15-19 years. Corresponding incidence rates in this cohort was 89 (95% CI, 62-124) for females, and 65 (95% CI, 43-95) for males aged 10-14. In the age group 15-19 years incidence rates were 49 (95% CI, 30-77) for females and 105 (95% CI, 76-141) for males.

Large register-based studies seem to yield lower incidence rates and sometimes a greater ratio of females. In contrast, clinical studies like this one and another Swedish study on paediatric patients by Askenberger et al with MRI-controlled diagnosis of first-time acute LPDs report higher incidence rates with greater ratio of males.[85] Askenberger et al noted a fraction of 56% males in a young population aged 9-14 years, and with twice as many boys than girls in the sub-group between 12 and 14 years. One can hypothesise that LPD is considered a female diagnosis and, thus, the diagnosis of LPD after knee trauma does not come to mind as easily in a male athlete as other differential diagnoses.[368] Possibly, the higher incidence rates in males reflect the superiority of consistent use of MRI in addition to clinical assessment over clinical assessment alone.

Table 9. Incidences of lateral patellar dislocations. First-time events per 100 000 person-years.

STUDY	Country	Cohort	Overall	Males	Females
Nietosvaara et al 1994	Finland	Hospital-based cohort, 1991-1992, n=69	[a]72 [b]107	-	-
Atkin et al 2000	USA	Insurance register, 1992-1994, n=74	[c]7 [d]31 [e]11	- [d]30 (13-47) [e]14 (5-23)	- [d]33(29-37) [e]9 (4-13)
Fithian et al 2004	USA	Insurance register, 1992-1996, n=125	[c]6 (5-7) [f]29 (21-37) [g]9 (4-14)	- [f]25 (16-34) [g]10 (2-18)	- [f]33 (22-44) [g]7 (3-11)
Sillanpaa et al 2008	Finland	Military hospital cohort. 1998-2002, n=72		[h]77 (74-98)	
Hsiao et al 2010	USA	Military register. 1998-2007, n=9299	69 (68-71)* [i]64** [j]54** [k]55**	39*	63*
Waterman et al 2012	USA	National ED register. 2003-2008. n=40 544[#]	[c]2.3	[c]2.5 (1.7-3.2) [l]5 (4-6) [m]3 (2-4)	[c]2.1 (1.6-2.6) [l]5 (3-6) [m]3 (2-3)
Askenberger et al 2014	Sweden	Hospital-based MRI cohort, 2010-2020, n=48	[n]60 [o]120	-	-
Peat et al 2014	Sweden	Public Health-care register. 2004-2012	[c]41 (39-44)	[c]40 (37-42)	[c]43 (40-45)
Gravesen et al 2017	Denmark	National patient registry, 1994-2013, n=24 154	[c]42 (37-47)		[f]108 (101-116)
Sanders et al 2018	USA	Historical cohort. 1990-2010, n=609	[c]23 (21-25) [p]148 [q]54	[c]22 [p]145 [q]52	[c]25 [p]150 [q]55
Paper II 2023	Sweden	Prospective clinical MRI cohort 2002-2008, n=177	[c]14 (12-16) [r]125 (96-160) [s]69 (47-97)	[c]17 (14-20) [r]135 (94-187) [s]97 (62-144)	[c]11 (9-14) [r]114 (76-165) [s]39 (18-74)
Lyons et al 2024	USA	National ED register. 2001-2020, n=159 529[#]	2.6 (2.0-3.1 [d]12 (9-14)	[t]9 [u]15 [v]5	[t]11 [u]10 [v]4

Incidences in ages (years) a) 0-15, b) 9-15, c) all ages, d) 10-19, e) 20-29, f) 10-17, g) 18-29, h) 18-23, i) <20, j) 20-24, k) 25-29, l) 0-19, m) 20-39, n) 9-14, o) 12-14, p) 14-18, q) 19-25, r) 13-15, s) 16-18, t) 10-14, u) 15-19, v) 20-24. * Adjusted for age, race, service, and rank. ** Adjusted for gender, race, service, and rank. [#] Estimated numbers.

First-time lateral patellar dislocation and sports

Sports was the most common eliciting activity (55%), and more so for males (65%) than for females (40%), confirming other studies.[80-82, 84, 85, 367, 369] However, the pronounced sex difference has not been shown before. In fact, more primary LPDs in females occurred during leisure or non-specific activities than during sports in this cohort. As with other soft-tissue knee injuries, football was the most common sport during LPD for both males and females which is in line with the report by Askenberger et al.[85] Mitchell et al reported an overall patellar instability rate (dislocations and subluxations) of 2 per 100 000 AEs in US high school athletes.[370] They found highest risk in girls' gymnastics followed by boys' American football, boys' wrestling, and girls' football. Competition conferred a higher risk than practice.

Kell et al noted that the increase in incidence rates of primary LPD in their study was driven by spikes during the early stages of the sporting seasons in autumn (September-October) and spring (April-May), in males, and in patients aged 10-18.[364] However, Lyons et al reported an equal increase in sports-related and non-sports-related, as well as male and female LPDs from the same time-period.[363]

One can assume that an LPD during sports would involve more impact force and a higher risk of concomitant chondral or osteochondral injuries. This could not be seen in this cohort, where the risk of such injuries where equal during sports and other activities. Chondral injuries were twice as common for males during sports (61%) than for females (31%), only reflecting the sex-dependent pattern of activity during injury. Less than half of the osteochondral fractures were associated with sports injuries.

Concomitant injuries and treatment with first-time LPD

Injury to the joint surfaces was noted in 43% of the knees with primary LPD. Others have reported cartilage injuries in 34-95% using MRI and/or arthroscopy as diagnostic tool.[25, 80, 148, 151, 168, 371-377] Good to very good agreement has been reported between arthroscopy and MRI in diagnosing cartilage lesions Outerbridge grades 3 and 4 in primary LPD, but poorer for milder injuries.[167, 378]

Eighteen percent of the knees with a primary LPD had an osteochondral fracture, in line with the 12% of knees with osteochondral fracture reported by Askenberger et al[85]. The patella and the lateral femoral condyle were roughly equally involved, with slightly more so on the patella in line with most other reports[377, 379-381]. No sex-dependence could be detected on the rate of chondral injuries in contrast to a recent report from Zheng et al.[382]

Surgical treatment is generally recommended if a traumatic LPD is complicated by an osteochondral or chondral injury, and a concomitant MPFL reconstruction has been recommended in this setting.[383] Plain radiographs and MRI, or MRI alone, should be carried out in the acute phase in order to rule out any chondral and osteochondral lesions and hemarthrosis should act as a warning sign.[362] In our study more than four out of ten osteochondral fractures were not suspected on plain radiographs. Surgical reinsertion is advocated, if possible, usually if the fragment is larger than 1 cm^2.[384] Excision and, when needed, microfracture procedures are recommended for fragments without potential for healing, but chondral-only fragments have lately been reported to have potential for healing after re-insertion.[385]

The recurrence rate is high after a primary LPD. Fithian et al reported in 2004 a 17% risk of recurrence after first-time dislocation and 49% after a recurrent event.[367] Palmu et al found a 71% re-dislocation rate over a mean of six years in patients aged less than 16 years at their first LPD.[386] In the absence of an osteochondral fracture, non-operative treatment is often considered gold standard after a first episode of an acute patellar dislocation. This is, however, under debate and among others the latest ESSKA consensus statement recommends this to be questioned for skeletally mature as well as immature patients in favour of an individual analysis of risk factors.[362, 383, 387, 388] Hinckel et al proposes in a state of the art review from 2025 that surgical intervention is recommended in the presence of osteochondral fractures requiring internal fixation or fragment removal, in patients at high risk of re-dislocation, and in patients with persistent functional instability.[389] As goes for non-operative treatment, there is no clear evidence to support any sort of bracing or casting as opposed to no bracing, in either the acute or nonacute phase of an LPD.[383, 390, 391] According to an individualised 'à la carte' approach as developed mainly in Lyon, a recurrent patellar dislocation is usually an indication for surgical treatment involving MPFL reconstruction in conjunction with bony procedures as needed.[368, 389, 392, 393]

Patients under the age of 16 years at their first LPD had a much higher risk of a primary contralateral LPD during the study period (7%) compared to those aged 16 years or older (1%). Numbers were small, but similar to those reported by Gravesen et al.[86] It is probable that the younger group would have more anatomical risk factors (e.g. trochlear dysplasia, greater sulcus angle, patella alta and elevated tibial tubercle-trochlear groove [TT-TG] distance) than the older group.[394, 395] However, it was not in the scope of this thesis to delve in to anatomical variations in the cohort.

What are the consequences

Surgery or no surgery

First of all, it must be stated that the included studies are purely observational. The follow-up cohort has been analysed as treated, with no account taken to the management of the injuries at baseline and thereafter. No information is presented on the rate or technique of cruciate ligament reconstructions, meniscus surgeries, collateral ligament injury management, and patellar instability or chondral injury operations. Likewise, no record of the non-operative treatment is presented. With the observational design, any such attempt could be suspected to be inherently biased and not add much information on the outcome on group level. The patients in the cohort received individualised treatment according to the best available knowledge at the time and the wishes of the individual, as part of normal clinical practice. This was true except for the sub-group that was included in the KANON trial (n=95/814), where no essential difference between the treatment-groups was found at eleven years.[104]

Obviously, there are pros and cons regarding not distinguishing between non-operative and operative management in the analyses of different traumatic knee injuries. However, all patients are different, and individualised treatment tailored to the specific needs of the patient is common everyday practice. The decision was made to not further divide patients into groups according to treatment. It could be argued that there is only a weak link between standardised surgery decisions and outcomes for most soft-tissue knee injuries.[95, 104, 115, 343, 352, 396-415] Each treatment decision should be unique and based upon a wide array of data, including the specific characteristics of the patient and the injury, in addition to be anchored in current evidence.[416] Further, the ultimate treatment is highly dependent on the individual care-giver.[417]

Post-traumatic knee osteoarthritis

Post-traumatic radiographic knee osteoarthritis

The follow-up results with a 3-fold increase of radiographic knee OA in the injured knee compared to the contralateral knee over a median of 12 years, are quite similar to the ones by Barenius et al in their 2014 report on knee OA prevalence at a mean 14 years after ACL reconstruction randomised to semitendinosus or bone-patellartendon-bone autograft.[418] They found a Kellgren-Lawrence grade ≥2 in 57% in the ACL-reconstructed knees and 18% in the contralateral knees. KOOS-values were lower for patients with knee OA in their report, and meniscus resection was a strong risk factor for OA. However, they did not see any differences between graft choice or time from injury to surgery.

The development of knee OA in the presented large prospective cohort of consecutive acute knee injured patients with hemarthrosis, was highly dependent on the type of initial injury as identified on MRI (Figure 17). The incidence proportion of radiographic knee OA in the injured knee ranged from 6% after traumatic hemarthrosis without any structural injuries to 50% after a combined ACL and meniscus injury, via 16% after a primary LPD. The risk of radiographic knee OA was 10 times higher in knees with a combined ACL and meniscus injury as compared to knees with only hemarthrosis. The corresponding risk ratio for the combined group with collateral ligament injuries grades 2-3, PCL injuries and osteochondral injuries was 6, and for LPD 4. Interestingly, a meniscal tear without concomitant ACL rupture did not confer any strongly increased risk as compared to the contralateral knee within the 12-year time frame.

Figure 17. Follow-up radiographs of right and left knees. ACL rupture and tears of both menisci in the right knee at baseline.

It is well known that an ACL injury is associated with increased risk of posttraumatic OA.[30, 419-421] This is also true for meniscal tears and even more so if the ACL injury is combined with a meniscal tear or a meniscectomy.[8, 112] Whether an ACL reconstruction is protective against OA development or not is debatable, with somewhat conflicting results reported.[103, 422] A Swedish study noted less radiographic tibiofemoral OA after 32-37 years in knees with early (mean 5 days) ACL reconstruction (50%) as compared to ACL-deficient knees that were treated non-operatively (75%), but still at a notably high level.[423] A matched-control study of total knee arthroplasties (TKA) in the UK have shown that previous ACL injury is associated with a sevenfold increased odds of treatment of end-stage knee OA with a TKA.[424] A recorded previous meniscal injury increased the odds 15-fold. The incidence of having a TKA has also been shown to be seven times greater among patients treated with an ACL reconstruction as compared to matched controls.[425]

A consensus group (OPTIKNEE) concluded in a 2022 review and meta-analysis of risk factors for knee OA after traumatic knee injury that very few studies considered knee injuries to other structures than the ACL and that there is a need for broader inclusion of different knee injuries.[112] A population-based longitudinal cohort study of 150 000 individuals aged 25-34 years in southern Sweden with and without different knee injuries with at least 10 years follow-up by Snoeker et al found a six times increased risk of a registered knee diagnosis after knee injury.[4] Combined cruciate ligament rupture and a meniscal tear conferred the largest risk with an adjusted hazard ratio of 9 (95% CI, 6-14), followed by cruciate ligament tear, meniscal tear and fractures of the proximal tibia or patella.

Primary as well as recurrent patellar dislocations have been associated with patellofemoral OA, although the traumatic impact tends to decrease after the first dislocation.[426, 427]. Maenpaa et al noted 22% patellofemoral OA in the affected knee 6-26 years after LPD and 11% in the unaffected knee.[428] Stable knees demonstrated more OA than unstable knees. A report from 1992 by Arnbjörnsson et al presented a small series of patients with bilateral recurrent patellofemoral instability treated surgically with stabilising measures in one knee and conservatively in the other.[11] Even though the knees had comparable short term results, at a mean of 14-years follow-up 75% of the operated knees displayed radiographic OA compared to 29% in the contralateral, conservatively treated knees. There is still no evidence that stabilising surgery would prevent future development of OA, even though MPFL reconstructions may be better than re-alignment techniques.[427, 429]

Loss of articular congruence, progressive cartilage degeneration, and early patellofemoral OA are feared when the LPD is complicated with an osteochondral fracture. Sanders et al evaluated risk factors of patellofemoral OA at a mean 12 years after 609 cases of first-time LPD and found that an osteochondral fracture, on radiographs or MRI, conferred a hazard ratio of 11 (95% CI, 5-27).[96]. This was twice as high as the increased risk with recurrent dislocations, and nearly three times as high as trochlear dysplasia.

Individuals with LPD in the follow-up cohort of this study, however, did not demonstrate any significant difference in patellofemoral OA development to the contralateral knee. Injured knees had an incidence rate of 15% (95% CI, 10-23) as compared to 8% (95% CI, 4-14) in the contralateral knee. In the matched pair analysis of the injured and contralateral knees with adjustments for previous trauma before index injury and new knee trauma during follow-up, the risk ratio of any OA in the injured knee was 1.4 (95% CI, 0.7-2.8) after LPD. Even though patellofemoral OA often is considered to be caused by chondral load due to anatomical risk factors and instability, surprisingly, LPD caused less patellofemoral OA than all other soft-tissue knee injuries in this cohort, except hemarthrosis alone.[430]

Post-traumatic radiographic and symptomatic knee osteoarthritis

Not all patients with radiographic OA are symptomatic, and individuals with very discrete findings on plain radiographs can suffer greatly from OA symptoms.[431] The incidence proportion of combined symptomatic and radiographic OA in the injured knee followed the same pattern as purely radiographic OA, depending on the index injury. A traumatic hemarthrosis without structural injury had a cumulative incidence of 4% symptomatic knees with radiographic signs of OA and an LPD had

13%. Nearly all the patients in these groups with radiographic OA were also symptomatic according to KOOS scores. This differed from the other groups (other injury, ACL injury ± meniscal tear, and meniscal tear) were about half of the patients with radiographic OA also were symptomatic. Individuals with an ACL injury without meniscal tear demonstrated symptomatic and radiographic OA in 18% of the cases after 8-18 years, and individuals with combined ACL and meniscus injuries in 29%. In the regression analysis adjusted for the logarithm of follow-up time, comparing individuals with a combined ACL and meniscus injury to individuals with hemarthrosis alone, the risk ratio of symptomatic and radiographic OA was 8. This further confirms that this injury combination is the most detrimental to the knee. However, confidence intervals in these analyses were wide due to the low numbers of OA in the reference category group of knees with hemarthrosis alone.

In their long-term follow-up of early ACL reconstruction compared to delayed or no reconstruction, Kvist et al found the same prevalence of symptomatic OA, irrespective of treatment at 32-37 years after injury despite a lower prevalence of radiographic OA in the early surgery group.[423] Identeg et al found a poor, statistically significant, correlation between PROMs (IKDC and Lysholm score) and the grade of radiographic knee OA in a follow-up study of 147 patients that had undergone ACL reconstruction.[432]

It is well known that knee injury is a major contributor to the development of knee OA with a four- to six-fold elevated risk in the general population as well as in young adults.[4, 30, 112] Osteoarthritis, with the knee being the most common site, has been ranked the 14:th most common cause of age-standardized years lived with disability in a global burden of disease study in 2021.[109] The global age-standardized prevalence of knee OA was estimated to about 4300 cases per 100 000 individuals for the year 2020, affecting one third of the population over the age of 70 and projected to involve 642 million individuals in the year 2050. The greatest potentially modifiable risk factors for incident knee OA during the lifespan are overweight/obesity and previous knee injury, which have been estimated to stand for 10% and 3% of all cases in a large review of more than 150 risk factors by Duong et al.[110] High to moderate certainty evidence was also found for an increased risk of knee OA with heavier occupational physical activity and higher bone mineral density, but at a lower level.

Other known risk factors of developing OA are participation in certain sports, namely, soccer, elite-level long-distance running, weightlifting and wrestling while recreational running may be protective.[433-435] Adjustments were made for previous injury to the injured and contralateral knee as recorded in medical charts at baseline, BMI at follow-up and self-reported new injuries to any of the knees between baseline and follow-up, however without any major impact on the results. Unfortunately, there was no available information on occupation or sporting habits in the cohort.

The impact of hemarthrosis

Of the 70 individuals in the follow-up cohort with a traumatic hemarthrosis without structural injuries as noted on sub-acute MRI, only 3% developed tibiofemoral OA in the index knee and 1% in the contra-lateral knee. The clinical routine at the study site was to aspirate the hemarthrosis in the acute phase, but that was only the case in 53% of the index injuries. Despite the hemarthrosis not being quantified, and in half of the cases evacuated, it seems like the bleeding itself did not generate a substantial incidence of OA. It is logical to assume that a traumatic hemarthrosis could be a substantial driver towards OA.[436, 437] Arthropathy is frequent with repeated joint bleeding in patients with haemophilia.[438] However, in a landmark study of prophylaxis versus episodic treatment of haemophiliacs, there was weak correlation between clinically evident hemarthroses and joint damage as seen on MRI.[439] The subject is under debate and suggestions have also been made that an acute hemarthrosis could have a protective effect, through for example enhanced meniscus healing.[440]

Wang et al identified 60 patients that had undergone an ACL reconstruction with an initial visit to the ED at the time of injury and divided them into two groups according to whether their hemarthrosis was aspirated (n=18) or not (n=42), and found that aspiration of a hemarthrosis could relieve pain, restore ROM, and improve the sensitivity of physical examination (Lachman test and pivot shift) at follow-up.[340] Their conclusion, which is easy to agree with, was that knee joint aspiration in acute traumatic hemarthrosis is at least a good diagnostic and pain relieving procedure.

