Loss of patellofemoral cartilage thickness over 5 years following ACL injury depends on the initial treatment strategy: results from the KANON trial

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ABSTRACT
Objectives To evaluate changes in patellofemoral cartilage thickness over 5 years after anterior cruciate ligament (ACL) injury and to determine the impact of treatment strategy.

Methods 121 adults (ages 18–35 years, 26% women) had an ACL injury and participated in the KANON randomised controlled trial. Of those, 117 had available MRIs at baseline (<4 weeks post-ACL rupture) and at least one follow-up measurement (2, 5 years). Patellofemoral cartilage thickness was analysed by manual segmentation (blinded to acquisition order). Patellar, trochlear and total patellofemoral cartilage thickness changes were compared between as-randomised (rehabilitation+early ACL reconstruction (ACLR) (n=59) vs rehabilitation+optional delayed ACLR (n=58)) and as-treated groups (rehabilitation+early ACLR (n=59) vs rehabilitation+delayed ACLR (n=29) vs rehabilitation alone (n=29)).

Results Patellofemoral cartilage thickness decreased −58 µm (95% CI −104 to −11 µm) over 5 years post-ACL rupture, with the greatest loss observed in trochlea during the first 2 years. Participants randomised to rehabilitation+early ACLR had significantly greater loss of patellar cartilage thickness compared with participants randomised to rehabilitation+optional delayed ACLR over the first 2 years (−25 µm (−52, 1 µm) vs +14 µm (−6 to 34 µm), p=0.02) as well as over 5 years (−36 µm (−78 to 5 µm) vs +18 µm (−7, 42 µm), p=0.02). There were no statistically significant differences in patellofemoral cartilage thickness changes between as-treated groups.

Conclusion Patellofemoral (particularly trochlear) cartilage thickness loss was observed in young adults following acute ACL rupture. Early ACLR was associated with greater patellofemoral cartilage thickness loss compared with optional delayed ACLR. Grant type (ie, hamstring-tendon vs bone-patellar tendon-bone (BPTB)) on patellofemoral cartilage deterioration compared with optional delayed surgery.

Trial registration number ISRCTN84752559; Post-results.

INTRODUCTION
Anterior cruciate ligament (ACL) injury in adolescents and young adults is associated with a higher risk of incident knee osteoarthritis (OA) at a relatively young age.1,2 Although typically recognised as a disease of the tibiofemoral joint, radiographic OA affects the patellofemoral joint in approximately half of all patients ≥10 years following ACL injury, irrespective of surgical management or not.3 Indeed, patellofemoral OA may be more common than tibiofemoral OA after ACL injury.4,5 and is associated with worse symptoms and deterioration in quality of life.4

The precise mechanism contributing to the development of radiographic patellofemoral OA after ACL injury is not well understood, but may relate to the rapid patellofemoral cartilage degeneration observed immediately following ACL injury and reconstruction (ACLR). Within the first year following ACLR, approximately half of all young adults have a patellofemoral cartilage lesion on MRI,6 while approximately half of all knees with normal cartilage at ACLR develop cartilage lesions on second-look arthroscopy after 18 months, mostly in the patellofemoral joint.7,8 Potter et al observed no statistically significant differences in worsening patellar cartilage lesions on MRI 7–11 years following ACL injury managed non-operatively compared with surgically,9 however, data from adequately designed randomised controlled trials (RCTs) are lacking.

In the only available high-quality RCT designed to study whether structured rehabilitation combined with early ACLR is superior to structured rehabilitation with optional delayed ACLR, the KANON trial failed to identify treatment-related differences in clinical outcomes, incident radiographic OA or meniscal surgeries over 2 and 5 years.10,11 A subset of 63 included patients of this study was suggested to display loss of femoral trochlear cartilage within the first 2 years post-injury, more so than any other joint surface.12,13 While longer-term tibiofemoral cartilage thickness changes (over 5 years) have been reported from the full KANON cohort,13 the cartilage changes in the patellofemoral joint as well as the influence of treatment of the ruptured ACL have not yet been reported. Similarly, the influence of graft type (ie, hamstring-tendon vs bone-patellar tendon-bone (BPTB)) on patellofemoral cartilage for those with an ACLR has not been analysed, but may be important for informing graft choice and efforts to minimise the burden of post-traumatic patellofemoral OA.