Patient-reported knee-related health after soft-tissue knee injury

Contrary to the development of radiographic OA, LPD seemed to fare worse regarding patient-reported knee symptoms according to the questionnaires for KOOS and knee satisfaction. They also scored the lowest in TAS at 12 years after index injury. A recent study on KOOS values collected from a representative Danish cohort in different age groups showed higher median scores for all subscales than the subjects in our cohort.[441] Most notably, LPD patients in our cohort scored a median of 59 in the subscale for knee-related quality of life at follow-up as compared to 88 in the Danish age-matched (18-29 years) sub-group. In the regression analysis, there was an 8 point mean difference (95% CI, -17, 1) to the group of hemarthrosis alone. However, median KOOS values were practically the same across different groups and virtually none reached clinical relevance. Collins et al have reported the minimal detectable changes for each subscale in KOOS as; Pain 6-6.1, symptoms 5-8.5, ADL 7-8, sports/recreation 5.8-12, and QOL 7-7.2.[442] Tegner activity rating scale was reported to have a minimal detectable change of 1.

The LPD patients also scored worst in TAS with a mean difference to hemarthrosis alone of -1.1.

Magnusen et al reported KOOS-values on 111 non-operated patients with LPD with and without recurrent dislocation at a mean follow-up of 3.4 years.[443] They found lower scores in all subscales for the group with a single event compared to age-matched controls, and even lower in the group with recurrent events.

Eighty-one percent (95% CI 78-84) of the knee-injured patients were satisfied with their knee function at 12 years after injury, which is in line with other studies.[444] Being satisfied with the knee function at 2 years after ACL reconstruction has been correlated to higher levels of return to pre-injury sports level, knee-related self-efficacy and quality of life.[163]

The personality of the injury or the injured personality

There are many risk factors of both soft-tissue injuries and their outcomes that are known, but others are probably unknown. In this study of traumatic hemarthroses, the crude risk ratio of developing radiographic knee OA after a structural injury to the knee was 7.8 as compared to the knees with hemarthrosis alone without structural injury. When comparing the structurally injured knees to the contralateral knee in the same person, the risk ratio of radiographic knee OA was only 1.9. Possibly, the results showing that the within-person risk of post-traumatic knee OA is much lower than the between-persons risk, even when comparing with another knee-traumatised group, points towards large inter-individual differences in risk. This have, to our knowledge, not been shown before. Genetical and behavioural factors seem to carry a large part of the risk of developing post-traumatic knee OA, amplifying or attenuating the specific effect of the knee injury per se.

Strengths and limitations

The work in this thesis has certain strengths, but also its limitations. Even though based on a large consecutive cohort, it may not be fully representative of the general population. An unknown number of patients could have consulted other institutions or not sought medical care after their knee injury. Some knee injured individuals that were attended to in primary care facilities, may not have been referred to the Orthopaedic Department. These limitations could suggest a possible under-estimation of the incidence of traumatic hemarthroses, and possibly a relative over-estimation of the most serious injuries. With the organisation of public healthcare in the study region at the time, broad and well-known inclusion criteria, and the very few other options of care, the vast majority of knee injured individuals could, however, be expected to be included in the study. Needle aspiration of hemarthrosis was made in 53% of the knees. Sudden onset of joint effusion after knee trauma is, however, considered pathognomonic of hemarthrosis and it could also be regarded as a strength that not only hemarthroses confirmed by aspiration were included.[445] The similar rate of injury detection in knees with and without aspiration further strengthens the notion that the inclusion was accurate.

The catchment area of the study site may not be representative of a broader population, nationally or internationally. However, the very similar annual incidence of ACL injuries with a nationwide Swedish study as well as the latest American epidemiological study on the subject, suggests that the population would be fairly representative of these populations.[88, 341]

MR images at baseline were read as part of the normal clinical routine, but systematically and according to strict predefined protocols by a few dedicated senior musculoskeletal radiologists. No intra- or interobserver variability assessments have been made.

Some patients were scheduled for surgery before inclusion and MRI due to osteochondral fractures seen on plain radiographs, which we found in Paper II. There is a risk that the same is true for other knee injuries, e.g. some incarcerated bucket-handle meniscus tears, however surgery records have not been scrutinised for this group. Poly-trauma cases, although few, may also have been missed or diagnosed after the sub-acute period.

We did not quantify radiographic OA before index injury but hypothesised that the prevalence would be insignificant due to the young ages at inclusion. Of the 782 individuals who went through full follow-up, 57% had a plain radiograph at the time of baseline injury as part of the acute work up. Osteophytes were noted in 4% and joint space narrowing in 0.4%, in the radiologists' clinical report. Hence, there is a risk that we have under-estimated the amount of pre-injury OA. We asked the patients at follow-up if they had any additional injuries to their knees but decided to regard the initial index injury where the patient sought medical attention due to an acute knee sprain followed by hemarthrosis as the main generator of OA development and knee-related health issues.

Furthermore, we treated all meniscal lesions as the same entity even though they may differ. A horizontal meniscus tear can be a sign of degeneration of the joint whereas a vertical rupture is more probable to be caused by trauma. A root avulsion is known to result in significant instability and elevated peak pressures.[446-449] Radial tears have different effects at different sites. A complete lateral meniscus radial tear significantly raises peak contact pressures as compared to an intact meniscus at all flexion angles, especially with a root tear and in high flexion angles [450]. Even small partial lateral meniscectomies result in changes in joint motion and meniscal forces.[451, 452] However, we decided to group all meniscal injuries together and treat them as one entity.

There was a tendency for older individuals to adhere to follow-up at a greater extent and for males to a lesser extent. Also, more individuals with an ACL rupture with or without meniscus injury adhered to followed up. There is a risk that those with more symptoms at the time of follow-up were more inclined to answer than others. The follow-up procedure was staged between different sub-groups for different reasons, mainly because of other projects in the same cohort, but also for logistic reasons. This led to slightly different follow-up times between groups, which hopefully is balanced in the regression models. Especially, LPD had an average follow-up earlier than other groups. These patients were also younger which can affect the outcome in development of OA and this group may develop OA at a later stage.

Many confounding factors are un-accounted for which is demonstrated by the between-person estimates (comparing injury types to "hemarthrosis alone") that are generally larger than the matched estimates (comparing to the contralateral knee), i.e. the higher risk in persons who had injury could be due to other causes than the injury itself, e.g. genetic factors.

The specific strength of this thesis is the prospective consecutive inclusion of all soft-tissue knee injuries, adult and paediatric, with traumatic hemarthrosis in a well-defined catchment area of a single hospital. The systematic use of MRI to ascertain diagnosis, regardless of initial clinical diagnosis, is another strength, together with the relatively low loss to follow-up.

Conclusions

- Nine out of ten knees with traumatic hemarthrosis had a serious soft-tissue injury. The occurrence and distribution of injuries were age and sex dependent. Traumatic knee injuries with hemarthrosis were more prevalent in males, but the incidence proportion of significant ligament injury with traumatic hemarthrosis was higher in females.
- Every second traumatic hemarthrosis involved an ACL rupture, but most ACL injuries were in combination with other injuries. Two out of five traumatic hemarthroses in individuals under the age of 16 years were LPDs, with the majority being isolated injuries.
- Three quarters of all injuries were sports related, with 80% of the ACL and MCL injuries. Football was the most common sport (50%), followed by alpine skiing (16%). Only half of the LPDs occurred during sports.
- Traumatic hemarthrosis occurred with an incidence of 140-162 within the 95% confidence interval and ACL ruptures with an incidence of 70-85 per 100 000 person-years in the ages 10-59 years. Incidences were highest in the second decade of life for females and in the third decade for males.
- The annual incidence of a first-time patellar dislocation was 14 per 100 000 person-years with peak incidence between 13-15 years of age. Higher incidences were seen for males in all age groups except those aged 10-12 years.
- Concomitant chondral injuries occurred in nearly half of the knees with primary LPD. One in five first-time LPD had an osteochondral fracture.
- Twelve years after soft-tissue knee injury, one in three developed radiographic OA in the injured knee, and one in five symptomatic OA.
- The risk of radiographic knee OA was ten times higher and symptomatic OA eight times higher after an ACL rupture combined with a meniscus tear compared to a traumatic hemarthrosis without structural injuries.
- Soft-tissue knee injury doubled the risk of knee OA compared to the contralateral knee.
- Four out of five were satisfied with their knee function 12 years after injury.

Future perspectives and clinical implications

Soft-tissue knee injuries have the potential for substantial long-term effects depending on the injury sustained. A better understanding of the injury panorama and long-term outcomes after traumatic knee injuries could lead to a more effective management of these patients, and thus less morbidity for the individual and cost for society.

Even though much has been investigated about soft-tissue knee injuries in general and ACL ruptures in particular, there are still pieces missing in the puzzle. Hopefully, the work described in this thesis will be of help in answering some of the unanswered questions. More high-quality evidence generated by RCTs with minimal cross-over is needed to fully understand the long-term consequences of specific treatments for different injuries. However, studies in clinical cohorts like this one, can shed light on where we are today and develop hypotheses for future directions. It is hard to know where to go if you don't know where you are. However, it seems clear that further studies are needed on ways to tailor both knee injury prevention and management of an eventual knee injury to the individual.

In a cohort like this, there are several possible further questions to be asked. With MRI diagnosis after every traumatic hemarthrosis, a comparison of time to diagnosis of ACL injuries could be made with a similar hospital with a different clinical routine. Also, the natural history of meniscal tears found on baseline MRI but left in situ could possibly be described or an attempt to classify the meniscal tears and their outcome according to tear type could be made. Outcomes related to the treatment of the different ligament injuries and multi-ligament knee injuries, to the treatment of the different meniscal tears, and to the treatment of paediatric ACL and meniscus injuries could be further investigated. Moreover, bone morphology at baseline MRI could be related to injury patterns, but also outcome.

First of all though, it would be interesting to delve into the recurrence of the first-time patellar dislocations and associated risk factors.

With this, I would recommend that a subacute MRI should be gold standard for all knee injuries with hemarthrosis considering the high rate of significant findings that will affect the patients, their care, and the outcome of their injury.

Summary in Swedish

Populärvetenskaplig sammanfattning

Knäledsstukningar är vanliga och ofta idrottsrelaterade, med potential att orsaka betydande funktionsnedsättning både på kort och lång sikt. En knästukning med betydande strukturell skada leder oftast till akut svullnad på grund av blödning i knäleden, så kallad hemartros. Detta förorsakar ofta ett besök till en vårdinrättning. De flesta knästukningar är mjukdelsskador, där främre korsbandsskador är vanliga, ofta i kombination med andra strukturella skador som meniskskador, skador på sidoledband, bakre korsband eller brosk. Hos barn är urledvridning av knäskålen, patellaluxation, den vanligaste skadan.

Syftet med denna avhandling var att undersöka panoramat av skador efter traumatisk hemartros i knäleden, deras epidemiologi och långtidskonsekvenser.

Alla patienter med akut knätrauma och efterföljande svullnad som sökte akut- eller ortopedmottagningen mellan 2002 och 2008 inkluderades fortlöpande i studien och undersöktes med subakut magnetkameraundersökning (MRI) (n=1145) inom en mediantid på åtta dagar. Röntgenmässiga tecken på artros (OA) samt subjektiva knärelaterade och generella hälsoutfall undersöktes vid minst åtta och median tolv år efter skadan (intervall 8–17 år).

Vid akut posttraumatisk smärta och svullnad är den kliniska diagnosen av den akut skadade knäleden osäker, och därför är de exakta andelarna av olika skador och deras epidemiologi i befolkningen inte helt kända. I **delarbete ett** i avhandlingen beskrev vi förekomsten av och kliniska data om olika knäskador och samtidiga skademönster i kohorten, relaterade till ålder, kön och aktivitet vid skadetillfället.

Hos barn är patellaluxation den vanligaste skadan, en diagnos som ofta förbises i akutskedet. Vid patellaluxation, särskilt i ett tidigare oskadat knä, riskerar de broskbeklädda ledytorna att skadas allvarligt. Vid sådana skador rekommenderas kirurgisk åtgärd. För att belysa förekomsten av traumatiska förstagångsluxationer (primära) av patella extraherades dessa från kohorten (n= 175) och ytterligare nio patienter som gick direkt till operation under studieperioden. I avhandlingens **delarbete två** presenterades ålders- och könsspecifik förekomst av primär patellaluxation i befolkningen samt ålders-, köns- och aktivitetsdata och förekomst av samtidiga broskskador inklusive broskbeklädda frakturer.

Mjukdelsskador i knäleden kan på längre sikt leda till försämrad hälsa och smärta, där olika skador ger olika grad av besvär. Uppgifter om främre korsbandsskador är mest rapporterade sedan tidigare. En långtidsuppföljning av baslinjekohorten inkluderande röntgenbilder av båda knälederna, klinisk undersökning och patientrapporterade utfallsmått genomfördes (n=814). I **delarbete tre** undersöktes risken för att utveckla röntgenmässig och symptomgivande artros vid mediantiden 12 år efter olika traumatiska mjukdelsskador i knäleden med hemartros. Vi beskrev också självrapporterade knäbesvär, funktion och livskvalitet. Vidare relaterades fynden till individernas andra knäled.

Omkring hälften av knästukningarna med hemartros (52%) resulterade i en främre korsbandsruptur med en förekomst på 70-85 per 100 000 personår, följt av meniskskador (41%), skador på inre sidoledbandet (28%), kompressionsfrakturer (21%) och patellaluxationer (17%). Tio procent av knäledsskadorna med blod i leden uppvisade inga tecken på allvarlig strukturell skada. Idrottsaktivitet var den vanligaste orsaken till skada (72%) och fotboll den vanligaste sporten. Den högsta förekomsten av primär patellaluxation sågs i åldrarna 13–15 år, både för pojkar (135/100 000) och flickor (114/100 000), därefter sjönk förekomsten gradvis men var högre för män än kvinnor i alla åldrar. En primär patellaluxation ledde till broskskador i 43% och i 18% var detta en broskbeklädd fraktur.

En av tre hade påvisbar artros på röntgen tolv år efter skadan i den drabbade knäleden, medan en av fem hade det i andra knäleden. En av fem ansågs även ha symptomgivande artros, dvs både röntgenmässig artros och självrapporterade knäledssymptom. En främre korsbandsskada i kombination med meniskskada hade starkast koppling till röntgenmässig artros i knäleden med förekomst i 44-56% av fallen, vilket var tre gånger så stor risk jämfört med den andra knäleden. Trots detta var 80% nöjda med sin knäfunktion vid uppföljningen. En patellaluxation var starkast kopplad till försämrad självrapporterad knärelaterad hälsa. Endast 4% av individerna med blod i leden utan strukturell skada utvecklade röntgenmässiga tecken till artros. Jämfört med patienter med endast hemartros ökade risken för på röntgen påvisad artros tio gånger hos dem med främre korsbandsruptur och samtidig meniskskada, medan risken för symptomgivande artros ökade åtta gånger. Dessa fynd tyder på att, utöver själva skadan, individuella riskfaktorer spelar en betydande roll för utfallet efter mjukdelsskador i knäleden vad gäller utveckling av artros och patientrapporterade symtom.

Vid knäledsstukning med blod i leden var alltså en korsbandsruptur den vanligaste skadan. En korsbandsruptur, särskilt med samtidig meniskskada, var även starkast kopplad till både röntgenmässig och symptomgivande artros på lång sikt.

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References

1. Palmer I. On the injuries to the ligaments of the knee joint: a clinical study, [s. n.] @ 1938.
2. Fithian DC, Paxton LW, Goltz DH. Fate of the anterior cruciate ligament-injured knee. Orthop Clin North Am 2002; 33: 621-636, v.
3. Gage BE, McIlvain NM, Collins CL, Fields SK, Comstock RD. Epidemiology of 6.6 million knee injuries presenting to United States emergency departments from 1999 through 2008. Acad Emerg Med 2012; 19: 378-385.
4. Snoeker B, Turkiewicz A, Magnusson K, Frobell R, Yu D, Peat G, et al. Risk of knee osteoarthritis after different types of knee injuries in young adults: a population-based cohort study. Br J Sports Med 2020; 54: 725-730.
5. Maffulli N, Longo UG, Gougoulias N, Loppini M, Denaro V. Long-term health outcomes of youth sports injuries. Br J Sports Med 2010; 44: 21-25.
6. 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: 321-328.
7. 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: 1673-1679.
8. 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: 1756-1769.
9. Oiestad BE, Engebretsen L, Storheim K, Risberg MA. Knee osteoarthritis after anterior cruciate ligament injury: a systematic review. Am J Sports Med 2009; 37: 1434-1443.
10. Salonen EE, Magga T, Sillanpaa PJ, Kiekara T, Maenpaa H, Mattila VM. Traumatic Patellar Dislocation and Cartilage Injury: A Follow-up Study of Long-Term Cartilage Deterioration. Am J Sports Med 2017; 45: 1376-1382.
11. Arnbjornsson A, Egund N, Rydling O, Stockerup R, Ryd L. The natural history of recurrent dislocation of the patella. Long-term results of conservative and operative treatment. J Bone Joint Surg Br 1992; 74: 140-142.
12. Maenpaa H, Lehto MU. Patellar dislocation. The long-term results of nonoperative management in 100 patients. Am J Sports Med 1997; 25: 213-217.
13. Fomin S, Gauffin H, Kvist J. Short-term recovery of physical activity and knee function after an acute knee injury. BMJ Open Sport Exerc Med 2020; 6: e000950.

14. Piussi R, Berghdal T, Sundemo D, Grassi A, Zaffagnini S, Sansone M, et al. Self-Reported Symptoms of Depression and Anxiety After ACL Injury: A Systematic Review. Orthop J Sports Med 2022; 10: 23259671211066493.
15. Noyes FR, Bassett RW, Grood ES, Butler DL. Arthroscopy in acute traumatic hemarthrosis of the knee. Incidence of anterior cruciate tears and other injuries. J Bone Joint Surg Am 1980; 62: 687-695, 757.
16. Sarimo J, Rantanen J, Heikkila J, Helttula I, Hiltunen A, Orava S. Acute traumatic hemarthrosis of the knee. Is routine arthroscopic examination necessary? A study of 320 consecutive patients. Scand J Surg 2002; 91: 361-364.
17. Abbasi D, May MM, Wall EJ, Chan G, Parikh SN. MRI findings in adolescent patients with acute traumatic knee hemarthrosis. J Pediatr Orthop 2012; 32: 760-764.
18. Vahasarja V, Kinnuen P, Serlo W. Arthroscopy of the acute traumatic knee in children. Prospective study of 138 cases. Acta Orthop Scand 1993; 64: 580-582.
19. Frobell RB, Lohmander LS, Roos HP. Acute rotational trauma to the knee: poor agreement between clinical assessment and magnetic resonance imaging findings. Scand J Med Sci Sports 2007; 17: 109-114.
20. Butler JC, Andrews JR. The role of arthroscopic surgery in the evaluation of acute traumatic hemarthrosis of the knee. Clin Orthop Relat Res 1988: 150-152.
21. Odgaard F, Tuxoe J, Joergensen U, Lange B, Lausten G, Brettlau T, et al. Clinical decision making in the acutely injured knee based on repeat clinical examination and MRI. Scand J Med Sci Sports 2002; 12: 154-162.
22. Brown JS, Frobell RB, Isacsson A, Englund M, Olsson O. Agreement Between Clinical Examination and Magnetic Resonance Imaging in Acute Knee Trauma With Hemarthrosis. Clin J Sport Med 2022; 32: 401-406.
23. Crawford R, Walley G, Bridgman S, Maffulli N. Magnetic resonance imaging versus arthroscopy in the diagnosis of knee pathology, concentrating on meniscal lesions and ACL tears: a systematic review. Br Med Bull 2007; 84: 5-23.
24. DeHaven KE. Diagnosis of acute knee injuries with hemarthrosis. Am J Sports Med 1980; 8: 9-14.
25. Kirsch MD, Fitzgerald SW, Friedman H, Rogers LF. Transient lateral patellar dislocation: diagnosis with MR imaging. AJR Am J Roentgenol 1993; 161: 109-113.
26. Major NM, Beard LN, Jr., Helms CA. Accuracy of MR imaging of the knee in adolescents. AJR Am J Roentgenol 2003; 180: 17-19.
27. Patel NK, Bucknill A, Ahearne D, Denning J, Desai K, Watson M. Early magnetic resonance imaging in acute knee injury: a cost analysis. Knee Surg Sports Traumatol Arthrosc 2012; 20: 1152-1158.
28. Saragaglia D, Banihachemi JJ, Refaie R. Acute instability of the patella: is magnetic resonance imaging mandatory? Int Orthop 2020; 44: 2299-2303.
29. Kaeding CC, Leger-St-Jean B, Magnussen RA. Epidemiology and Diagnosis of Anterior Cruciate Ligament Injuries. Clin Sports Med 2017; 36: 1-8.
30. Poulsen E, Goncalves GH, Bricca A, Roos EM, Thorlund JB, Juhl CB. Knee osteoarthritis risk is increased 4-6 fold after knee injury - a systematic review and meta-analysis. Br J Sports Med 2019; 53: 1454-1463.

31. Hirschmann MT, Muller W. Complex function of the knee joint: the current understanding of the knee. Knee Surg Sports Traumatol Arthrosc 2015; 23: 2780-2788.
32. Hunziker EB, Quinn TM, Hauselmann HJ. Quantitative structural organization of normal adult human articular cartilage. Osteoarthritis Cartilage 2002; 10: 564-572.
33. Draper CE, Besier TF, Gold GE, Fredericson M, Fiene A, Beaupre GS, et al. Is cartilage thickness different in young subjects with and without patellofemoral pain? Osteoarthritis Cartilage 2006; 14: 931-937.
34. Sophia Fox AJ, Bedi A, Rodeo SA. The basic science of articular cartilage: structure, composition, and function. Sports Health 2009; 1: 461-468.
35. Walker PS, Erkman MJ. The role of the menisci in force transmission across the knee. Clin Orthop Relat Res 1975: 184-192.
36. LaPrade CM, Ellman MB, Rasmussen MT, James EW, Wijdicks CA, Engebretsen L, et al. Anatomy of the anterior root attachments of the medial and lateral menisci: a quantitative analysis. Am J Sports Med 2014; 42: 2386-2392.
37. Mameri ES, Dasari SP, Fortier LM, Verdejo FG, Gursoy S, Yanke AB, et al. Review of Meniscus Anatomy and Biomechanics. Curr Rev Musculoskelet Med 2022; 15: 323-335.
38. Pekala PA, Rosa MA, Lazarz DP, Pekala JR, Baginski A, Gobbi A, et al. Clinical Anatomy of the Anterior Meniscofemoral Ligament of Humphrey: An Original MRI Study, Meta-analysis, and Systematic Review. Orthop J Sports Med 2021; 9: 2325967120973192.
39. Pekala PA, Lazarz DP, Rosa MA, Pekala JR, Baginski A, Gobbi A, et al. Clinical Anatomy of the Posterior Meniscofemoral Ligament of Wrisberg: An Original MRI Study, Meta-analysis, and Systematic Review. Orthop J Sports Med 2021; 9: 2325967120973195.
40. Gupte CM, Shaerf DA, Sandison A, Bull AM, Amis AA. Neural Structures within Human Meniscofemoral Ligaments: A Cadaveric Study. ISRN Anat 2014; 2014: 719851.
41. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. Am J Sports Med 1982; 10: 90-95.
42. Orellana F, Grassi A, Hlushchuk R, Wahl P, Nuss KM, Neels A, et al. Revealing the complexity of meniscus microvasculature through 3D visualization and analysis. Sci Rep 2024; 14: 10875.
43. Grood ES, Suntay WJ. A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. J Biomech Eng 1983; 105: 136-144.
44. Girgis FG, Marshall JL, Monajem A. The cruciate ligaments of the knee joint. Anatomical, functional and experimental analysis. Clin Orthop Relat Res 1975: 216-231.
45. Cone SG, Howe D, Fisher MB. Size and Shape of the Human Anterior Cruciate Ligament and the Impact of Sex and Skeletal Growth: A Systematic Review. JBJS Rev 2019; 7: e8.

46. Amis AA, Gupte CM, Bull AM, Edwards A. Anatomy of the posterior cruciate ligament and the meniscofemoral ligaments. Knee Surg Sports Traumatol Arthrosc 2006; 14: 257-263.
47. LaPrade MD, Kennedy MI, Wijdicks CA, LaPrade RF. Anatomy and biomechanics of the medial side of the knee and their surgical implications. Sports Med Arthrosc Rev 2015; 23: 63-70.
48. James EW, LaPrade CM, LaPrade RF. Anatomy and biomechanics of the lateral side of the knee and surgical implications. Sports Med Arthrosc Rev 2015; 23: 2-9.
49. Segond P. Recherches cliniques et expérimentales sur les épanchements sanguins du genou par entorse, Aux bureaux du Progrès médical 1879.
50. Woods GW, Stanley RF, Tullos HS. Lateral capsular sign: x-ray clue to a significant knee instability. Am J Sports Med 1979; 7: 27-33.
51. Goldman AB, Pavlov H, Rubenstein D. The Segond fracture of the proximal tibia: a small avulsion that reflects major ligamentous damage. AJR Am J Roentgenol 1988; 151: 1163-1167.
52. Hess T, Rupp S, Hopf T, Gleitz M, Liebler J. Lateral tibial avulsion fractures and disruptions to the anterior cruciate ligament. A clinical study of their incidence and correlation. Clin Orthop Relat Res 1994: 193-197.
53. Vincent JP, Magnussen RA, Gezmez F, Uguen A, Jacobi M, Weppe F, et al. The anterolateral ligament of the human knee: an anatomic and histologic study. Knee Surg Sports Traumatol Arthrosc 2012; 20: 147-152.
54. Claes S, Vereecke E, Maes M, Victor J, Verdonk P, Bellemans J. Anatomy of the anterolateral ligament of the knee. J Anat 2013; 223: 321-328.
55. Philippot R, Boyer B, Testa R, Farizon F, Moyen B. The role of the medial ligamentous structures on patellar tracking during knee flexion. Knee Surg Sports Traumatol Arthrosc 2012; 20: 331-336.
56. Hippocrates. On the articulations. The genuine works of Hippocrates. Clin Orthop Relat Res 2002: 19-25.
57. Schindler OS. Surgery for anterior cruciate ligament deficiency: a historical perspective. Knee Surg Sports Traumatol Arthrosc 2012; 20: 5-47.
58. Snook GA. The father of sports medicine (Galen). Am J Sports Med 1978; 6: 128-131.
59. Georgoulis AD, Kiapidou IS, Velogianni L, Stergiou N, Boland A. Herodicus, the father of sports medicine. Knee Surg Sports Traumatol Arthrosc 2007; 15: 315-318.
60. D'Ambrosi R, Meena A, Arora ES, Attri M, Schafer L, Migliorini F. Reconstruction of the anterior cruciate ligament: a historical view. Ann Transl Med 2023; 11: 364.
61. Di Matteo B, Tarabella V, Filardo G, Tomba P, Vigano A, Marcacci M, et al. Knee multi-ligament reconstruction: a historical note on the fundamental landmarks. Knee Surg Sports Traumatol Arthrosc 2015; 23: 2773-2779.
62. Walden M, Krosshaug T, Bjorneboe J, Andersen TE, Faul O, Hagglund M. Three distinct mechanisms predominate in non-contact anterior cruciate ligament injuries in male professional football players: a systematic video analysis of 39 cases. Br J Sports Med 2015; 49: 1452-1460.

63. Andersen TE, Tenga A, Engebretsen L, Bahr R. Video analysis of injuries and incidents in Norwegian professional football. Br J Sports Med 2004; 38: 626-631.
64. Owusu-Akyaw KA, Kim SY, Spritzer CE, Collins AT, Englander ZA, Utturkar GM, et al. Determination of the Position of the Knee at the Time of an Anterior Cruciate Ligament Rupture for Male Versus Female Patients by an Analysis of Bone Bruises. Am J Sports Med 2018; 46: 1559-1565.
65. Nikku R, Nietosvaara Y, Aalto K, Kallio PE. The mechanism of primary patellar dislocation: trauma history of 126 patients. Acta Orthop 2009; 80: 432-434.
66. Hawkins RD, Fuller CW. Risk assessment in professional football: an examination of accidents and incidents in the 1994 World Cup finals. Br J Sports Med 1996; 30: 165-170.
67. Yawn BP, Amadio P, Harmsen WS, Hill J, Ilstrup D, Gabriel S. Isolated acute knee injuries in the general population. J Trauma 2000; 48: 716-723.
68. Nielsen AB, Yde J. Epidemiology of acute knee injuries: a prospective hospital investigation. J Trauma 1991; 31: 1644-1648.
69. Ferry T, Bergstrom U, Hedstrom EM, Lorentzon R, Zeisig E. Epidemiology of acute knee injuries seen at the Emergency Department at Umea University Hospital, Sweden, during 15 years. Knee Surg Sports Traumatol Arthrosc 2014; 22: 1149-1155.
70. Maniar N, Verhagen E, Bryant AL, Opar DA. Trends in Australian knee injury rates: An epidemiological analysis of 228,344 knee injuries over 20 years. Lancet Reg Health West Pac 2022; 21: 100409.
71. Peat G, Bergknut C, Frobell R, Joud A, Englund M. Population-wide incidence estimates for soft tissue knee injuries presenting to healthcare in southern Sweden: data from the Skane Healthcare Register. Arthritis Res Ther 2014; 16: R162.
72. Casteleyn PP, Handelberg F, Opdecam P. Traumatic haemarthrosis of the knee. J Bone Joint Surg Br 1988; 70: 404-406.
73. Gillquist J, Hagberg G, Oretorp N. Arthroscopy in acute injuries of the knee joint. Acta Orthop Scand 1977; 48: 190-196.
74. Palmieri-Smith RM, Villwock M, Downie B, Hecht G, Zernicke R. Pain and effusion and quadriceps activation and strength. J Athl Train 2013; 48: 186-191.
75. Chinzei N, Brophy RH, Duan X, Cai L, Nunley RM, Sandell LJ, et al. Molecular influence of anterior cruciate ligament tear remnants on chondrocytes: a biologic connection between injury and osteoarthritis. Osteoarthritis Cartilage 2018; 26: 588-599.
76. Friel NA, Chu CR. The role of ACL injury in the development of posttraumatic knee osteoarthritis. Clin Sports Med 2013; 32: 1-12.
77. Kingery MT, Anil U, Berlinberg EJ, Clair AJ, Kenny L, Strauss EJ. Changes in the Synovial Fluid Cytokine Profile of the Knee Between an Acute Anterior Cruciate Ligament Injury and Surgical Reconstruction. Am J Sports Med 2022; 50: 451-460.
78. Inclan PM, Rai MF, Chen L, Brophy RH. Aspiration of the Knee Prior to Anterior Cruciate Ligament Reconstruction Delays the Onset of Arthrofibrosis. J Am Acad Orthop Surg 2025.

79. Fithian DC, Paxton EW, Cohen AB. Indications in the treatment of patellar instability. J Knee Surg 2004; 17: 47-56.
80. Nietosvaara Y, Aalto K, Kallio PE. Acute patellar dislocation in children: incidence and associated osteochondral fractures. J Pediatr Orthop 1994; 14: 513-515.
81. Atkin DM, Fithian DC, Marangi KS, Stone ML, Dobson BE, Mendelsohn C. Characteristics of patients with primary acute lateral patellar dislocation and their recovery within the first 6 months of injury. Am J Sports Med 2000; 28: 472-479.
82. Sillanpaa P, Mattila VM, Iivonen T, Visuri T, Pihlajamaki H. Incidence and risk factors of acute traumatic primary patellar dislocation. Med Sci Sports Exerc 2008; 40: 606-611.
83. Hsiao M, Owens BD, Burks R, Sturdivant RX, Cameron KL. Incidence of acute traumatic patellar dislocation among active-duty United States military service members. Am J Sports Med 2010; 38: 1997-2004.
84. Waterman BR, Belmont PJ, Jr., Owens BD. Patellar dislocation in the United States: role of sex, age, race, and athletic participation. J Knee Surg 2012; 25: 51-57.
85. Askenberger M, Ekstrom W, Finnbogason T, Janarv PM. Occult Intra-articular Knee Injuries in Children With Hemarthrosis. Am J Sports Med 2014; 42: 1600-1606.
86. Gravesen KS, Kallemose T, Blond L, Troelsen A, Barfod KW. High incidence of acute and recurrent patellar dislocations: a retrospective nationwide epidemiological study involving 24.154 primary dislocations. Knee Surg Sports Traumatol Arthrosc 2017.
87. Sanders TL, Pareek A, Hewett TE, Stuart MJ, Dahm DL, Krych AJ. Incidence of First-Time Lateral Patellar Dislocation: A 21-Year Population-Based Study. Sports Health 2018; 10: 146-151.
88. Nordenvall R, Bahmanyar S, Adami J, Stenros C, Wredmark T, Fellander-Tsai L. A population-based nationwide study of cruciate ligament injury in Sweden, 2001-2009: incidence, treatment, and sex differences. Am J Sports Med 2012; 40: 1808-1813.
89. Sanders TL, Maradit Kremers H, Bryan AJ, Larson DR, Dahm DL, Levy BA, et al. Incidence of Anterior Cruciate Ligament Tears and Reconstruction: A 21-Year Population-Based Study. Am J Sports Med 2016; 44: 1502-1507.
90. Shea KG, Carey JL. Management of anterior cruciate ligament injuries: evidence-based guideline. J Am Acad Orthop Surg 2015; 23: e1-5.
91. Behairy NH, Dorgham MA, Khaled SA. Accuracy of routine magnetic resonance imaging in meniscal and ligamentous injuries of the knee: comparison with arthroscopy. Int Orthop 2009; 33: 961-967.
92. Qiu RY, Fitzpatrick DWD, Cohen D, Kay J, Almasri M, de Sa DL. MRI as the optimal imaging modality for assessment and management of osteochondral fractures and loose bodies following traumatic patellar dislocation: a systematic review. Knee Surg Sports Traumatol Arthrosc 2023; 31: 1744-1752.
93. Filbay SR, Ackerman IN, Russell TG, Crossley KM. Return to sport matters-longer-term quality of life after ACL reconstruction in people with knee difficulties. Scand J Med Sci Sports 2017; 27: 514-524.

94. O'Sullivan O, Ladlow P, Steiner K, Kuyser D, Ali O, Stocks J, et al. Knee MRI biomarkers associated with structural, functional and symptomatic changes at least a year from ACL injury - A systematic review. Osteoarthr Cartil Open 2023; 5: 100385.
95. Filbay SR, Roemer FW, Lohmander LS, Turkiewicz A, Roos EM, Frobell R, et al. Evidence of ACL healing on MRI following ACL rupture treated with rehabilitation alone may be associated with better patient-reported outcomes: a secondary analysis from the KANON trial. Br J Sports Med 2023; 57: 91-98.
96. Sanders TL, Pareek A, Johnson NR, Stuart MJ, Dahm DL, Krych AJ. Patellofemoral Arthritis After Lateral Patellar Dislocation: A Matched Population-Based Analysis. Am J Sports Med 2017; 45: 1012-1017.
97. 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: 261-267.
98. Neuman P, Englund M, Kostogiannis I, Friden T, Roos H, Dahlberg LE. Prevalence of tibiofemoral osteoarthritis 15 years after nonoperative treatment of anterior cruciate ligament injury: a prospective cohort study. Am J Sports Med 2008; 36: 1717-1725.
99. Nelson F, Billinghurst RC, Pidoux I, Reiner A, Langworthy M, McDermott M, et al. Early post-traumatic osteoarthritis-like changes in human articular cartilage following rupture of the anterior cruciate ligament. Osteoarthritis Cartilage 2006; 14: 114-119.
100. Church S, Keating JF. Reconstruction of the anterior cruciate ligament: timing of surgery and the incidence of meniscal tears and degenerative change. J Bone Joint Surg Br 2005; 87: 1639-1642.
101. Ohly NE, Murray IR, Keating JF. Revision anterior cruciate ligament reconstruction: timing of surgery and the incidence of meniscal tears and degenerative change. J Bone Joint Surg Br 2007; 89: 1051-1054.
102. Kennedy J, Jackson MP, O'Kelly P, Moran R. Timing of reconstruction of the anterior cruciate ligament in athletes and the incidence of secondary pathology within the knee. J Bone Joint Surg Br 2010; 92: 362-366.
103. Nordenvall R, Bahmanyar S, Adami J, Mattila VM, Fellander-Tsai L. Cruciate ligament reconstruction and risk of knee osteoarthritis: the association between cruciate ligament injury and post-traumatic osteoarthritis. a population based nationwide study in Sweden, 1987-2009. PLoS One 2014; 9: e104681.
104. Lohmander LS, Roemer FW, Frobell RB, Roos EM. Treatment for Acute Anterior Cruciate Ligament Tear in Young Active Adults. NEJM Evid 2023; 2: EVIDoa2200287.
105. Li H, Chen C, Chen S. Posttraumatic knee osteoarthritis following anterior cruciate ligament injury: Potential biochemical mediators of degenerative alteration and specific biochemical markers. Biomed Rep 2015; 3: 147-151.
106. Braunstein EM, White SJ, Russell W, Harris JE. Paleoradiologic evaluation of the Egyptian royal mummies. Skeletal Radiol 1988; 17: 348-352.