Using data from the KANON trial, we therefore aimed to evaluate the long-term (5 year) changes in patellofemoral cartilage thickness in young active adults with acute ACL rupture and...
to compare these changes between treatment groups as-randomised (full analysis set) and as-treated. We also aimed to explore the influence of graft choice on patellofemoral cartilage thickness changes in knees that underwent ACLR.

**MATERIALS AND METHODS**

**Study design and participants**

The KANON RCT (ISRCTN84752559) enrolled 121 young active adults aged 18–35 years with an acute ACL rupture to a previously uninjured knee. Major exclusion criteria were total collateral ligament rupture and a full-thickness cartilage lesion on MRI. Sixty-two participants were randomised to a structured exercise programme plus early ACLR (defined as ‘early ACLR’ group) and 59 to a similar structured exercise programme, with the option of having a delayed ACLR if needed (prespecified criteria: symptomatic instability caused by ACL insufficiency and positive pivot shift test) or if requested by the patient. The study was approved by the Lund University ethics committee, participants provided informed consent and clinical results after 2 years (n=121) and 5 years (n=120) have been published.

Early ACLR was performed within 10 weeks of injury using an ipsilateral hamstring-tendon (n=36) or BPTB autograft (n=25) based on surgeon preference (one participant declined to undergo ACLR after randomisation but was excluded from current analyses due to missing follow-up MRIs). From the initial 121 KANON participants, 30 (51%) of the 59 participants randomised to rehabilitation plus optional delayed ACLR underwent delayed surgery over 5 years: 23 within the first 2 years (13 BPTB, 10 hamstring-tendon), seven in the following 3 years (2 BPTB, 5 hamstring-tendon). Meniscal injuries observed on baseline MRI (n=69, 57%) were of similar prevalence in all treatment groups and were treated with partial resection or fixation when clinically indicated.

All participants performed the same goal-oriented, progressive physiotherapist supervised exercise-therapy programme that commenced before or at the time of randomisation (and therefore included the preoperative period for those undergoing ACLR). The programme included four levels of function—progression was guided by goals for range of motion, muscle function and functional performance. Pain, swelling and general discomfort slowed the progression. The programme continued for ≥4 months until the final goals were reached (see Frobell et al for details).

**MRI acquisition and analysis**

Baseline MRIs were acquired within 4 weeks of ACL rupture, using a 1.5T Gyroscan Intera magnet (Philips) and a circular polarised surface coil. A sagittal three-dimensional gradient-echo sequence (repetition time 20 ms, echo time 7.9 ms, flip angle 25°) that has previously been validated for the purposes of quantitative cartilage analysis was obtained with a 3.0 mm slice thickness, 1.5 mm slice gap and a 0.29 mm in-plane resolution (field-of-view 15 cm, matrix 512×512 pixels). Identical sequences were acquired at 2-year (n=112) and 5-year follow-up (n=112). A total of 107 participants had both 2-year and 5-year follow-up images.

All available knee image datasets for each participant were processed together, with blinding of the readers to the acquisition time-point, and were quality controlled by an expert reader. The subchondral bone plate area and the cartilage surface area were segmented manually in each image showing the patella and femoral trochlea as previously described. Briefly, the trochlea was separated from the femoral condyles by a 3D plane through the trochlear notch in parallel with the femoral shaft and parallel to the posterior ends of both (the medial and lateral) femoral condyles (figure 1). The mean cartilage thickness over the subchondral bone was computed in each cartilage plate in 3D, independent of the original section orientation, using the bone interface and cartilage surface segmentations (figure 1). Precision errors (root mean square SD and coefficient of variation) for patellar and trochlear cartilage thickness in subjects with and without OA using similar techniques were found to be 53–70 µm (2.3%–2.6%). These precision errors are similar to the smallest detectable change threshold at 95% CI for patellofemoral cartilage thickness (66 µm) calculated from the current cohort using the formula: 1.96×√2×SE of measurement.

**Statistical analysis**

Patellar, trochlear and patellofemoral (patellar and trochlear cartilage thickness summed) changes were computed as absolute values (µm) from baseline to year 5, with the early (baseline to year 2) and later (year 2 to year 5) time periods also reported. Differences in cartilage thickness changes between as-randomised groups, as-treated groups and graft type (performed only on those who underwent ACLR), were evaluated for each of the follow-up periods using linear mixed-effects models with unstructured covariance matrices. Presented p values represent the interaction between fixed-effects of group and follow-up.

**Figure 1** Assessment of patellar and trochlear cartilage thickness segmentation: (A) three-dimensional gradient-echo sequence; (B) patellar (magenta) and trochlear (aqua) cartilage thickness, with the trochlea separated from the femoral condyles by 3D plane through the trochlear notch in parallel with the femoral shaft (purple line).
periods, adjusting for age and sex, which have been shown to influence patellofemoral cartilage thickness change. The number of individuals with total patellofemoral cartilage loss over the entire 5-year observation period exceeding the smallest detectable change (66 µm) was also calculated. All statistical analyses were completed using Stata V.14.2 (Stata, Texas, USA). P < 0.05 were considered statistically significant.

RESULTS

Baseline demographic and cartilage thickness data of all participants with baseline and at least one follow-up MRI (n = 117) are presented in table 1. For the entire cohort, patellofemoral cartilage thickness decreased by a mean −58 µm (1.2%) (95% CI −104 to −11 µm) over the 5-year period (equivalent to 0.2% annual loss), with the greatest cartilage loss observed in the trochlea during the first 2 years (1.0% annual loss) (table 2). Forty-five (40%) participants displayed total patellofemoral cartilage loss over 5 years that exceeded the smallest detectable change threshold.

In the as-randomised analysis, the early ACLR group was observed to encounter a greater loss of patellofemoral cartilage thickness compared with the optional delayed ACLR group during all follow-up periods, with statistically significant differences observed in the patella over the first 2 years and over the entire 5-year observation period (table 2). On average, the early ACLR group displayed a loss of patellofemoral cartilage thickness over 5 years. This loss was most prominent in the trochlea, where a decrease of −72 µm (3.1%) (95% CI −118 to −26) was found over 5 years, equivalent to 0.6% annual loss, whereas in the patella, a decrease of −36 µm (1.3%) (95% CI −78 to 5) was observed. Cartilage thickness changes were typically smaller between years 2 and 5 than during the first 2 years, without any significant changes within any of the as-randomised groups or significant differences between groups observed. Twenty-nine

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**Table 1** Baseline characteristics of the 117 participants with baseline MRI split by treatment group and ACLR graft type at 5 years*

<table>
<thead>
<tr>
<th>Outcome (µm)</th>
<th>Total cohort</th>
<th>Early ACLR</th>
<th>Optional delayed ACLR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>All subjects (n=59)</td>
<td>All subjects (n=58)†</td>
<td>Delayed ACLR† (n=29)</td>
<td>Rehabilitation alone (n=29)†</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>12 (20)</td>
<td>20 (34)</td>
<td>11 (38)</td>
<td>9 (31)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.4±3.2</td>
<td>23.8 (2.6)</td>
<td>23.3±2.0</td>
<td>24.3±3.1</td>
</tr>
<tr>
<td>Days to ACLR</td>
<td>44±12</td>
<td>NA</td>
<td>559±420</td>
<td>NA</td>
</tr>
<tr>
<td>Year 2/5 follow-up missing MRI, n (%)</td>
<td>1 (2)/0 (0)</td>
<td>4 (7)/5 (9)</td>
<td>2 (7)/2 (7)</td>
<td>2 (7)/3 (10)</td>
</tr>
<tr>
<td>Cartilage thickness, mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total PF joint</td>
<td>5.0±0.5</td>
<td>4.8±0.6</td>
<td>4.7±0.6</td>
<td>4.8±0.6</td>
</tr>
</tbody>
</table>

*Values are mean±SD unless indicated otherwise. No statistically significant baseline characteristic differences exist between as-treated groups (p>0.05). †BMI missing for one participant from rehabilitation alone group.

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**Table 2** Patellofemoral cartilage thickness changes over the total (0–5 years), early (0–2 years) and late (2–5 years) follow-up periods: total cohort and as-randomised*