107. Rothschild BM, Xiaoting Z, Martin LD. Osteoarthritis in the early avian radiation: Earliest recognition of the disease in birds. Cretaceous Research 2012; 35: 178-180.
108. 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: 1286-1293.
109. Collaborators GBDO. Global, regional, and national burden of osteoarthritis, 1990-2020 and projections to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Rheumatol 2023; 5: e508-e522.
110. Duong V, Abdel Shaheed C, Ferreira ML, Narayan SW, Venkatesha V, Hunter DJ, et al. Risk factors for the development of knee osteoarthritis across the lifespan: A systematic review and meta-analysis. Osteoarthritis Cartilage 2025.
111. 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: 219-222.
112. Whittaker JL, Losciale JM, Juhl CB, Thorlund JB, Lundberg M, Truong LK, et al. Risk factors for knee osteoarthritis after traumatic knee injury: a systematic review and meta-analysis of randomised controlled trials and cohort studies for the OPTIKNEE Consensus. Br J Sports Med 2022; 56: 1406-1421.
113. Osmani HT, Nicolaou N, Anand S, Gower J, Steering G, Metcalfe A, et al. Future research priorities for soft-tissue knee injuries. Bone Joint J 2024; 106-B: 232-239.
114. Frobell RB, Lohmander LS, Roos EM. The challenge of recruiting patients with anterior cruciate ligament injury of the knee into a randomized clinical trial comparing surgical and non-surgical treatment. Contemp Clin Trials 2007; 28: 295-302.
115. Frobell RB, Roos EM, Roos HP, Ranstam J, Lohmander LS. A randomized trial of treatment for acute anterior cruciate ligament tears. N Engl J Med 2010; 363: 331-342.
116. Kastelein M, Luijsterburg PA, Wagemakers HP, Bansraj SC, Berger MY, Koes BW, et al. Diagnostic value of history taking and physical examination to assess effusion of the knee in traumatic knee patients in general practice. Arch Phys Med Rehabil 2009; 90: 82-86.
117. Fetto JF, Marshall JL. Medial collateral ligament injuries of the knee: a rationale for treatment. Clin Orthop Relat Res 1978: 206-218.
118. Rossi R, Dettoni F, Bruzzone M, Cottino U, D'Elicio DG, Bonasia DE. Clinical examination of the knee: know your tools for diagnosis of knee injuries. Sports Med Arthrosc Rehabil Ther Technol 2011; 3: 25.
119. Hefti F, Muller W, Jakob RP, Staubli HU. Evaluation of knee ligament injuries with the IKDC form. Knee Surg Sports Traumatol Arthrosc 1993; 1: 226-234.
120. Rubinstein RA, Jr., Shelbourne KD, McCarroll JR, VanMeter CD, Rettig AC. The accuracy of the clinical examination in the setting of posterior cruciate ligament injuries. Am J Sports Med 1994; 22: 550-557.
121. Johnson LL, van Dyk GE, Green JR, 3rd, Pittsley AW, Bays B, Gully SM, et al. Clinical assessment of asymptomatic knees: comparison of men and women. Arthroscopy 1998; 14: 347-359.

122. Sheehan FT, Derasari A, Fine KM, Brindle TJ, Alter KE. Q-angle and J-sign: indicative of maltracking subgroups in patellofemoral pain. Clin Orthop Relat Res 2010; 468: 266-275.
123. Post WR. Clinical evaluation of patients with patellofemoral disorders. Arthroscopy 1999; 15: 841-851.
124. Nissen CW, Cullen MC, Hewett TE, Noyes FR. Physical and arthroscopic examination techniques of the patellofemoral joint. J Orthop Sports Phys Ther 1998; 28: 277-285.
125. Kolowich PA, Paulos LE, Rosenberg TD, Farnsworth S. Lateral release of the patella: indications and contraindications. Am J Sports Med 1990; 18: 359-365.
126. Aglietti P, Insall JN, Cerulli G. Patellar pain and incongruence. I: Measurements of incongruence. Clin Orthop Relat Res 1983: 217-224.
127. Nomura E, Inoue M, Kobayashi S. Generalized joint laxity and contralateral patellar hypermobility in unilateral recurrent patellar dislocators. Arthroscopy 2006; 22: 861-865.
128. Hughston JC. Subluxation of the patella. J Bone Joint Surg Am 1968; 50: 1003-1026.
129. Ahmad CS, McCarthy M, Gomez JA, Shubin Stein BE. The moving patellar apprehension test for lateral patellar instability. Am J Sports Med 2009; 37: 791-796.
130. Sanchis-Alfonso V, Rosello-Sastre E. Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuroanatomic basis for anterior knee pain in the active young patient. Am J Sports Med 2000; 28: 725-731.
131. Fulkerson JP. Evaluation of the peripatellar soft tissues and retinaculum in patients with patellofemoral pain. Clin Sports Med 1989; 8: 197-202.
132. Korkala OL, Isotalo TM, Lavonius MI, Niskanen RO. Outcome and clinical signs of arthroscopically graded patellar chondromalacia with or without lateral release. Ann Chir Gynaecol 1995; 84: 276-279.
133. Niskanen RO, Paavilainen PJ, Jaakkola M, Korkala OL. Poor correlation of clinical signs with patellar cartilaginous changes. Arthroscopy 2001; 17: 307-310.
134. Hand CJ, Spalding TJ. Association between anatomical features and anterior knee pain in a "fit" service population. J R Nav Med Serv 2004; 90: 125-134.
135. Beighton P, Horan F. Orthopaedic aspects of the Ehlers-Danlos syndrome. J Bone Joint Surg Br 1969; 51: 444-453.
136. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. Ann Rheum Dis 1973; 32: 413-418.
137. Khanna AJ, Cosgarea AJ, Mont MA, Andres BM, Domb BG, Evans PJ, et al. Magnetic resonance imaging of the knee. Current techniques and spectrum of disease. J Bone Joint Surg Am 2001; 83-A Suppl 2 Pt 2: 128-141.
138. Roemer FW, Lynch JA, Niu J, Zhang Y, Crema MD, Tolstykh I, et al. A comparison of dedicated 1.0 T extremity MRI vs large-bore 1.5 T MRI for semiquantitative whole organ assessment of osteoarthritis: the MOST study. Osteoarthritis Cartilage 2010; 18: 168-174.

139. Nouri N, Bouaziz MC, Riahi H, Mechri M, Kherfani A, Ouertatani M, et al. Traumatic Meniscus and Cruciate Ligament Tears in Young Patients: A Comparison of 3T Versus 1.5T MRI. J Belg Soc Radiol 2017; 101: 14.
140. Grossman JW, De Smet AA, Shinki K. Comparison of the accuracy rates of 3-T and 1.5-T MRI of the knee in the diagnosis of meniscal tear. AJR Am J Roentgenol 2009; 193: 509-514.
141. Van Dyck P, Vanhoenacker FM, Lambrecht V, Wouters K, Gielen JL, Dossche L, et al. Prospective comparison of 1.5 and 3.0-T MRI for evaluating the knee menisci and ACL. J Bone Joint Surg Am 2013; 95: 916-924.
142. Wong S, Steinbach L, Zhao J, Stehling C, Ma CB, Link TM. Comparative study of imaging at 3.0 T versus 1.5 T of the knee. Skeletal Radiol 2009; 38: 761-769.
143. Kijowski R, Blankenbaker DG, Davis KW, Shinki K, Kaplan LD, De Smet AA. Comparison of 1.5- and 3.0-T MR imaging for evaluating the articular cartilage of the knee joint. Radiology 2009; 250: 839-848.
144. Van Dyck P, Kenis C, Vanhoenacker FM, Lambrecht V, Wouters K, Gielen JL, et al. Comparison of 1.5- and 3-T MR imaging for evaluating the articular cartilage of the knee. Knee Surg Sports Traumatol Arthrosc 2014; 22: 1376-1384.
145. Cheng Q, Zhao FC. Comparison of 1.5- and 3.0-T magnetic resonance imaging for evaluating lesions of the knee: A systematic review and meta-analysis (PRISMA-compliant article). Medicine (Baltimore) 2018; 97: e12401.
146. 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: 2178-2187.
147. Frobell RB, Roos HP, Roos EM, Hellio Le Graverand MP, Buck R, Tamez-Pena J, 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: 829-836.
148. Elias DA, White LM, Fithian DC. Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. Radiology 2002; 225: 736-743.
149. Lance E, Deutsch AL, Mink JH. Prior lateral patellar dislocation: MR imaging findings. Radiology 1993; 189: 905-907.
150. Quinn SF, Brown TR, Demlow TA. MR imaging of patellar retinacular ligament injuries. J Magn Reson Imaging 1993; 3: 843-847.
151. Virolainen H, Visuri T, Kuusela T. Acute dislocation of the patella: MR findings. Radiology 1993; 189: 243-246.
152. Paakkala A, Sillanpaa P, Huhtala H, Paakkala T, Maenpaa H. Bone bruise in acute traumatic patellar dislocation: volumetric magnetic resonance imaging analysis with follow-up mean of 12 months. Skeletal Radiol 2010; 39: 675-682.
153. Boegard TL, Rudling O, Petersson IF, Jonsson K. Joint space width of the tibiofemoral and of the patellofemoral joint in chronic knee pain with or without radiographic osteoarthritis: a 2-year follow-up. Osteoarthritis Cartilage 2003; 11: 370-376.

154. Altman RD, Gold GE. Atlas of individual radiographic features in osteoarthritis, revised. Osteoarthritis Cartilage 2007; 15 Suppl A: A1-56.
155. Altman RD, Hochberg M, Murphy WA, Jr., Wolfe F, Lequesne M. Atlas of individual radiographic features in osteoarthritis. Osteoarthritis Cartilage 1995; 3 Suppl A: 3-70.
156. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis 1957; 16: 494-502.
157. Roos EM, Roos HP, Ekdahl C, Lohmander LS. Knee injury and Osteoarthritis Outcome Score (KOOS)--validation of a Swedish version. Scand J Med Sci Sports 1998; 8: 439-448.
158. Roos EM, Roos HP, Lohmander LS, Ekdahl C, Beynnon BD. Knee Injury and Osteoarthritis Outcome Score (KOOS)--development of a self-administered outcome measure. J Orthop Sports Phys Ther 1998; 28: 88-96.
159. Roos EM, Lohmander LS. The Knee injury and Osteoarthritis Outcome Score (KOOS): from joint injury to osteoarthritis. Health Qual Life Outcomes 2003; 1: 64.
160. Tegner Y, Lysholm J. Rating systems in the evaluation of knee ligament injuries. Clin Orthop Relat Res 1985: 43-49.
161. Cherkin DC, Deyo RA, Street JH, Barlow W. Predicting poor outcomes for back pain seen in primary care using patients' own criteria. Spine (Phila Pa 1976) 1996; 21: 2900-2907.
162. Faltstrom A, Hagglund M, Kvist J. Patient-reported knee function, quality of life, and activity level after bilateral anterior cruciate ligament injuries. Am J Sports Med 2013; 41: 2805-2813.
163. Ardern CL, Osterberg A, Sonesson S, Gauffin H, Webster KE, Kvist J. Satisfaction With Knee Function After Primary Anterior Cruciate Ligament Reconstruction Is Associated With Self-Efficacy, Quality of Life, and Returning to the Preinjury Physical Activity. Arthroscopy 2016; 32: 1631-1638 e1633.
164. Mariani PP, Puddu G, Ferretti A. Hemathrosis treated by aspiration and casting. How to condemn the knee. Am J Sports Med 1982; 10: 343-345.
165. Noyes FR, Paulos L, Mooar LA, Signer B. Knee sprains and acute knee hemarthrosis: misdiagnosis of anterior cruciate ligament tears. Phys Ther 1980; 60: 1596-1601.
166. Hardaker WT, Jr., Garrett WE, Jr., Bassett FH, 3rd. Evaluation of acute traumatic hemarthrosis of the knee joint. South Med J 1990; 83: 640-644.
167. von Engelhardt LV, Raddatz M, Bouillon B, Spahn G, David A, Haage P, et al. How reliable is MRI in diagnosing cartilaginous lesions in patients with first and recurrent lateral patellar dislocations? BMC Musculoskelet Disord 2010; 11: 149.
168. Seeley M, Bowman KF, Walsh C, Sabb BJ, Vanderhave KL. Magnetic resonance imaging of acute patellar dislocation in children: patterns of injury and risk factors for recurrence. J Pediatr Orthop 2012; 32: 145-155.
169. Munshi M, Davidson M, MacDonald PB, Froese W, Sutherland K. The efficacy of magnetic resonance imaging in acute knee injuries. Clin J Sport Med 2000; 10: 34-39.

170. Stannard JP, Lopez R, Volgas D. Soft tissue injury of the knee after tibial plateau fractures. J Knee Surg 2010; 23: 187-192.
171. Stiell IG, Greenberg GH, Wells GA, McKnight RD, Cwinn AA, Cacciotti T, et al. Derivation of a decision rule for the use of radiography in acute knee injuries. Ann Emerg Med 1995; 26: 405-413.
172. Stiell IG, Wells GA, Hoag RH, Sivilotti ML, Cacciotti TF, Verbeek PR, et al. Implementation of the Ottawa Knee Rule for the use of radiography in acute knee injuries. JAMA 1997; 278: 2075-2079.
173. Kazemi SM, Khorram R, Fayyazishishavan E, Amani-Beni R, Haririan Y, Hosseini Khameneh SM, et al. Diagnostic Accuracy of Ottawa Knee Rule for Diagnosis of Fracture in Patients with Knee Trauma; a Systematic Review and Meta-analysis. Arch Acad Emerg Med 2023; 11: e30.
174. Sims JI, Chau MT, Davies JR. Diagnostic accuracy of the Ottawa Knee Rule in adult acute knee injuries: a systematic review and meta-analysis. Eur Radiol 2020; 30: 4438-4446.
175. Bergh C, Wennergren D, Moller M, Brisby H. Fracture incidence in adults in relation to age and gender: A study of 27,169 fractures in the Swedish Fracture Register in a well-defined catchment area. PLoS One 2020; 15: e0244291.
176. Molloy T, Gompels B, McDonnell S. Assessing diagnostic challenges in acute soft-tissue knee injuries. Bone Jt Open 2024; 5: 984-991.
177. Molloy T, Gompels B, Castagno S, McDonnell S. The Cambridge Knee Injury Tool (CamKIT): a clinical prediction tool for acute soft tissue knee injuries. BMJ Open Sport Exerc Med 2025; 11: e002357.
178. Englund M, Guermazi A, Gale D, Hunter DJ, Aliabadi P, Clancy M, et al. Incidental meniscal findings on knee MRI in middle-aged and elderly persons. N Engl J Med 2008; 359: 1108-1115.
179. Weitz FK, Sillanpaa PJ, Mattila VM. The incidence of paediatric ACL injury is increasing in Finland. Knee Surg Sports Traumatol Arthrosc 2020; 28: 363-368.
180. Beck NA, Lawrence JTR, Nordin JD, DeFor TA, Tompkins M. ACL Tears in School-Aged Children and Adolescents Over 20 Years. Pediatrics 2017; 139.
181. Miro EW, Taylor E, Curtin A, Newman MG, Ose D, Knox J. Cumulative Incidence of All-Cause Knee Injury, Concussion, and Stress Fracture among Transgender Patients on Gender-Affirming Hormone Therapy: An Exploratory Retrospective Cohort Study. Int J Environ Res Public Health 2023; 20.
182. Prieto-Gonzalez P, Martinez-Castillo JL, Fernandez-Galvan LM, Casado A, Soporki S, Sanchez-Infante J. Epidemiology of Sports-Related Injuries and Associated Risk Factors in Adolescent Athletes: An Injury Surveillance. Int J Environ Res Public Health 2021; 18.
183. Moses B, Orchard J, Orchard J. Systematic review: Annual incidence of ACL injury and surgery in various populations. Res Sports Med 2012; 20: 157-179.
184. 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. Scandinavian Journal of Medicine & Science in Sports 2005; 15: 118-125.

185. Pressman A, Johnson DH. A review of ski injuries resulting in combined injury to the anterior cruciate ligament and medial collateral ligaments. Arthroscopy 2003; 19: 194-202.
186. Friden T, Erlandsson T, Zatterstrom R, Lindstrand A, Moritz U. Compression or distraction of the anterior cruciate injured knee. Variations in injury pattern in contact sports and downhill skiing. Knee Surg Sports Traumatol Arthrosc 1995; 3: 144-147.
187. Posch M, Schranz A, Lener M, Tecklenburg K, Burtscher M, Ruedl G. In recreational alpine skiing, the ACL is predominantly injured in all knee injuries needing hospitalisation. Knee Surg Sports Traumatol Arthrosc 2021; 29: 1790-1796.
188. Shi H, Jiang Y, Ren S, Hu X, Huang H, Ao Y. Sex differences in the knee orthopaedic injury patterns among recreational alpine skiers. BMC Sports Sci Med Rehabil 2020; 12: 74.
189. Mancino F, Gabr A, Plastow R, Haddad FS. Anterior cruciate ligament injuries in female athletes. Bone Joint J 2023; 105-B: 1033-1037.
190. Mancino F, Kayani B, Gabr A, Fontalis A, Plastow R, Haddad FS. Anterior cruciate ligament injuries in female athletes: risk factors and strategies for prevention. Bone Jt Open 2024; 5: 94-100.
191. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. Arthroscopy 2007; 23: 1320-1325 e1326.
192. Stovitz SD, Shrier I. Determination of athlete-exposures in team sports: it depends on the research question. Br J Sports Med 2025.
193. Danielsen AC, Gompers A, Bekker S, Richardson SS. Limitations of athlete-exposures as a construct for comparisons of injury rates by gender/sex: a narrative review. Br J Sports Med 2025; 59: 177-184.
194. Walden M, Hagglund M, Werner J, Ekstrand J. The epidemiology of anterior cruciate ligament injury in football (soccer): a review of the literature from a gender-related perspective. Knee Surg Sports Traumatol Arthrosc 2011; 19: 3-10.
195. Walden M, Hagglund M, Magnusson H, Ekstrand J. Anterior cruciate ligament injury in elite football: a prospective three-cohort study. Knee Surg Sports Traumatol Arthrosc 2011; 19: 11-19.
196. Parkkari J, Pasanen K, Mattila VM, Kannus P, Rimpela A. The risk for a cruciate ligament injury of the knee in adolescents and young adults: a population-based cohort study of 46 500 people with a 9 year follow-up. Br J Sports Med 2008; 42: 422-426.
197. Montalvo AM, Schneider DK, Yut L, Webster KE, Beynnon B, Kocher MS, et al. "What's my risk of sustaining an ACL injury while playing sports?" A systematic review with meta-analysis. Br J Sports Med 2019; 53: 1003-1012.
198. Bram JT, Magee LC, Mehta NN, Patel NM, Ganley TJ. Anterior Cruciate Ligament Injury Incidence in Adolescent Athletes: A Systematic Review and Meta-analysis. Am J Sports Med 2021; 49: 1962-1972.