<table>
<thead>
<tr>
<th>Outcome (µm)</th>
<th>Total cohort</th>
<th>Early ACLR</th>
<th>Optional delayed ACLR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in cartilage thickness between baseline and 5-year follow-up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Participants included in analysis</td>
<td>n=112</td>
<td>n=59</td>
<td>n=53</td>
<td></td>
</tr>
<tr>
<td>Patella</td>
<td>−11 (−36 to 14)</td>
<td>−36 (−78 to 5)</td>
<td>18 (−7 to 42)</td>
<td>0.022</td>
</tr>
<tr>
<td>Trochlea</td>
<td>−47 (−73 to −20)</td>
<td>−72 (−118 to −26)</td>
<td>−19 (−43 to 5)</td>
<td>0.058</td>
</tr>
<tr>
<td>Total patellofemoral joint</td>
<td>−58 (−104 to −11)</td>
<td>−108 (−189 to −27)</td>
<td>−1 (−40 to 37)</td>
<td>0.021</td>
</tr>
<tr>
<td>Change in cartilage thickness between baseline and 2-year follow-up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Participants included in analysis</td>
<td>n=112</td>
<td>n=58</td>
<td>n=54</td>
<td></td>
</tr>
<tr>
<td>Patella</td>
<td>−7 (−23 to 10)</td>
<td>−25 (−52 to 1)</td>
<td>14 (−6 to 34)</td>
<td>0.018</td>
</tr>
<tr>
<td>Trochlea</td>
<td>−45 (−66 to −24)</td>
<td>−63 (−100 to −26)</td>
<td>−26 (−467 to −5)</td>
<td>0.074</td>
</tr>
<tr>
<td>Total patellofemoral joint</td>
<td>−52 (−85 to −18)</td>
<td>−88 (−145 to −31)</td>
<td>−12 (−44 to 20)</td>
<td>0.020</td>
</tr>
<tr>
<td>Change in cartilage thickness between 2-year and 5-year follow-up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Participants included in analysis</td>
<td>n=107</td>
<td>n=58</td>
<td>n=49</td>
<td></td>
</tr>
<tr>
<td>Patella</td>
<td>−4 (−22 to 15)</td>
<td>−13 (−39 to 13)</td>
<td>7 (−19 to 33)</td>
<td>0.306</td>
</tr>
<tr>
<td>Trochlea</td>
<td>−1 (−16 to 14)</td>
<td>−9 (−30 to 12)</td>
<td>8 (−12 to 29)</td>
<td>0.265</td>
</tr>
<tr>
<td>Total patellofemoral joint</td>
<td>−5 (−34 to 24)</td>
<td>−22 (−64 to 20)</td>
<td>15 (−24 to 55)</td>
<td>0.227</td>
</tr>
</tbody>
</table>

P values calculated from linear mixed effects model (ie, the interaction between fixed-effects of group and follow-up periods, adjusted for age and sex).

*Changes in cartilage thickness (µm) are mean and 95% CI.

ACLR, anterior cruciate ligament reconstruction.
(49%) and 16 (30%) participants had total patellofemoral cartilage loss that exceeded smallest detectable change in the early ACLR group and optional delayed ACLR group, respectively.

In the as-treated analysis, no statistically significant difference was observed between any of the three groups during any follow-up period (Table 3). This was despite eight participants in both rehabilitation alone and delayed ACLR groups (31% and 30%, respectively) displaying total patellofemoral cartilage loss exceeding smallest detectable change compared with 29 (49%) participants in the early ACLR group. Similarly, sensitivity analyses adjusting for graft type resulted in similar observations and does not exceed smallest detectable change compared with 29 (49%) and 16 (30%) participants had total patellofemoral cartilage loss that exceeded smallest detectable change in the early ACLR group and optional delayed ACLR group, respectively.

In the as-treated analysis, no statistically significant difference was observed between any of the three groups during any follow-up period (table 3). This was despite eight participants in both rehabilitation alone and delayed ACLR groups (31% and 30%, respectively) displaying total patellofemoral cartilage loss exceeding smallest detectable change compared with 29 (49%) participants in the early ACLR group. Similarly, sensitivity analyses adjusting for graft type resulted in similar observations and 5-year patellofemoral cartilage thickness changes did not significantly differ between all knees treated with ACLR (early and delayed) at 5 years and those treated with rehabilitation alone (data not shown).

In the analysis of graft type, performed in all knees that underwent ACLR over the 5-year period, we observed numerically greater patellofemoral cartilage thickness loss in knees treated with BPTB autografts than hamstring autografts for all follow-up periods although there was no statistically significant difference between the two groups (Table 4).

**Discussion**

This is the first study to report cartilage thickness loss after ACL injury, depending on the randomised choice of treatment.
strategy. The prospective randomised controlled KANON trial of young adults had only minimal loss to follow-up and compared a strategy of rehabilitation plus optional surgical ACLR to early ACLR. Early ACLR resulted in significantly greater loss of patellofemoral cartilage thickness over 5 years compared with a strategy of rehabilitation with delayed optional ACLR. Post-ACLR injury cartilage thickness loss was most pronounced in the trochlea and more prominent in the immediate 2-year interval after injury. We found no significant difference in patellofemoral cartilage thickness change between those treated with rehabilitation alone, early ACLR or delayed ACLR, most likely due to the small sample size of the latter two groups. Further, graft choice in knees with an ACLR did not appear to significantly influence patellofemoral cartilage change after ACL injury.