199. Childers J, Eng E, Lack B, Lin S, Knapik DM, Kaplan DJ, et al. Reported Anterior Cruciate Ligament Injury Incidence in Adolescent Athletes Is Greatest in Female Soccer Players and Athletes Participating in Club Sports: A Systematic Review and Meta-analysis. Arthroscopy 2025; 41: 774-784 e772.
200. Ventaja-Cruz J, Cuevas Rincon JM, Tejada-Medina V, Martin-Moya R. A Bibliometric Study on the Evolution of Women's Football and Determinants Behind Its Growth over the Last 30 Years. Sports (Basel) 2024; 12.
201. Hallen A, Tomas R, Ekstrand J, Bengtsson H, Van den Steen E, Hagglund M, et al. UEFA Women's Elite Club Injury Study: a prospective study on 1527 injuries over four consecutive seasons 2018/2019 to 2021/2022 reveals thigh muscle injuries to be most common and ACL injuries most burdensome. Br J Sports Med 2024; 58: 128-135.
202. Montalvo AM, Schneider DK, Silva PL, Yut L, Webster KE, Riley MA, et al. 'What's my risk of sustaining an ACL injury while playing football (soccer)?' A systematic review with meta-analysis. Br J Sports Med 2019; 53: 1333-1340.
203. Szymski D, Achenbach L, Weber J, Huber L, Memmel C, Kerschbaum M, et al. Reduced performance after return to competition in ACL injuries: an analysis on return to competition in the 'ACL registry in German Football'. Knee Surg Sports Traumatol Arthrosc 2023; 31: 133-141.
204. Arundale AJH, Silvers-Granelli HJ, Snyder-Mackler L. Career Length and Injury Incidence After Anterior Cruciate Ligament Reconstruction in Major League Soccer Players. Orthop J Sports Med 2018; 6: 2325967117750825.
205. Walden M, Hagglund M, Magnusson H, Ekstrand J. ACL injuries in men's professional football: a 15-year prospective study on time trends and return-to-play rates reveals only 65% of players still play at the top level 3 years after ACL rupture. Br J Sports Med 2016; 50: 744-750.
206. Montalvo AM, Schneider DK, Webster KE, Yut L, Galloway MT, Heidt RS, Jr., et al. Anterior Cruciate Ligament Injury Risk in Sport: A Systematic Review and Meta-Analysis of Injury Incidence by Sex and Sport Classification. J Athl Train 2019; 54: 472-482.
207. Ardern CL, Taylor NF, Feller JA, Webster KE. Fifty-five per cent return to competitive sport following anterior cruciate ligament reconstruction surgery: an updated systematic review and meta-analysis including aspects of physical functioning and contextual factors. Br J Sports Med 2014; 48: 1543-1552.
208. Saber B, Bridger D, Agrawal DK. A Critical Analysis of the Factors Contributing to Anterior Cruciate Ligament Injuries in Female Athletes. J Orthop Sports Med 2024; 6: 203-209.
209. Apseloff NA, Hughes JD, Devitt BM, Musahl V. Primary Anterior Cruciate Ligament Injury: Extrinsic and Intrinsic Risk Factors. J Am Acad Orthop Surg 2024.
210. Magnusson K, Turkiewicz A, Snoeker B, Hughes V, Englund M. The heritability of doctor-diagnosed traumatic and degenerative meniscus tears. Osteoarthritis Cartilage 2021; 29: 979-985.

211. Magnusson K, Turkiewicz A, Hughes V, Frobell R, Englund M. High genetic contribution to anterior cruciate ligament rupture: Heritability ~69. Br J Sports Med 2020.
212. Bram JT, Pascual-Leone N, Patel NM, DeFrancesco CJ, Talathi NS, Ganley TJ. Do Pediatric Patients With Anterior Cruciate Ligament Tears Have a Higher Rate of Familial Anterior Cruciate Ligament Injury? Orthop J Sports Med 2020; 8: 2325967120959665.
213. Liu SH, Al-Shaikh RA, Panossian V, Finerman GA, Lane JM. Estrogen affects the cellular metabolism of the anterior cruciate ligament. A potential explanation for female athletic injury. Am J Sports Med 1997; 25: 704-709.
214. Dos'Santos T, Stebbings GK, Morse C, Shashidharan M, Daniels KAJ, Sanderson A. Effects of the menstrual cycle phase on anterior cruciate ligament neuromuscular and biomechanical injury risk surrogates in eumenorrheic and naturally menstruating women: A systematic review. PLoS One 2023; 18: e0280800.
215. Somerson JS, Isby IJ, Hagen MS, Kweon CY, Gee AO. The Menstrual Cycle May Affect Anterior Knee Laxity and the Rate of Anterior Cruciate Ligament Rupture: A Systematic Review and Meta-Analysis. JBJS Rev 2019; 7: e2.
216. Fry SA, Hirpara A, Whitney KE, Keeter CL, Constantine EP, Williams KG, et al. Use of Hormonal Contraceptives Is Associated With Decreased Incidence of Anterior Cruciate Ligament Injuries Requiring Reconstruction in Female Patients. Arthroscopy 2025.
217. Brandon ML, Haynes PT, Bonamo JR, Flynn MI, Barrett GR, Sherman MF. The association between posterior-inferior tibial slope and anterior cruciate ligament insufficiency. Arthroscopy 2006; 22: 894-899.
218. Dejour H, Bonnin M. Tibial translation after anterior cruciate ligament rupture. Two radiological tests compared. J Bone Joint Surg Br 1994; 76: 745-749.
219. Kostogiannis I, Sward P, Neuman P, Friden T, Roos H. The influence of posterior-inferior tibial slope in ACL injury. Knee Surg Sports Traumatol Arthrosc 2011; 19: 592-597.
220. Rozinthe A, van Rooij F, Demey G, Saffarini M, Dejour D. Tibial slope correction combined with second revision ACLR grants good clinical outcomes and prevents graft rupture at 7-15-year follow-up. Knee Surg Sports Traumatol Arthrosc 2022; 30: 2336-2341.
221. Rahnemai-Azar AA, Yaseen Z, van Eck CF, Irrgang JJ, Fu FH, Musahl V. Increased Lateral Tibial Plateau Slope Predisposes Male College Football Players to Anterior Cruciate Ligament Injury. J Bone Joint Surg Am 2016; 98: 1001-1006.
222. Misir A, Sayer G, Uzun E, Guney B, Guney A. Individual and Combined Anatomic Risk Factors for the Development of an Anterior Cruciate Ligament Rupture in Men: A Multiple Factor Analysis Case-Control Study. Am J Sports Med 2022; 50: 433-440.
223. Anderson AF, Lipscomb AB, Liudahl KJ, Addlestone RB. Analysis of the intercondylar notch by computed tomography. Am J Sports Med 1987; 15: 547-552.

224. LaPrade RF, Burnett QM, 2nd. Femoral intercondylar notch stenosis and correlation to anterior cruciate ligament injuries. A prospective study. Am J Sports Med 1994; 22: 198-202; discussion 203.
225. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. Am J Sports Med 1998; 26: 402-408.
226. Bayer S, Meredith SJ, Wilson KW, de Sa D, Pauyo T, Byrne K, et al. Knee Morphological Risk Factors for Anterior Cruciate Ligament Injury: A Systematic Review. J Bone Joint Surg Am 2020; 102: 703-718.
227. Davila JN. Do Looks or Measurements Really Matter When It Comes to ACL Tears?: Commentary on an article by Steve Bayer, BA, et al.: "Knee Morphological Risk Factors for Anterior Cruciate Ligament Injury. A Systematic Review". J Bone Joint Surg Am 2020; 102: e37.
228. Hinz M, Brunner M, Winkler PW, Sanchez Carbonel JF, Fritsch L, Vieider RP, et al. The Posterior Tibial Slope Is Not Associated With Graft Failure and Functional Outcomes After Anatomic Primary Isolated Anterior Cruciate Ligament Reconstruction. Am J Sports Med 2023; 51: 3670-3676.
229. Moran TE, Driskill EK, Tagliero AJ, Klosterman EL, Ramamurti P, Reahl GB, et al. Combined tibial deflexion osteotomy and anterior cruciate ligament reconstruction improves knee function and stability: A systematic review. J ISAKOS 2024; 9: 709-716.
230. Zeng C, Borim FM, Lording T. Increased posterior tibial slope is a risk factor for anterior cruciate ligament injury and graft failure after reconstruction: A systematic review. J ISAKOS 2025; 12: 100854.
231. Voskuilen R, Dietvorst M, van der Steen M, Janssen RPA. A small notch width index, steeper medial and lateral tibial slope and higher lateral/medial tibial slope ratio are relevant knee morphological factors for ACL injuries in skeletally immature patients-A systematic review. J Exp Orthop 2025; 12: e70211.
232. Vasudevan RS, Rupp GE, Zogby AM, Wilps T, Paras T, Pennock AT. Decreased Posterior Tibial Slope and Its Association With Pediatric Posterior Cruciate Ligament Injury. Am J Sports Med 2024; 52: 1498-1504.
233. Nedaie S, Vivekanantha P, O'Hara K, Slawaska-Eng D, Cohen D, Abouali J, et al. Decreased posterior tibial slope is a risk factor for primary posterior cruciate ligament rupture and posterior cruciate ligament reconstruction failure: A systematic review. Knee Surg Sports Traumatol Arthrosc 2024; 32: 167-180.
234. Livingston LA, Mandigo JL. Bilateral within-subject Q angle asymmetry in young adult females and males. Biomed Sci Instrum 1997; 33: 112-117.
235. Nguyen AD, Boling MC, Levine B, Shultz SJ. Relationships between lower extremity alignment and the quadriceps angle. Clin J Sport Med 2009; 19: 201-206.
236. Alpay Y, Ezici A, Kurk MB, Ozyalvac ON, Akpinar E, Bayhan AI. Increased femoral anteversion related to infratrochanteric femoral torsion is associated with ACL rupture. Knee Surg Sports Traumatol Arthrosc 2020; 28: 2567-2571.

237. Kaneko M, Sakuraba K. Association between Femoral Anteversion and Lower Extremity Posture upon Single-leg Landing: Implications for Anterior Cruciate Ligament Injury. J Phys Ther Sci 2013; 25: 1213-1217.
238. Amraee D, Alizadeh MH, Minoonejhad H, Razi M, Amraee GH. Predictor factors for lower extremity malalignment and non-contact anterior cruciate ligament injuries in male athletes. Knee Surg Sports Traumatol Arthrosc 2017; 25: 1625-1631.
239. Pasanen K, Seppanen A, Leppanen M, Tokola K, Jarvela T, Vasankari T, et al. Knee laxity, joint hypermobility, femoral anteversion, hamstring extensibility and navicular drop as risk factors for non-contact ACL injury in female athletes: A 4.5-year prospective cohort study. Knee Surg Sports Traumatol Arthrosc 2025.
240. Lindskog J, Piussi R, Simonson R, Hogberg J, Samuelsson K, Thomee R, et al. Lower rates of return to sport in patients with generalised joint hypermobility two years after ACL reconstruction: a prospective cohort study. BMC Sports Sci Med Rehabil 2023; 15: 100.
241. Kamatsuki Y, Qvale MS, Steffen K, Wangensteen A, Krosshaug T. Anatomic Risk Factors for Initial and Secondary Noncontact Anterior Cruciate Ligament Injury: A Prospective Cohort Study in 880 Female Elite Handball and Soccer Players. Am J Sports Med 2025; 53: 123-131.
242. Zsidai B, Piussi R, Thomee R, Sundemo D, Musahl V, Samuelsson K, et al. Generalised joint hypermobility leads to increased odds of sustaining a second ACL injury within 12 months of return to sport after ACL reconstruction. Br J Sports Med 2023; 57: 972-978.
243. Steffen F, Rolvien T, Krause M, Hollander K, Hoenig T, Frosch KH, et al. Analysis of Genu Varum in Young Male Professional Soccer Players. Orthop J Sports Med 2025; 13: 23259671251330602.
244. Colyn W, Agricola R, Arnout N, Verhaar JA, Bellemans J. How does lower leg alignment differ between soccer players, other athletes, and non-athletic controls? Knee Surg Sports Traumatol Arthrosc 2016; 24: 3619-3626.
245. Isin A, Melekoglu T. Genu varum and football participation: Does football participation affect lower extremity alignment in adolescents? Knee 2020; 27: 1801-1810.
246. Moyer RF, Birmingham TB, Chesworth BM, Kean CO, Giffin JR. Alignment, body mass and their interaction on dynamic knee joint load in patients with knee osteoarthritis. Osteoarthritis Cartilage 2010; 18: 888-893.
247. Olah T, Reinhard J, Laschke MW, Goebel LKH, Walter F, Schmitt G, et al. Axial alignment is a critical regulator of knee osteoarthritis. Sci Transl Med 2022; 14: eabn0179.
248. Van Rossom S, Wesseling M, Smith CR, Thelen DG, Vanwanseele B, Dieter VA, et al. The influence of knee joint geometry and alignment on the tibiofemoral load distribution: A computational study. Knee 2019; 26: 813-823.
249. Parsons JL, Coen SE, Bekker S. Anterior cruciate ligament injury: towards a gendered environmental approach. Br J Sports Med 2021; 55: 984-990.

250. Fox A, Bonacci J, Hoffmann S, Nimphius S, Saunders N. Anterior cruciate ligament injuries in Australian football: should women and girls be playing? You're asking the wrong question. BMJ Open Sport Exerc Med 2020; 6: e000778.
251. Zbrojkiewicz D, Vertullo C, Grayson JE. Increasing rates of anterior cruciate ligament reconstruction in young Australians, 2000-2015. Med J Aust 2018; 208: 354-358.
252. Nicholls M, Ingvarsson T, Briem K. Younger age increases the risk of sustaining multiple concomitant injuries with an ACL rupture. Knee Surg Sports Traumatol Arthrosc 2021; 29: 2701-2708.
253. Webster KE, Hewett TE. Meta-analysis of meta-analyses of anterior cruciate ligament injury reduction training programs. J Orthop Res 2018; 36: 2696-2708.
254. Thorborg K, Krommes KK, Esteve E, Clausen MB, Bartels EM, Rathleff MS. Effect of specific exercise-based football injury prevention programmes on the overall injury rate in football: a systematic review and meta-analysis of the FIFA 11 and 11+ programmes. Br J Sports Med 2017; 51: 562-571.
255. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R. Prevention of noncontact anterior cruciate ligament injuries in elite and adolescent female team handball athletes. Instr Course Lect 2007; 56: 407-418.
256. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. BMJ 2005; 330: 449.
257. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R. Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. Clin J Sport Med 2003; 13: 71-78.
258. Hagglund M, Atroshi I, Wagner P, Walden M. Superior compliance with a neuromuscular training programme is associated with fewer ACL injuries and fewer acute knee injuries in female adolescent football players: secondary analysis of an RCT. Br J Sports Med 2013; 47: 974-979.
259. Walden M, Atroshi I, Magnusson H, Wagner P, Hagglund M. Prevention of acute knee injuries in adolescent female football players: cluster randomised controlled trial. BMJ 2012; 344: e3042.
260. Akerlund I, Walden M, Sonesson S, Hagglund M. Forty-five per cent lower acute injury incidence but no effect on overuse injury prevalence in youth floorball players (aged 12-17 years) who used an injury prevention exercise programme: two-armed parallel-group cluster randomised controlled trial. Br J Sports Med 2020; 54: 1028-1035.
261. Asker M, Hagglund M, Walden M, Kallberg H, Skillgate E. The Effect of Shoulder and Knee Exercise Programmes on the Risk of Shoulder and Knee Injuries in Adolescent Elite Handball Players: A Three-Armed Cluster Randomised Controlled Trial. Sports Med Open 2022; 8: 91.
262. Gill VS, Tummala SV, Boddu SP, Brinkman JC, McQuivey KS, Chhabra A. Biomechanics and situational patterns associated with anterior cruciate ligament injuries in the National Basketball Association (NBA). Br J Sports Med 2023; 57: 1395-1399.

263. Montgomery C, Blackburn J, Withers D, Tierney G, Moran C, Simms C. Mechanisms of ACL injury in professional rugby union: a systematic video analysis of 36 cases. Br J Sports Med 2018; 52: 994-1001.
264. Boden BP, Sheehan FT, Torg JS, Hewett TE. Noncontact anterior cruciate ligament injuries: mechanisms and risk factors. J Am Acad Orthop Surg 2010; 18: 520-527.
265. Gopinatth V, Smith MV, Matava MJ, Brophy RH, Knapik DM. Most Anterior Cruciate Ligament Injuries in Professional Athletes Occur Without Contact to the Injured Knee: A Systematic Review of Video Analysis Studies. Arthroscopy 2025; 41: 1155-1162 e1151.
266. Leppanen M, Pasanen K, Kujala UM, Vasankari T, Kannus P, Ayramo S, et al. Stiff Landings Are Associated With Increased ACL Injury Risk in Young Female Basketball and Floorball Players. Am J Sports Med 2017; 45: 386-393.
267. Krosshaug T, Nakamae A, Boden BP, Engebretsen L, Smith G, Slauterbeck JR, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. Am J Sports Med 2007; 35: 359-367.
268. Koga H, Nakamae A, Shima Y, Iwasa J, Myklebust G, Engebretsen L, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. Am J Sports Med 2010; 38: 2218-2225.
269. Olsen OE, Myklebust G, Engebretsen L, Bahr R. Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. Am J Sports Med 2004; 32: 1002-1012.
270. Della Villa F, Buckthorpe M, Grassi A, Nabiuzzi A, Tosarelli F, Zaffagnini S, et al. Systematic video analysis of ACL injuries in professional male football (soccer): injury mechanisms, situational patterns and biomechanics study on 134 consecutive cases. Br J Sports Med 2020; 54: 1423-1432.
271. Sheehan FT, Sipprell WH, 3rd, Boden BP. Dynamic sagittal plane trunk control during anterior cruciate ligament injury. Am J Sports Med 2012; 40: 1068-1074.
272. Hewett TE, Torg JS, Boden BP. Video analysis of trunk and knee motion during non-contact anterior cruciate ligament injury in female athletes: lateral trunk and knee abduction motion are combined components of the injury mechanism. Br J Sports Med 2009; 43: 417-422.
273. Boden BP, Sheehan FT. Mechanism of non-contact ACL injury: OREF Clinical Research Award 2021. J Orthop Res 2022; 40: 531-540.
274. Mattu AT, Ghali B, Linton V, Zheng A, Pike I. Prevention of Non-Contact Anterior Cruciate Ligament Injuries among Youth Female Athletes: An Umbrella Review. Int J Environ Res Public Health 2022; 19.
275. Myer GD, Sugimoto D, Thomas S, Hewett TE. The influence of age on the effectiveness of neuromuscular training to reduce anterior cruciate ligament injury in female athletes: a meta-analysis. Am J Sports Med 2013; 41: 203-215.
276. Martinez-Hernandez D, Quinn M, Jones P. Linear advancing actions followed by deceleration and turn are the most common movements preceding goals in male professional soccer. Sci Med Footb 2023; 7: 25-33.

277. Chandler PT, Pinder SJ, Curran JD, Gabbett TJ. Physical demands of training and competition in collegiate netball players. J Strength Cond Res 2014; 28: 2732-2737.

278. Stolen T, Chamari K, Castagna C, Wisloff U. Physiology of soccer: an update. Sports Med 2005; 35: 501-536.

279. Spencer M, Lawrence S, Rechichi C, Bishop D, Dawson B, Goodman C. Time-motion analysis of elite field hockey, with special reference to repeated-sprint activity. J Sports Sci 2004; 22: 843-850.

280. Granero-Gil P, Gomez-Carmona CD, Bastida-Castillo A, Rojas-Valverde D, de la Cruz E, Pino-Ortega J. Influence of playing position and laterality in centripetal force and changes of direction in elite soccer players. PLoS One 2020; 15: e0232123.

281. Morgan OJ, Drust B, Ade JD, Robinson MA. Change of direction frequency off the ball: new perspectives in elite youth soccer. Sci Med Footb 2022; 6: 473-482.

282. Bloomfield J, Polman R, O'Donoghue P. Physical Demands of Different Positions in FA Premier League Soccer. J Sports Sci Med 2007; 6: 63-70.

283. Donelon TA, Edwards J, Brown M, Jones PA, O'Driscoll J, Dos'Santos T. Differences in Biomechanical Determinants of ACL Injury Risk in Change of Direction Tasks Between Males and Females: A Systematic Review and Meta-Analysis. Sports Med Open 2024; 10: 29.

284. Faltstrom A, Asker M, Weiss N, Lyberg V, Walden M, Hagglund M, et al. Poor knee strength is associated with higher incidence of knee injury in adolescent female football players: The Karolinska football injury cohort. Knee Surg Sports Traumatol Arthrosc 2024.

285. Ramachandran AK, Pedley JS, Moeskops S, Oliver JL, Myer GD, Lloyd RS. Changes in Lower Limb Biomechanics Across Various Stages of Maturation and Implications for ACL Injury Risk in Female Athletes: a Systematic Review. Sports Med 2024; 54: 1851-1876.

286. Onell C, Skillgate E, Cote P, Walden M, Kallberg H, Hagglund M, et al. Dietary habits and knee and shoulder injury incidence in adolescent male and female handball players: the Swedish Handball Cohort. BMJ Open Sport Exerc Med 2025; 11: e002332.

287. Kosaka M, Nakase J, Numata H, Oshima T, Takata Y, Moriyama S, et al. Psychological traits regarding competitiveness are related to the incidence of anterior cruciate ligament injury in high school female athletes. Knee 2016; 23: 681-685.

288. Arundale AJH, Silvers-Granelli HJ, Myklebust G. ACL injury prevention: Where have we come from and where are we going? J Orthop Res 2022; 40: 43-54.

289. Thomson A, Whiteley R, Bleakley C. Higher shoe-surface interaction is associated with doubling of lower extremity injury risk in football codes: a systematic review and meta-analysis. Br J Sports Med 2015; 49: 1245-1252.

290. Thomson A, Whiteley R, Wilson M, Bleakley C. Six different football shoes, one playing surface and the weather; Assessing variation in shoe-surface traction over one season of elite football. PLoS One 2019; 14: e0216364.

291. Howard M, Solaru S, Kang HP, Bolia IK, Hatch GFR, Tibone JE, et al. Epidemiology of Anterior Cruciate Ligament Injury on Natural Grass Versus Artificial Turf in Soccer: 10-Year Data From the National Collegiate Athletic Association Injury Surveillance System. Orthop J Sports Med 2020; 8: 2325967120934434.
292. Gould HP, Lostetter SJ, Samuelson ER, Guyton GP. Lower Extremity Injury Rates on Artificial Turf Versus Natural Grass Playing Surfaces: A Systematic Review. Am J Sports Med 2023; 51: 1615-1621.
293. Kuitunen I, Immonen V, Pakarinen O, Mattila VM, Ponkilainen VT. Incidence of football injuries sustained on artificial turf compared to grass and other playing surfaces: a systematic review and meta-analysis. EClinicalMedicine 2023; 59: 101956.
294. Kushner JN, Swickley T, Bandi R, Lian J, Knecht MK, Sacca L. Examining the Prevalence of Anterior Cruciate Ligament Injuries on Artificial Turf Surfaces Compared to Natural Grass Surfaces in Athletes: A Scoping Review. Cureus 2024; 16: e63770.
295. Moller M, Myklebust G, Moller S, Wedderkopp N, Lind M, Nielsen RO. Handball playing volume and knee injury risk in youth handball: the influence of sex. J Sci Med Sport 2025; 28: 365-369.
296. Etzel CM, Meghani O, Owens BD, Kocher MS, Field AE. Predictors of Anterior Cruciate Ligament Tears in Adolescents and Young Adults. Orthop J Sports Med 2024; 12: 23259671241272699.
297. Crotti M, Heering T, Lander N, Fox A, Barnett LM, Duncan MJ. Extrinsic Risk Factors for Primary Noncontact Anterior Cruciate Ligament Injury in Adolescents Aged between 14 and 18 years: A Systematic Review. Sports Med 2024; 54: 875-894.
298. Myer GD, Jayanthi N, Difiori JP, Faigenbaum AD, Kiefer AW, Logerstedt D, et al. Sport Specialization, Part I: Does Early Sports Specialization Increase Negative Outcomes and Reduce the Opportunity for Success in Young Athletes? Sports Health 2015; 7: 437-442.
299. Mandorino M, Figueiredo AJ, Gjaka M, Tessitore A. Injury incidence and risk factors in youth soccer players: a systematic literature review. Part II: Intrinsic and extrinsic risk factors. Biol Sport 2023; 40: 27-49.
300. Schilaty ND, Bates NA, Sanders TL, Krych AJ, Stuart MJ, Hewett TE. Incidence of Second Anterior Cruciate Ligament Tears (1990-2000) and Associated Factors in a Specific Geographic Locale. Am J Sports Med 2017; 45: 1567-1573.
301. Sanders TG, Medynski MA, Feller JF, Lawhorn KW. Bone contusion patterns of the knee at MR imaging: footprint of the mechanism of injury. Radiographics 2000; 20 Spec No: S135-151.
302. Zhang L, Hacke JD, Garrett WE, Liu H, Yu B. Bone Bruises Associated with Anterior Cruciate Ligament Injury as Indicators of Injury Mechanism: A Systematic Review. Sports Med 2019; 49: 453-462.

303. Patel SA, Hageman J, Quatman CE, Wordeman SC, Hewett TE. Prevalence and location of bone bruises associated with anterior cruciate ligament injury and implications for mechanism of injury: a systematic review. Sports Med 2014; 44: 281-293.
304. Rosen MA, Jackson DW, Berger PE. Occult osseous lesions documented by magnetic resonance imaging associated with anterior cruciate ligament ruptures. Arthroscopy 1991; 7: 45-51.
305. Speer KP, Spritzer CE, Bassett FH, 3rd, Feagin JA, Jr., Garrett WE, Jr. Osseous injury associated with acute tears of the anterior cruciate ligament. Am J Sports Med 1992; 20: 382-389.
306. Vellet AD, Marks PH, Fowler PJ, Munro TG. Occult posttraumatic osteochondral lesions of the knee: prevalence, classification, and short-term sequelae evaluated with MR imaging. Radiology 1991; 178: 271-276.
307. Speer KP, Warren RF, Wickiewicz TL, Horowitz L, Henderson L. Observations on the injury mechanism of anterior cruciate ligament tears in skiers. Am J Sports Med 1995; 23: 77-81.
308. Baessler AM, Buchman JM, Witte DA, Spence DD, Throckmorton TW, Brolin TJ, et al. Skeletally Immature Patients With Classic Anterior Cruciate Ligament Bone Bruise Patterns Have a Higher Likelihood of Having an Intact Anterior Cruciate Ligament Compared With Skeletally Mature Patients. J Pediatr Orthop 2023; 43: 13-17.
309. Hinton RY, Rivera VR, Pautz MJ, Sponseller PD. Ligamentous laxity of the knee during childhood and adolescence. J Pediatr Orthop 2008; 28: 184-187.
310. Yao L, Lee JK. Occult intraosseous fracture: detection with MR imaging. Radiology 1988; 167: 749-751.
311. Hernandez-Molina G, Guermazi A, Niu J, Gale D, Goggins J, Amin S, et al. Central bone marrow lesions in symptomatic knee osteoarthritis and their relationship to anterior cruciate ligament tears and cartilage loss. Arthritis Rheum 2008; 58: 130-136.
312. Felson DT, McLaughlin S, Goggins J, LaValley MP, Gale ME, Totterman S, et al. Bone marrow edema and its relation to progression of knee osteoarthritis. Ann Intern Med 2003; 139: 330-336.
313. Kaplan PA, Walker CW, Kilcoyne RF, Brown DE, Tusek D, Dussault RG. Occult fracture patterns of the knee associated with anterior cruciate ligament tears: assessment with MR imaging. Radiology 1992; 183: 835-838.
314. Mink JH, Deutsch AL. Occult cartilage and bone injuries of the knee: detection, classification, and assessment with MR imaging. Radiology 1989; 170: 823-829.
315. Roemer FW, Bohndorf K. Long-term osseous sequelae after acute trauma of the knee joint evaluated by MRI. Skeletal Radiol 2002; 31: 615-623.
316. Bernholt DL, DePhillipo NN, Crawford MD, Aman ZS, Grantham WJ, LaPrade RF. Incidence of Displaced Posterolateral Tibial Plateau and Lateral Femoral Condyle Impaction Fractures in the Setting of Primary Anterior Cruciate Ligament Tear. Am J Sports Med 2020; 48: 545-553.

317. Nakamae A, Engebretsen L, Bahr R, Krosshaug T, Ochi M. Natural history of bone bruises after acute knee injury: clinical outcome and histopathological findings. Knee Surg Sports Traumatol Arthrosc 2006; 14: 1252-1258.
318. Filardo G, Andriolo L, di Laura Frattura G, Napoli F, Zaffagnini S, Candrian C. Bone bruise in anterior cruciate ligament rupture entails a more severe joint damage affecting joint degenerative progression. Knee Surg Sports Traumatol Arthrosc 2019; 27: 44-59.
319. Ward P, Chang P, Radtke L, Brophy RH. Clinical Implications of Bone Bruise Patterns Accompanying Anterior Cruciate Ligament Tears. Sports Health 2022; 14: 585-591.
320. Familiari F, Tollefson LV, Izzo A, Russo R, Mercurio M, Gasparini G, et al. High-grade medial femoral condyle and medial tibial plateau bone bruises predict ramp lesions of the medial meniscus in patients with anterior cruciate ligament tears: A prospective clinical and MRI evaluation. J Exp Orthop 2025; 12: e70238.
321. Cristiani R, van de Bunt F, Kvist J, Stalman A. High prevalence of meniscal ramp lesions in anterior cruciate ligament injuries. Knee Surg Sports Traumatol Arthrosc 2023; 31: 316-324.
322. Song GY, Zhang H, Wang QQ, Zhang J, Li Y, Feng H. Bone Contusions After Acute Noncontact Anterior Cruciate Ligament Injury Are Associated With Knee Joint Laxity, Concomitant Meniscal Lesions, and Anterolateral Ligament Abnormality. Arthroscopy 2016; 32: 2331-2341.
323. Lintin L, Chowdhury R, Yoong P, Chung SL, Mansour R, Teh J, et al. The anterolateral ligament in acute knee trauma: patterns of injury on MR imaging. Skeletal Radiol 2020; 49: 1765-1772.
324. Kia C, Cavanaugh Z, Gillis E, Dwyer C, Chadayammuri V, Muench LN, et al. Size of Initial Bone Bruise Predicts Future Lateral Chondral Degeneration in ACL Injuries: A Radiographic Analysis. Orthop J Sports Med 2020; 8: 2325967120916834.
325. Galloway C, Ward H, Higbie S, Kleihege J, Kumaravel M, Lowe WR, et al. Relationship Between Bone Bruise Volume and Patient Outcomes After ACL Reconstruction. Orthop J Sports Med 2023; 11: 23259671221146205.
326. Lattermann C, Jacobs CA, Reinke EK, Scaramuzza EA, Huston LJ, Dunn WR, et al. Are Bone Bruise Characteristics and Articular Cartilage Pathology Associated with Inferior Outcomes 2 and 6 Years After Anterior Cruciate Ligament Reconstruction? Cartilage 2017; 8: 139-145.
327. Boks SS, Vroegindeweij D, Koes BW, Bernsen RM, Hunink MG, Bierma-Zeinstra SM. MRI follow-up of posttraumatic bone bruises of the knee in general practice. AJR Am J Roentgenol 2007; 189: 556-562.
328. Theologis AA, Kuo D, Cheng J, Bolbos RI, Carballido-Gamio J, Ma CB, 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: 65-76.

329. Papalia R, Torre G, Vasta S, Zampogna B, Pedersen DR, Denaro V, et al. Bone bruises in anterior cruciate ligament injured knee and long-term outcomes. A review of the evidence. Open Access J Sports Med 2015; 6: 37-48.
330. Agostinone P, Di Paolo S, Lucidi GA, Dal Fabbro G, Grassi A, Zaffagnini S. Severe bicompartmental bone bruise is associated with rotatory instability in anterior cruciate ligament injury. Knee Surg Sports Traumatol Arthrosc 2022; 30: 1725-1732.
331. Bernholt DL, Dornan GJ, DePhillipo NN, Aman ZS, Kennedy MI, LaPrade RF. High-Grade Posterolateral Tibial Plateau Impaction Fractures in the Setting of a Primary Anterior Cruciate Ligament Tear Are Correlated With an Increased Preoperative Pivot Shift and Inferior Postoperative Outcomes After Anterior Cruciate Ligament Reconstruction. Am J Sports Med 2020; 48: 2185-2194.
332. Sward P, Struglics A, Englund M, Roos HP, Frobell RB. Soft tissue knee injury with concomitant osteochondral fracture is associated with higher degree of acute joint inflammation. Am J Sports Med 2014; 42: 1096-1102.
333. Harilainen A, Myllynen P, Antila H, Seitsalo S. The significance of arthroscopy and examination under anaesthesia in the diagnosis of fresh injury haemarthrosis of the knee joint. Injury 1988; 19: 21-24.
334. Bollen SR, Scott BW. Rupture of the anterior cruciate ligament--a quiet epidemic? Injury 1996; 27: 407-409.
335. Parwaiz H, Teo AQ, Servant C. Anterior cruciate ligament injury: A persistently difficult diagnosis. Knee 2016; 23: 116-120.
336. Guillodo Y, Rannou N, Dubrana F, Lefevre C, Saraux A. Diagnosis of anterior cruciate ligament rupture in an emergency department. J Trauma 2008; 65: 1078-1082.
337. Allott NEH, Banger MS, McGregor AH. Evaluating the diagnostic pathway for acute ACL injuries in trauma centres: a systematic review. BMC Musculoskelet Disord 2022; 23: 649.
338. Ball S, Haddad FS. The impact of an Acute Knee Clinic. Ann R Coll Surg Engl 2010; 92: 685-688.
339. Clifford C, Ayre C, Edwards L, Guy S, Jones A. Acute knee clinics are effective in reducing delay to diagnosis following anterior cruciate ligament injury. Knee 2021; 30: 267-274.
340. Wang JH, Lee JH, Cho Y, Shin JM, Lee BH. Efficacy of knee joint aspiration in patients with acute ACL injury in the emergency department. Injury 2016; 47: 1744-1749.
341. Bergstein VE, Ahiarakwe U, Haft M, Fox H, Best MJ. Decreasing Incidence of Anterior Cruciate Ligament Tears and Increasing Utilization of Anterior Cruciate Ligament Reconstruction in the United States From 2010 to 2020. Arthroscopy 2025; 41: 1912-1918 e1911.
342. LaBella CR, Hennrikus W, Hewett TE, Council on Sports M, Fitness, Section on O. Anterior cruciate ligament injuries: diagnosis, treatment, and prevention. Pediatrics 2014; 133: e1437-1450.

343. Diermeier T, Rothrauff BB, Engebretsen L, Lynch AD, Svantesson E, Hamrin Senorski E, et al. Treatment after anterior cruciate ligament injury: Panther Symposium ACL Treatment Consensus Group. J ISAKOS 2021; 6: 129-137.
344. Collins JE, Katz JN, Donnell-Fink LA, Martin SD, Losina E. Cumulative incidence of ACL reconstruction after ACL injury in adults: role of age, sex, and race. Am J Sports Med 2013; 41: 544-549.
345. Sanders TL, Maradit Kremers H, Bryan AJ, Kremers WK, Levy BA, Dahm DL, et al. Incidence of and Factors Associated With the Decision to Undergo Anterior Cruciate Ligament Reconstruction 1 to 10 Years After Injury. Am J Sports Med 2016; 44: 1558-1564.
346. Mall NA, Chalmers PN, Moric M, Tanaka MJ, Cole BJ, Bach BR, Jr., et al. Incidence and trends of anterior cruciate ligament reconstruction in the United States. Am J Sports Med 2014; 42: 2363-2370.
347. Paudel YR, Sommerfeldt M, Voaklander D. Increasing incidence of anterior cruciate ligament reconstruction: a 17-year population-based study. Knee Surg Sports Traumatol Arthrosc 2023; 31: 248-255.
348. Lundblad M, Hagglund M, Thomee C, Hamrin Senorski E, Ekstrand J, Karlsson J, et al. Medial collateral ligament injuries of the knee in male professional football players: a prospective three-season study of 130 cases from the UEFA Elite Club Injury Study. Knee Surg Sports Traumatol Arthrosc 2019; 27: 3692-3698.
349. Svantesson E, Hamrin Senorski E, Alentorn-Geli E, Westin O, Sundemo D, Grassi A, et al. Increased risk of ACL revision with non-surgical treatment of a concomitant medial collateral ligament injury: a study on 19,457 patients from the Swedish National Knee Ligament Registry. Knee Surg Sports Traumatol Arthrosc 2019; 27: 2450-2459.
350. Niknam K, Goldberg D, Markes AR, Feeley BT, Zhang AL, Ma CB, et al. Concomitant Medial Collateral Ligament Injury Increases the Risk of Revision Anterior Cruciate Ligament Reconstruction. Arthroscopy 2024.
351. Shultz CL, Poehlein E, Morriss NJ, Green CL, Hu J, Lander S, et al. Nonoperative Management, Repair, or Reconstruction of the Medial Collateral Ligament in Combined Anterior Cruciate and Medial Collateral Ligament Injuries-Which Is Best? A Systematic Review and Meta-analysis. Am J Sports Med 2024; 52: 522-534.
352. Svantesson J, Piussi R, Weissglas E, Svantesson E, Horvath A, Borjesson E, et al. Shedding light on the non-operative treatment of the forgotten side of the knee: rehabilitation of medial collateral ligament injuries-a systematic review. BMJ Open Sport Exerc Med 2024; 10: e001750.
353. Lucidi GA, Solaro L, Grassi A, Alhalalmeh MI, Ratti S, Manzoli L, et al. Current trends in the medial side of the knee: not only medial collateral ligament (MCL). J Orthop Traumatol 2024; 25: 69.
354. Miyamoto RG, Bosco JA, Sherman OH. Treatment of medial collateral ligament injuries. J Am Acad Orthop Surg 2009; 17: 152-161.
355. Hunter DJ, Black AM, Culos-Reed SN, Lun VMY, Mohtadi NG. Exploring Predictors of Brace-Wearing Adherence in Non-Surgical Treatment of Acute Knee Medial Collateral Ligament Injuries. Clin J Sport Med 2025; 35: 423-430.