The focus of longitudinal structural changes following ACL injury has previously been on the tibiofemoral joint. Recently, an overall annual 0.4% tibiofemoral cartilage thickening was reported for the full KANON cohort, which is in contrast to our findings of 0.2% annual patellofemoral and 0.6% trochlear cartilage thinning in the same sample. These patellofemoral cartilage findings over 5 years extend data on the short-term cartilage change in a subset of the KANON cohort that did not consider the impact of treatment type and reinforce the alarmingly rapid (1%/2% per year) cartilage loss in the trochlea during the first 2 years following ACL injury. Although the patellofemoral cartilage thickness loss seemed to occur more slowly during the later (2–5-year) interval, the annual loss of patellofemoral cartilage over 5 years was similar to that of knees with established OA and is in stark contrast to small gains in patellofemoral cartilage thickness over 2 years in healthy younger (16–18 years old) athletes using the same cartilage analysis technology. Notably, 45 (40%) participants displayed total patellofemoral cartilage loss over 5 years that exceeded the smallest detectable change threshold (66 µm). Deterioration of patellofemoral cartilage after ACL injury was reported to be prognostic for worse patient-reported symptoms and function and may be linked to observations of more prevalent radiographic OA in the patellofemoral joint compared with the tibiofemoral joint over longer-term follow-up.

Although cartilage thickness loss occurred most prominently in the trochlea in all treatment groups, early ACLR appears to have a more deleterious effect on patellar cartilage compared with optional delayed ACLR (that is rehabilitation first, and ACLR only if needed or requested by the patient), particularly over the first 2 years. The mean difference in trochlear cartilage thickness loss between the two as-randomised groups was of similar magnitude to the patella, yet in the trochlea these differences did not reach statistical significance (p=0.058 and 0.074). The mechanisms behind the differences in cartilage loss between treatment strategies are not known, but may relate to the prolonged inflammatory response as a consequence of surgically induced trauma. This may be particularly pertinent when in close temporal context of the first trauma (ACL rupture), which may be associated with damage to cartilage type II collagen network and important cartilage proteins. Prolonged inflammation in the setting of progressive loading during postoperative rehabilitation, and possibly during early phases of return to sports, may be particularly pertinent to patellar cartilage, the only joint cartilage in the knee exhibiting a dose-dependent response of greater deformation with more intense loading. Distinct biomechanical features in ACLR knees and prolonged underloading of the knee following ACLR, which have been linked to patellofemoral cartilage changes and an elevated risk of postoperative radiographic OA, may also contribute to early ACLR knees displaying greater patellofemoral cartilage loss. Postoperative quadriceps muscle inhibition and prolonged weakness following early ACLR may also be detrimental for patellofemoral cartilage, as it increases the risk of patellofemoral cartilage defect progression in older patients with OA. However, no differences in muscle strength were observed between early versus optional delayed ACLR groups at the completion of rehabilitation in the KANON trial.

In as-treated analyses, patellofemoral cartilage thickness in knees treated with either rehabilitation alone or rehabilitation plus delayed ACLR remained relatively stable over the 5-year period (both group means <1%/ loss; 8 (31%) and 8 (30%) participants in rehabilitation alone and delayed ACLR groups, respectively, had total patellofemoral cartilage loss that exceeded smallest detectable change). This was in contrast to a somewhat larger loss of patellar and trochlear cartilage thickness in the early ACLR group (group mean >3%/ loss; 29 (49%) participants had total patellofemoral cartilage loss that exceeded smallest detectable change). However, these differences failed to reach statistical significance, likely due to the small sample sizes of the rehabilitation only and rehabilitation plus delayed ACLR groups and the limited statistical power. In our sensitivity analysis, we also observed no statistically significant differences in cartilage thickness changes between all ACLRs (early and delayed) versus no ACLR at 5 years, likely due to the similar amount of cartilage thickness loss in delayed ACLR and rehabilitation only groups and statistical power. It is important to note that longer-term follow-up evaluations are needed to reveal whether cartilage loss in those with an early ACLR is linked to more frequent end-stage joint disease compared with delayed ACLR or rehabilitation alone.