356. Tan SHS, Kripesh A, Chan CX, Krishna L. Gender Differences in Intra-articular and Extra-articular Injuries Associated with Acute Anterior Cruciate Ligament Ruptures. J Knee Surg 2019; 32: 616-619.
357. Wessel LM, Scholz S, Rusch M. Characteristic pattern and management of intra-articular knee lesions in different pediatric age groups. J Pediatr Orthop 2001; 21: 14-19.
358. Luhmann SJ. Acute traumatic knee effusions in children and adolescents. J Pediatr Orthop 2003; 23: 199-202.
359. Wessel LM, Scholz S, Rusch M, Kopke J, Loff S, Duchene W, et al. Hemarthrosis after trauma to the pediatric knee joint: what is the value of magnetic resonance imaging in the diagnostic algorithm? J Pediatr Orthop 2001; 21: 338-342.
360. Wolfe S, Varacallo MA, Thomas JD, Carroll JJ, Kahwaji CI. Patellar Instability. In: StatPearls Treasure Island (FL)2025.
361. Majewski M, Susanne H, Klaus S. Epidemiology of athletic knee injuries: A 10-year study. Knee 2006; 13: 184-188.
362. Blond L, Askenberger M, Stephen J, Akmese R, Balcarek P, El Attal R, et al. Management of first-time patellar dislocation: The ESSKA 2024 formal consensus-Part 1. Knee Surg Sports Traumatol Arthrosc 2025; 33: 1925-1932.
363. Lyons JG, Hudson TL, Krishnamurthy AB. Epidemiology of patellar dislocations in the United States from 2001 to 2020: results of a national emergency department database. Phys Sportsmed 2024; 52: 26-35.
364. Kell D, Houlihan N, Huang K, Sarkar S, Schlechter J, Williams BA. Patellar Instability Events Requiring Emergency Department Management - A 20-year Analysis. J Pediatr Soc North Am 2025; 10: 100152.
365. McFarlane KH, Coene RP, Feldman L, Miller PE, Heyworth BE, Kramer DE, et al. Increased incidence of acute patellar dislocations and patellar instability surgical procedures across the United States in paediatric and adolescent patients. J Child Orthop 2021; 15: 149-156.
366. Poorman MJ, Talwar D, Sanjuan J, Baldwin KD, Sutliff N, Franklin CC. Increasing hospital admissions for patellar instability: a national database study from 2004 to 2017. Phys Sportsmed 2020; 48: 215-221.
367. Fithian DC, Paxton EW, Stone ML, Silva P, Davis DK, Elias DA, et al. Epidemiology and natural history of acute patellar dislocation. Am J Sports Med 2004; 32: 1114-1121.
368. Dejour DH, Mazy D, Pineda T, Cance N, Dan MJ, Giovannetti de Sanctis E. Patellar instability: current approach. EFORT Open Rev 2025; 10: 378-387.
369. Lewallen L, McIntosh A, Dahm D. First-Time Patellofemoral Dislocation: Risk Factors for Recurrent Instability. J Knee Surg 2015; 28: 303-309.
370. Mitchell J, Magnussen RA, Collins CL, Currie DW, Best TM, Comstock RD, et al. Epidemiology of Patellofemoral Instability Injuries Among High School Athletes in the United States. Am J Sports Med 2015; 43: 1676-1682.
371. Stanitski CL. Articular hypermobility and chondral injury in patients with acute patellar dislocation. Am J Sports Med 1995; 23: 146-150.

372. Stanitski CL, Paletta GA, Jr. Articular cartilage injury with acute patellar dislocation in adolescents. Arthroscopic and radiographic correlation. Am J Sports Med 1998; 26: 52-55.

373. Nomura E, Inoue M, Kurimura M. Chondral and osteochondral injuries associated with acute patellar dislocation. Arthroscopy 2003; 19: 717-721.

374. Nomura E, Inoue M. Cartilage lesions of the patella in recurrent patellar dislocation. Am J Sports Med 2004; 32: 498-502.

375. Hawkins RJ, Bell RH, Anisette G. Acute patellar dislocations. The natural history. Am J Sports Med 1986; 14: 117-120.

376. Vollnberg B, Koehlitz T, Jung T, Scheffler S, Hoburg A, Khandker D, et al. Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation. Eur Radiol 2012; 22: 2347-2356.

377. Seeley MA, Knesek M, Vanderhave KL. Osteochondral injury after acute patellar dislocation in children and adolescents. J Pediatr Orthop 2013; 33: 511-518.

378. Outerbridge RE. The etiology of chondromalacia patellae. J Bone Joint Surg Br 1961; 43-B: 752-757.

379. Uimonen M, Ponkilainen V, Paloneva J, Mattila VM, Nurmi H, Repo JP. Characteristics of Osteochondral Fractures Caused by Patellar Dislocation. Orthop J Sports Med 2021; 9: 2325967120974649.

380. Felus J, Kowalczyk B, Starmach M, Wyrobek L. Osteochondral Fractures in Acute Patellar Dislocations in Adolescents: Midterm Results of Surgical Treatment. Orthop J Sports Med 2022; 10: 23259671221107608.

381. Wang A, Torres-Izquierdo B, Nepple JJ. Osteochondral Fractures in Adolescents With First-time Patellar Dislocation: Three-dimensional Characterization and Association With Radiographic Features. Am J Sports Med 2025; 53: 682-689.

382. Zheng L, Ding HY, Feng Y, Sun BS, Zhu LL, Zhang GY. Gender-related differences in concomitant articular injuries after acute lateral patellar dislocation. Injury 2021; 52: 1549-1555.

383. Balcarek P, Blond L, Beaufils P, Askenberger M, Stephen JM, Akmese R, et al. Management of first-time patellar dislocation: The ESSKA 2024 formal consensus-Part 2. Knee Surg Sports Traumatol Arthrosc 2025.

384. Gesslein M, Merkl C, Bail HJ, Krutsch V, Biber R, Schuster P. Refixation of Large Osteochondral Fractures After Patella Dislocation Shows Better Mid- to Long-Term Outcome Compared With Debridement. Cartilage 2021; 13: 966S-973S.

385. Fabricant PD, Yen YM, Kramer DE, Kocher MS, Micheli LJ, Lawrence JTR, et al. Fixation of Traumatic Chondral-Only Fragments of the Knee in Pediatric and Adolescent Athletes: A Retrospective Multicenter Report. Orthop J Sports Med 2018; 6: 2325967117753140.

386. Palmu S, Kallio PE, Donell ST, Helenius I, Nietosvaara Y. Acute patellar dislocation in children and adolescents: a randomized clinical trial. J Bone Joint Surg Am 2008; 90: 463-470.

387. Drapeau-Zgoralski V, Swift B, Caines A, Kerrigan A, Carsen S, Pickell M. Lateral Patellar Instability. J Bone Joint Surg Am 2023; 105: 397-409.

388. Pagliazzi G, Napoli F, Previtali D, Filardo G, Zaffagnini S, Candrian C. A Meta-analysis of Surgical Versus Nonsurgical Treatment of Primary Patella Dislocation. Arthroscopy 2019; 35: 2469-2481.
389. Hinckel B, Smith J, Tanaka MJ, Matsushita T, Martinez-Cano JP. Patellofemoral instability part 1 (When to operate and soft tissue procedures): State of the art. J ISAKOS 2025; 10: 100278.
390. Vermeulen D, van der Valk MR, Kaas L. Plaster, splint, brace, tape or functional mobilization after first-time patellar dislocation: what's the evidence? EFORT Open Rev 2019; 4: 110-114.
391. Smith TO, Davies L, Donell ST. Immobilization Regime Following Lateral Patellar Dislocation: A Systematic Review and Meta-Analysis of the Current Evidence Base. Eur J Trauma Emerg Surg 2010; 36: 353-360.
392. Hinckel BB, Liebensteiner M, Smith JT, Gomes Gobbi R, Arendt EA. Patellofemoral instability part 2 (Bony procedure for patellar surgical stabilization): State of the art. J ISAKOS 2025; 10: 100347.
393. Hinckel BB, Conte P, Smith JT, Nakamura N, Kon E. Patellofemoral joint including patellar instability part 3 (Patellofemoral cartilage treatment): State of the art. J ISAKOS 2025; 10: 100366.
394. Askenberger M, Janarv PM, Finnbogason T, Arendt EA. Morphology and Anatomic Patellar Instability Risk Factors in First-Time Traumatic Lateral Patellar Dislocations: A Prospective Magnetic Resonance Imaging Study in Skeletally Immature Children. Am J Sports Med 2017; 45: 50-58.
395. Christensen TC, Sanders TL, Pareek A, Mohan R, Dahm DL, Krych AJ. Risk Factors and Time to Recurrent Ipsilateral and Contralateral Patellar Dislocations. Am J Sports Med 2017; 45: 2105-2110.
396. Frobell RB, Roos HP, Roos EM, Roemer FW, Ranstam J, Lohmander LS. Treatment for acute anterior cruciate ligament tear: five year outcome of randomised trial. BMJ 2013; 346: f232.
397. Reijman M, Eggerding V, van Es E, van Arkel E, van den Brand I, van Linge J, et al. Early surgical reconstruction versus rehabilitation with elective delayed reconstruction for patients with anterior cruciate ligament rupture: COMPARE randomised controlled trial. BMJ 2021; 372: n375.
398. Eggerding V, Reijman M, Meuffels DE, van Es E, van Arkel E, van den Brand I, et al. ACL reconstruction for all is not cost-effective after acute ACL rupture. Br J Sports Med 2022; 56: 24-28.
399. Beard DJ, Davies L, Cook JA, Stokes J, Leal J, Fletcher H, et al. Comparison of surgical or non-surgical management for non-acute anterior cruciate ligament injury: the ACL SNNAP RCT. Health Technol Assess 2024; 28: 1-97.
400. Monk AP, Davies LJ, Hopewell S, Harris K, Beard DJ, Price AJ. Surgical versus conservative interventions for treating anterior cruciate ligament injuries. Cochrane Database Syst Rev 2016; 4: CD011166.

401. Vermeijden HD, Yang XA, Rademakers MV, Kerkhoffs G, van der List JP, DiFelice GS. Early and Delayed Surgery for Isolated ACL and Multiligamentous Knee Injuries Have Equivalent Results: A Systematic Review and Meta-analysis. Am J Sports Med 2023; 51: 1106-1116.
402. Agolley D, Gabr A, Benjamin-Laing H, Haddad FS. Successful return to sports in athletes following non-operative management of acute isolated posterior cruciate ligament injuries: medium-term follow-up. Bone Joint J 2017; 99-B: 774-778.
403. Skou ST, Holmich P, Lind M, Jensen HP, Jensen C, Garval M, et al. Early Surgery or Exercise and Education for Meniscal Tears in Young Adults. NEJM Evid 2022; 1: EVIDoa2100038.
404. Damsted C, Thorlund JB, Holmich P, Lind M, Varnum C, Villumsen MD, et al. Effect of exercise therapy versus surgery on mechanical symptoms in young patients with a meniscal tear: a secondary analysis of the DREAM trial. Br J Sports Med 2023; 57: 521-527.
405. Damsted C, Skou ST, Holmich P, Lind M, Varnum C, Jensen HP, et al. Early Surgery Versus Exercise Therapy and Patient Education for Traumatic and Nontraumatic Meniscal Tears in Young Adults-An Exploratory Analysis From the DREAM Trial. J Orthop Sports Phys Ther 2024; 54: 340-349.
406. Clausen SH, Skou ST, Boesen MP, Radev DI, Kurt EY, Damsted C, et al. Two-year MRI-defined structural damage and patient-reported outcomes following surgery or exercise for meniscal tears in young adults. Br J Sports Med 2023; 57: 1566-1572.
407. Clausen SH, Boesen M, Thorlund JB, Vach W, Lind M, Holmich P, et al. Can Baseline MRI Findings Identify Who Responds Better to Early Surgery Versus Exercise and Education in Young Patients With Meniscal Tears? A Subgroup Analysis From the DREAM Trial. J Orthop Sports Phys Ther 2025; 55: 1-11.
408. van der Graaff SJA, Reijman M, Meuffels DE, Koopmanschap MA, Group SS, Eijgenraam SM, et al. Cost-effectiveness of arthroscopic partial meniscectomy versus physical therapy for traumatic meniscal tears in patients aged under 45 years. Bone Joint J 2023; 105-B: 1177-1183.
409. van der Graaff SJA, Eijgenraam SM, Meuffels DE, van Es EM, Verhaar JAN, Hofstee DJ, et al. Arthroscopic partial meniscectomy versus physical therapy for traumatic meniscal tears in a young study population: a randomised controlled trial. Br J Sports Med 2022; 56: 870-876.
410. Lee DY, Kang DG, Jo HS, Heo SJ, Bae JH, Hwang SC. A systematic review and meta-analysis comparing conservative and surgical treatments for acute patellar dislocation in children and adolescents. Knee Surg Relat Res 2023; 35: 18.
411. Yao LW, Zhang C, Liu Y, Cao DG, Li DJ, Xu DD, et al. Comparison operative and conservative management for primary patellar dislocation: an up-to-date meta-analysis. Eur J Orthop Surg Traumatol 2015; 25: 783-788.
412. Saccomanno MF, Sircana G, Fodale M, Donati F, Milano G. Surgical versus conservative treatment of primary patellar dislocation. A systematic review and meta-analysis. Int Orthop 2016; 40: 2277-2287.

413. Smith TO, Gaukroger A, Metcalfe A, Hing CB. Surgical versus non-surgical interventions for treating patellar dislocation. Cochrane Database Syst Rev 2023; 1: CD008106.
414. Mostrom EB, Mikkelsen C, Weidenhielm L, Janarv PM. Long-term follow-up of nonoperatively and operatively treated acute primary patellar dislocation in skeletally immature patients. ScientificWorldJournal 2014; 2014: 473281.
415. Sihvonen R, Paavola M, Malmivaara A, Itala A, Joukainen A, Kalske J, et al. Arthroscopic partial meniscectomy for a degenerative meniscus tear: a 5 year follow-up of the placebo-surgery controlled FIDELITY (Finnish Degenerative Meniscus Lesion Study) trial. Br J Sports Med 2020; 54: 1332-1339.
416. Whittaker JL, Culvenor AG, Juhl CB, Berg B, Bricca A, Filbay SR, et al. OPTIKNEE 2022: consensus recommendations to optimise knee health after traumatic knee injury to prevent osteoarthritis. Br J Sports Med 2022; 56: 1393-1405.
417. van de Graaf VA, Bloembergen CM, Willigenburg NWP, Noorduyn JCAM, Saris DB, Harris IA, et al. Can even experienced orthopaedic surgeons predict who will benefit from surgery when patients present with degenerative meniscal tears? A survey of 194 orthopaedic surgeons who made 3880 predictions. Br J Sports Med 2020; 54: 354-359.
418. Barenius B, Ponzer S, Shalabi A, Bujak R, Norlen L, Eriksson K. Increased risk of osteoarthritis after anterior cruciate ligament reconstruction: a 14-year follow-up study of a randomized controlled trial. Am J Sports Med 2014; 42: 1049-1057.
419. Webster KE, Hewett TE. Anterior Cruciate Ligament Injury and Knee Osteoarthritis: An Umbrella Systematic Review and Meta-analysis. Clin J Sport Med 2022; 32: 145-152.
420. 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: 269-273.
421. Lohmander LS, Ostenberg 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: 3145-3152.
422. Sanders TL, Kremers HM, Bryan AJ, Fruth KM, Larson DR, Pareek A, et al. Is Anterior Cruciate Ligament Reconstruction Effective in Preventing Secondary Meniscal Tears and Osteoarthritis? Am J Sports Med 2016; 44: 1699-1707.
423. Kvist J, Filbay S, Andersson C, Ardern CL, Gauffin H. Radiographic and Symptomatic Knee Osteoarthritis 32 to 37 Years After Acute Anterior Cruciate Ligament Rupture. Am J Sports Med 2020; 48: 2387-2394.
424. Khan T, Alvand A, Prieto-Alhambra D, Culliford DJ, Judge A, Jackson WF, et al. ACL and meniscal injuries increase the risk of primary total knee replacement for osteoarthritis: a matched case-control study using the Clinical Practice Research Datalink (CPRD). Br J Sports Med 2019; 53: 965-968.
425. Leroux T, Ogilvie-Harris D, Dwyer T, Chahal J, Gandhi R, Mahomed N, et al. The risk of knee arthroplasty following cruciate ligament reconstruction: a population-based matched cohort study. J Bone Joint Surg Am 2014; 96: 2-10.

426. Migliorini F, Marsilio E, Cuozzo F, Oliva F, Eschweiler J, Hildebrand F, et al. Chondral and Soft Tissue Injuries Associated to Acute Patellar Dislocation: A Systematic Review. Life (Basel) 2021; 11.
427. Arendt EA, Berruto M, Filardo G, Ronga M, Zaffagnini S, Farr J, et al. Early osteoarthritis of the patellofemoral joint. Knee Surg Sports Traumatol Arthrosc 2016; 24: 1836-1844.
428. Maenpaa H, Lehto MU. Patellofemoral osteoarthritis after patellar dislocation. Clin Orthop Relat Res 1997: 156-162.
429. Sillanpaa P, Mattila VM, Visuri T, Maenpaa H, Pihlajamaki H. Ligament reconstruction versus distal realignment for patellar dislocation. Clin Orthop Relat Res 2008; 466: 1475-1484.
430. Arias C, Lustig S. Physiopathology of patello-femoral osteoarthritis: current concepts. J ISAKOS 2024; 9: 806-813.
431. Bedson J, Croft PR. The discordance between clinical and radiographic knee osteoarthritis: a systematic search and summary of the literature. BMC Musculoskelet Disord 2008; 9: 116.
432. Identeg F, Senorski EH, Svantesson E, Samuelsson K, Sernert N, Kartus JT, et al. Poor Associations Between Radiographic Tibiofemoral Osteoarthritis and Patient-Reported Outcomes at 16 Years After Anterior Cruciate Ligament Reconstruction. Orthop J Sports Med 2020; 8: 2325967120951174.
433. Toivanen AT, Heliovaara M, Impivaara O, Arokoski JP, Knekt P, Lauren H, et al. Obesity, physically demanding work and traumatic knee injury are major risk factors for knee osteoarthritis--a population-based study with a follow-up of 22 years. Rheumatology (Oxford) 2010; 49: 308-314.
434. 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: 497-506.
435. Alentorn-Geli E, Samuelsson K, Musahl V, Green CL, Bhandari M, Karlsson J. The Association of Recreational and Competitive Running With Hip and Knee Osteoarthritis: A Systematic Review and Meta-analysis. J Orthop Sports Phys Ther 2017; 47: 373-390.
436. Evers BJ, Van Den Bosch MHJ, Blom AB, van der Kraan PM, Koeter S, Thurlings RM. Post-traumatic knee osteoarthritis; the role of inflammation and hemarthrosis on disease progression. Front Med (Lausanne) 2022; 9: 973870.
437. van Meegeren ME, Roosendaal G, Jansen NW, Lafeber FP, Mastbergen SC. Blood-Induced Joint Damage: The Devastating Effects of Acute Joint Bleeds versus Micro-Bleeds. Cartilage 2013; 4: 313-320.
438. Valentino LA. Blood-induced joint disease: the pathophysiology of hemophilic arthropathy. J Thromb Haemost 2010; 8: 1895-1902.
439. Manco-Johnson MJ, Abshire TC, Shapiro AD, Riske B, Hacker MR, Kilcoyne R, et al. Prophylaxis versus episodic treatment to prevent joint disease in boys with severe hemophilia. N Engl J Med 2007; 357: 535-544.