Although we observed slightly greater patellofemoral cartilage thickness loss in knees treated with BPTB autografts than hamstring autografts for all follow-up periods in those treated with (early or delayed) ACLR, these differences did not reach statistical significance. No minimal clinical important difference threshold currently exists for cartilage loss given the complex non-linear and multifactorial relationship between structural pathology and pain. However, taken together with second-look arthroscopy data showing rapid patellofemoral cartilage deterioration following hamstring-tendon autograft, our findings suggest that recommending a hamstring-tendon autograft on the basis that it will avert the risk of patellofemoral cartilage loss may be misguided. The trend we observed for greater patellar cartilage loss in those with a BPTB autograft in the later follow-up period (ie, 2–5 years) may be related to these participants having more frequent radiographic changes in the patellofemoral joint compared with knees with a hamstring-tendon autograft. These more obvious differences in the later follow-up period potentially reflect a slow loss of cartilage related to altered mechanical loading due to postoperative patellar tendon shortening, which was shown to be associated with the severity of patellofemoral radiographic changes. Other long-term follow-up studies have also observed slightly higher rates of radiographic patellofemoral OA (although not statistically significant) after BPTB (30%–41%) compared with hamstring-tendon autografts (16%–30%).

The KANON-trial was designed to investigate differences in clinical outcome as primary endpoint. Consequently, the sample size was not calculated to study differences in cartilage thickness, which may have resulted in a lack of sufficient power to detect statistically significant difference in as-treated and ACLR autograft analyses. Based on the observed baseline to year 5 cartilage thickness changes, approximately 100 participants per group would be required to achieve 80% power for as-treated analysis and approximately 200 participants per group for the graft type analysis. Nevertheless, the similarly rapid trochlear cartilage thinning (2%/year annually) observed in a subset (n=61) of the KANON cohort using Br J Sports Med: first published as 10.1136/bjsports-2018-100167 on 8 February 2019. Downloaded from http://bjsm.bmj.com/ on June 24, 2020 at Serials Division La Trobe University.
different imaging analysis techniques (during the first 2 years only and not analysed based on randomised or as-treated groups) increases the external validity of our overall findings. The sample was also somewhat heterogeneous because the status of impairments (eg, effusion, pain, quadriceps strength) at surgery was not uniformly documented. The identification of young adults who display accelerated cartilage thickness loss during the early time period following ACL injury creates the ideal platform to develop and test secondary OA prevention strategies targeted to this particularly high risk group.

In conclusion, patellofemoral, especially trochlear, cartilage thickness loss was observed in young adults following acute ACL rupture, particularly during the first 2 years after injury. A strategy of rehabilitation plus early ACLR was associated with significantly greater loss of patellofemoral (particularly patellar) cartilage thickness over 5 years compared with a strategy of rehabilitation with optional delayed ACLR, indicating that early surgical intervention may be associated with greater short-term structural patellofemoral cartilage deterioration compared with optional delayed surgery.

What are the findings?

- Undergoing early reconstruction resulted in significantly greater patellofemoral cartilage loss within the first 5 years after anterior cruciate ligament injury compared with a strategy of optional delayed reconstruction.
- Surgical graft choice did not influence patellofemoral cartilage loss.

How might it impact on clinical practice in the future?

- Early reconstruction is unable to prevent structural deterioration in the patellofemoral joint after anterior cruciate ligament injury—from the viewpoint of patellofemoral pathology, early reconstruction is not indicated.

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Contributors

All authors contributed to the conception and design of the study. LSL, RF, FE and WW contributed to the acquisition of the data. All authors contributed to the analysis and interpretation of the study, revised the manuscript critically for important intellectual content and approved the final version of the manuscript.

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Disclaimer

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Competing interests

FE is CEO of Chondrometrics GmbH, a company providing MR image analysis services to academic researchers and to industry. He has provided consulting services to EMD Serono, Bioclinical/Synarc, Samumed, Servier, Kolen-Tissuergene, Roche and Galapagos, has prepared educational sessions for Medtronic and has received research support from Pfizer, Eli Lilly, Merck Serono, Novartis, Stryker, Abbvie, Kolen, Synarc, Ampio, BICL, Orthotrophix, Kolen-Tissue Gene, Servier and Galapagos. WW has a part time employment with Chondrometrics GmbH and is a co-owner of Chondrometrics GmbH.

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REFERENCES


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