440. Lyons LP, Weinberg JB, Wittstein JR, McNulty AL. Blood in the joint: effects of hemarthrosis on meniscus health and repair techniques. Osteoarthritis Cartilage 2021; 29: 471-479.
441. Larsen P, Rathleff MS, Roos EM, Elsoe R. Knee injury and osteoarthritis outcome score (KOOS) - National record-based reference values. Knee 2023; 43: 144-152.
442. Collins NJ, Misra D, Felson DT, Crossley KM, Roos EM. Measures of knee function: International Knee Documentation Committee (IKDC) Subjective Knee Evaluation Form, Knee Injury and Osteoarthritis Outcome Score (KOOS), Knee Injury and Osteoarthritis Outcome Score Physical Function Short Form (KOOS-PS), Knee Outcome Survey Activities of Daily Living Scale (KOS-ADL), Lysholm Knee Scoring Scale, Oxford Knee Score (OKS), Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Activity Rating Scale (ARS), and Tegner Activity Score (TAS). Arthritis Care Res (Hoboken) 2011; 63 Suppl 11: S208-228.
443. Magnussen RA, Verlage M, Stock E, Zurek L, Flanigan DC, Tompkins M, et al. Primary patellar dislocations without surgical stabilization or recurrence: how well are these patients really doing? Knee Surg Sports Traumatol Arthrosc 2017; 25: 2352-2356.
444. Kannus P, Jarvinen M. Long-term prognosis of nonoperatively treated acute knee distortions having primary hemarthrosis without clinical instability. Am J Sports Med 1987; 15: 138-143.
445. Mathison DJ, Teach SJ. Approach to knee effusions. Pediatr Emerg Care 2009; 25: 773-786; quiz 787-778.
446. Allaire R, Muriuki M, Gilbertson L, Harner CD. Biomechanical consequences of a tear of the posterior root of the medial meniscus. Similar to total meniscectomy. J Bone Joint Surg Am 2008; 90: 1922-1931.
447. Cinque ME, Chahla J, Moatshe G, Faucett SC, Krych AJ, LaPrade RF. Meniscal root tears: a silent epidemic. Br J Sports Med 2018; 52: 872-876.
448. Smith PA, Bezold WA, Cook CR, Krych AJ, Stuart MJ, Wijdicks C, et al. Kinematic Analysis of Lateral Meniscal Oblique Radial Tears in the Anterior Cruciate Ligament-Deficient Knee. Am J Sports Med 2021; 49: 3898-3905.
449. Garcia JR, Ayala SG, Allende F, Mameri E, Haynes M, Familiari F, et al. Diagnosis and Treatment Strategies of Meniscus Root Tears: A Scoping Review. Orthop J Sports Med 2024; 12: 23259671241283962.
450. Ohori T, Mae T, Shino K, Fujie H, Hirose T, Tachibana Y, et al. Different effects of the lateral meniscus complete radial tear on the load distribution and transmission functions depending on the tear site. Knee Surg Sports Traumatol Arthrosc 2021; 29: 342-351.
451. Novaretti JV, Lian J, Patel NK, Chan CK, Cohen M, Musahl V, et al. Partial Lateral Meniscectomy Affects Knee Stability Even in Anterior Cruciate Ligament-Intact Knees. J Bone Joint Surg Am 2020; 102: 567-573.

452. Thorlund JB, Holsgaard-Larsen A, Creaby MW, Jorgensen GM, Nissen N, Englund M, et al. Changes in knee joint load indices from before to 12 months after arthroscopic partial meniscectomy: a prospective cohort study. Osteoarthritis Cartilage 2016; 24: 1153-1159.

Appendix

Supplemental Table 1. Incidence proportions of radiographic and symptomatic osteoarthritis
Bold indicates where the 95% CI does not overlap with the contralateral knee and italics where the 95% CI does not overlap with the group of knees with hemarthrosis alone and no structural injury.

		Injured knee			Contralateral knee		
Injury type, n (proportion, 95% CI	Sympt OA index	OA index	TF OA index	PF OA index	OA contra	TF OA contra	PF OA contra
Hemarthrosis alone – No structural injury	3/70 (4%, 1-12)	4/70 (6%, 2-14)	2/70 (3%, 0-10)	2/70 (3%, 0-10)	1/70 (1%, 0-9)	1/70 (1%, 0-9)	0/70 (0%, -)
LPD	16/124 (13%, 8-20)	20/124 (16%, 11-24)	15/124 (12%, 7-19)	19/124 (15%, 10-23)	14/124 (11%, 7-18)	10/124 (8%, 4-14)	10/124 (8%, *4-14*)
Other injury	10/67 (15%, 8-26)	19/67 (28%, *19-40*)	11/67 (16%, 9-27)	15/67 (22%, *14-34*)	14/67 (21%, *13-32*)	8/67 (12%, 6-22)	10/124 (8%, *4-14*)
Meniscus/not ACL	17/69 (24%, *16-36*)	27/70 (39%, *28-50*)	20/70 (29%, *19-40*)	22/70 (31%, *22-43*)	22/70 (31%, *22-43*)	16/70 (23%, *14-34*)	20/70 (29%, *19-40*)
ACL/not meniscus	35/196 (18%, *13-24*)	64/197 (32%, ***26-39***)	44/197 (22%, ***17-29***)	49/197 (25%, ***19-31***)	35/196 (18%, *13-24*)	21/196 (11%, 7-16)	25/196 (13%, *9-18*)
ACL and meniscus	74/254 (29%, *24-35*)	129/256 (50%, ***44-56***)	103/256 (40%, ***34-46***)	92/256 (36%, ***30-42***)	52/256 (20%, *16-26*)	33/256 (13%, 9-18)	44/256 (17%, *13-22*)
All injuries except Hemarthrosis alone	152/710 (21%, *18-25*)	259/714 (36%, ***33-40***)	193/714 (27%, ***24-30***)	197/714 (28%, ***24-31***)	137/713 (19%, *16-22*)	88/713 (12%, *10-15*)	113/713 (16%, *13-19*)
Meniscus and/or ACL	126/519 (24%, *21-28*)	220/523 (42%, ***38-46***)	167/523 (32%, ***28-36***)	163/523 (31%, ***27-35***)	109/522 (21%, *17-25*)	70/522 (13%, *11-17*)	89/522 17%, *14-21*)
All injuries	155/780 (20%, 17-23)	263/784 (34%, **30-37**)	195/784 (25%, **22-28**)	199/784 (25%, **22-29**)	138/783 (18%, 15-20)	89/783 (11%, 9-14)	113/783 (14%, 12-17)

Due to missing data, OA for ACL/not meniscus in the contralateral knee was calculated on 196 individuals. Symptomatic OA for Meniscus/not ACL was calculated on 69 individuals, for ACL/not meniscus on 196 individuals, and for ACL and meniscus on 254 individuals. OA, Osteoarthritis in any compartment. Symptomatic OA, osteoarthritis in combination with KOOS scores equivalent with symptoms. KOOS, knee injury and osteoarthritis outcome score. PF, patellofemoral. TF, tibiofemoral. Contra, contralateral knee. CI, confidence interval.

Supplemental Table 2. Risk ratios in a matched pair analysis of radiographic osteoarthritis with 95 % confidence intervals in the injured knee compared to the contralateral knee with adjustment for known previous injury before index trauma.

	Crude		After adjustment for earlier injury*		After adjustment for earlier or new injury*	
	RR	95% CI	RR	95% CI	RR	95% CI
	Any radiographic knee OA					
Injured knee	1.90	1.54, 2.33	1.85	1.45, 2.34	1.88	1.53, 2.32
Any structural injury	1.88	1.53, 2.32	1.83	1.44, 2.33	1.87	1.51, 2.31
Hemarthrosis alone#						
LPD	1.43	0.72, 2.83	1.37	0.66, 2.85	1.42	0.71, 2.82
Other injury	1.36	0.68, 2.71	1.35	0.62, 2.92	1.14	0.56, 2.32
Meniscus/not ACL	1.23	0.70, 2.15	1.14	0.58, 2.26	1.32	0.74, 2.34
ACL/not meniscus	1.80	1.19, 2.72	1.93	1.19, 3.14	1.89	1.24, 2.87
ACL and meniscus	2.48	1.80, 3.42	2.33	1.61, 3.38	2.42	1.74, 3.37
	Radiographic TF knee OA					
Injured knee	2.19	1.71, 2.82	2.35	1.73, 3.20	2.23	1.72, 2.90
Any structural injury	2.19	1.70, 2.82	2.40	1.75, 3.29	2.24	1.72, 2.91
Hemarthrosis alone#						
LPD	1.50	0.67, 3.34	1.83	0.76, 4.38	1.53	0.67, 3.45
Other injury	1.37	0.55, 3.42	1.44	0.51, 4.07	1.01	0.39, 2.64
Meniscus/not ACL	1.25	0.65, 2.41	1.23	0.55, 2.78	1.47	0.74, 2.91
ACL/not meniscus	2.10	1.25, 3.52	2.54	1.32, 4.88	2.21	1.29, 3.78
ACL and meniscus	3.12	2.11, 4.62	3.40	2.11, 5.49	3.21	2.13, 4.86
	Radiographic PF knee OA					
Injured knee	1.75	1.39, 2.21	1.64	1.2, 2.14	1.74	1.38, 2.21
Any structural injury	1.73	1.38, 2.19	1.61	1.23, 2.11	1.73	1.36, 2.19
Hemarthrosis alone#						
LPD	1.90	0.88, 4.09	1.71	0.77, 3.81	1.89	0.87, 4.08
Other injury	1.07	0.52, 2.22	1.02	0.45, 2.34	0.89	0.42, 1.89
Meniscus/not ACL	1.10	0.60, 2.02	0.95	0.45, 1.99	1.18	0.64, 2.19
ACL/not meniscus	1.92	1.18, 3.11	1.87	1.07, 3.29	2.07	1.26, 3.39
ACL and meniscus	2.09	1.46, 2.99	1.93	1.28, 2.93	2.03	1.40, 2.94

TF, tibiofemoral, PF, patellofemoral. OA, osteoarthritis, RR, risk ratio. CI, confidence interval. Injured knee, all knee injuries including hemarthrosis alone (no structural injury). Any structural injury, all injuries except hemarthrosis alone.

*Here only persons with non-missing data on earlier or new injury are included.

#Hemarthrosis alone not possible to estimate due to low counts, almost no OA in this group (n=4).

Supplemental Table 3. Between-persons analyses comparing the specific injury types to other structural injury than LPD, ACL injury and/or meniscus lesion. Estimates are risk ratios of OA in the index knee from a Poisson regression model with robust standard errors

Injury type	Adjusted for the logarithm of follow-up time, age, sex and injury at sports		Also adjusted for earlier or new injury to index knee		Also adjusted for BMI	
	Any radiographic knee OA					
	RR	95% CI	RR	95% CI	RR	95% CI
Other injury	Reference category					
Any structural injury vs other injury	1.44	1.00, 2.09	1.43	1.00, 2.03	1.50	1.06, 2.10
LPD	1.08	0.63, 1.86	0.99	0.57, 1.69	0.99	0.58, 1.67
Meniscus/not ACL	1.07	0.68, 1.68	1.10	0.71, 1.70	1.19	0.77, 1.82
ACL/not meniscus	1.23	0.82, 1.84	1.24	0.84, 1.83	1.30	0.89, 1.90
ACL and meniscus	1.86	1.28, 2.70	1.82	1.27, 2.61	1.90	1.34, 2.69
	Radiographic TF knee OA					
Other injury	Reference category					
Any structural injury vs other injury	1.88	1.08, 3.27	1.85	1.09, 3.15	1.97	1.20, 3.25
LPD	1.26	0.60, 2.65	1.11	0.53, 2.31	1.11	0.55, 2.26
Meniscus/not ACL	1.42	0.75, 2.69	1.48	0.81, 2.73	1.63	0.91, 2.92
ACL/not meniscus	1.46	0.80, 2.66	1.48	0.83, 2.62	1.60	0.93, 2.75
ACL and meniscus	2.57	1.47, 4.49	2.50	1.46, 4.27	2.67	1.61, 4.42
	Radiographic PF knee OA					
Other injury	Reference category					
Any structural injury vs other injury	1.41	0.93, 2.15	1.40	0.93, 2.10	1.50	1.03, 2.19
LPD	1.53	0.85, 2.75	1.41	0.79, 2.52	1.41	0.80, 2.47
Meniscus/not ACL	1.03	0.63, 1.70	1.05	0.65, 1.71	1.18	0.74, 1.88
ACL/not meniscus	1.22	0.77, 1.94	1.23	0.78, 1.93	1.32	0.87, 2.02
ACL and meniscus	1.70	1.10, 2.61	1.67	1.10, 2.55	1.78	1.20, 2.64
	Radiographic and symptomatic knee OA					
Other injury	Reference category					
Any structural injury vs other injury	1.66	0.93, 2.94	1.62	0.94, 2.77	1.77	1.07, 2.94
LPD	1.70	0.81, 3.57	1.45	0.70, 3.00	1.45	0.71, 2.99
Meniscus/not ACL	1.21	0.61, 2.39	1.28	0.68, 2.41	1.43	0.78, 2.62
ACL/not meniscus	1.37	0.73, 2.58	1.40	0.77, 2.52	1.54	0.88, 2.69
ACL and meniscus	2.04	1.14, 3.68	1.96	1.13, 3.41	2.15	1.28, 3.63

TF, tibiofemoral, PF, patellofemoral. OA, osteoarthritis, RR, risk ratio. CI, confidence interval. BMI, bone mass index. Any structural injury, all knee injuries except the reference category.

Supplemental Table 4a. Between-persons analysis of KOOS comparing the injury types. Mean differences from a tobit regression model adjusted for age, sex, if injury during sports participation and logarithm of follow-up time and thereafter additionally adjusted for earlier or new injury to index knee and BMI at follow-up.

PROM	Non missing (n)	Median [IQR]	Adjusted (points)	95% CI	Additionally adjusted (points)	95% CI
KOOS Symptom - all	812	86 [21]				
Hemarthrosis alone (ref)	72	89 [23]	0.00		0.00	
LPD	132	84 [21]	-3.75	-10.52, 3.03	1.17	-5.62, 7.96
Other injury	70	89 [25]	1.65	-5.74, 9.05	4.15	-3.11, 11.40
Meniscus/not ACL	73	86 [21]	-2.50	-9.77, 4.77	-1.75	-8.93, 5.44
ACL/not meniscus	199	93 [25]	0.06	-6.19, 6.30	1.04	-5.09, 7.16
ACL and meniscus	266	86 [21]	-4.74	-10.67, 1.20	-2.50	-8.35, 3.35
KOOS Pain - all	806	92 [22]				
Hemarthrosis alone (ref)	72	92 [21]	0.00		0.00	
LPD	131	86 [19]	-6.03	-13.15, 1.09	-0.75	-7.82, 6.31
Other injury	70	92 [19]	2.29	-5.53, 10.10	5.24	-2.38, 12.87
Meniscus/not ACL	73	94 [19]	1.02	-6.68, 8.72	2.57	-5.01, 10.14
ACL/not meniscus	197	94 [17]	1.80	-4.81, 8.41	3.06	-3.36, 9.47
ACL and meniscus	263	92 [22]	-1.63	-7.88, 4.62	0.89	-5.22, 7.00
KOOS ADL - all	811	97 [12]				
Hemarthrosis alone (ref)	72	96 [11]	0.00		0.00	
LPD	132	96 [14]	-3.78	-10.65, 3.09	1.20	-5.58, 7.97
Other injury	70	97 [10]	3.41	-4.08, 10.89	5.82	-1.43, 13.06
Meniscus/not ACL	73	96 [21]	-0.17	-7.56, 7.21	1.42	-5.82, 8.65
ACL/not meniscus	199	99 [10]	2.74	-3.63, 9.11	4.01	-2.15, 10.18
ACL and meniscus	265	97 [13]	1.20	-4.84, 7.24	3.60	-2.27, 9.46
KOOS Sport - all	809	75 [50]				
Hemarthrosis alone (ref)	72	80 [50]	0.00		0.00	
LPD	132	70 [50]	-13.30	-24.42, -2.18	-4.42	-15.35, 6.51
Other injury	70	80 [45]	4.52	-7.60, 16.64	9.26	-2.42, 20.95
Meniscus/not ACL	72	75 [38]	-3.18	-15.13, 8.77	-0.83	-12.43, 10.8
ACL/not meniscus	198	80 [40]	-0.72	-10.99, 9.55	1.49	-8.37, 11.35
ACL and meniscus	265	75 [45]	-7.98	-17.73, 1.76	-3.15	-12.58, 6.29

BMI, bone mass index. PROM, patient-reported outcome measure. KOOS, knee osteoarthritis and outcome score. ADL, activities of daily living. QoL, quality of life.

Supplemental Table 4b. Between-persons analysis of KOOS QoL, Tegner and Knee satisfaction score comparing the injury types. Mean differences from a tobit regression model adjusted for age, sex, if injury during sports participation and logarithm of follow-up time and thereafter additionally adjusted for earlier and new injury to index knee and BMI at follow-up.

PROM	Non missing (n)	Median [IQR]	Adjusted (points)	95% CI	Addition-ally adjusted (points)	95% CI
KOOS QoL - all	809	75 [38]				
Hemarthrosis alone (ref)	72	75 [38]	0.00		0.00	
LPD	132	59 [44]	-14.76	-23.76, -5.76	-8.15	-17.05, 0.75
Other injury	70	78 [44]	-0.43	-10.20, 9.34	2.45	-6.99, 11.90
Meniscus/not ACL	72	72 [31]	-5.57	-15.25, 4.11	-5.05	-14.49, 4.39
ACL/not meniscus	198	75 [38]	-3.88	-12.17, 4.41	-2.88	-10.88, 5.11
ACL and meniscus	265	69 [38]	-10.18	-18.06, -2.29	-7.37	-15.04, 0.30
Tegner Activity Scale						
Hemarthrosis alone (ref)	72	4 [4]	0.00		0.00	
LPD	131	3 [2]	-1.02	-1.65, - 0.39	-1.14	-1.79, -0.49
Other injury	70	3 [2]	-0.58	-1.26, 0.10	-0.56	-1.25, 0.12
Meniscus/not ACL	73	3 [3]	-0.44	-1.12, 0.23	-0.57	-1.25, 0.12
ACL/not meniscus	200	4 [4]	-0.27	-0.84, 0.31	-0.29	-0.87, 0.29
ACL and meniscus	264	4 [4]	-0.50	-1.05, 0.05	-0.53	-1.09, 0.02
Knee satisfaction score						
Hemarthrosis alone (ref)	72	8 [3]	0.00		0.00	
LPD	132	7 [3]	-0.90	-1.75, -0.05	-0.22	-1.06, 0.63
Other injury	69	8 [3]	-0.09	-1.01, 0.84	0.28	-0.62, 1.18
Meniscus/not ACL	73	8 [3]	-0.21	-1.13, 0.71	-0.04	-0.94, 0.86
ACL/not meniscus	200	8 [2]	-0.08	-0.86, 0.70	0.10	-0.67, 0.86
ACL+meniscus	261	8 [3]	-0.51	-1.26, 0.23	-0.17	-0.90, 0.56

BMI, bone mass index. PROM, patient-reported outcome measure. Tegner, Tegner activity scale. Knee satisfaction score, “Are you satisfied with your current knee function?” on a 10-point Likert scale from 1 “Not satisfied at all” to 10 “Very satisfied”. CI, confidence interval.