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An Age-Aware, Mechanics-First Framework for Focal Cartilage Defects in Young Active Patients: Prioritising Joint Preservation over Technique Selection

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Abstract. In active patients aged between 18 and 40, a focal knee cartilage defect is primarily a problem of decades-long joint preservation. However, the longest randomised follow-up covers only about 15 years, far short of these patients' remaining joint lifespan. Current pathways select treatment mainly according to lesion size and depth. We propose, as a hypothesis-level decision framework, an age-aware, mechanics-first hierarchy comprising three interacting layers: Layer 1, mechanical prerequisites (limb alignment, meniscal integrity, ligamentous stability, and the opposing surface); Layer 2, rehabilitation feasibility; and Layer 3, technique selection according to lesion morphology, spanning microfracture, AMIC, OATS, MACI and OCA. One direct signal, the correlation between limb alignment and autologous chondrocyte implantation graft survival, together with convergent but indirect evidence, suggests that long-term durability may depend more on the mechanical environment than on a technique's cellular complexity. It is concluded that this is a testable hypothesis rather than an established fact. The supporting comparisons are drawn across heterogeneous cohorts, and the rehabilitation-capacity prompt is unvalidated. The framework's contribution is to move the determinants of joint-preservation upstream of technique choice and to motivate trials with follow-up durations that match these patients' remaining joint lifespan.

Keywords: Articular cartilage repair, Young active patients, Limb alignment, Rehabilitation feasibility

1. Introduction

In skeletally mature patients aged between 18 and 40 with high activity demands, a focal knee cartilage defect is a fundamental problem of long-term joint preservation rather than a localised symptomatic complaint. Total knee arthroplasty is normally not a first-line option because implant longevity is limited and the revision burden remains high in young and high-demand patients. Articular cartilage is avascular and aneural with very limited intrinsic repair capacity. Once injured, cellular stress, inflammatory signalling, and abnormal loading drive regenerative failure [1]. In a population-based cohort, a prior knee injury in young adults was associated with an approximately

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sixfold higher adjusted risk of subsequent symptomatic osteoarthritis (adjusted HR 5.7) [2]. This finding establishes the focal defect as an initiating event for osteoarthritis that unfolds over decades.

Current practice selects treatments according to lesion size, depth, and chondral or osteochondral status [3]. For this population, however, the evidence horizon is itself the central tension. The longest randomised follow-up reaches only about 15 years [4], whereas these patients have 40 to 60 years of remaining joint lifespan. Even at age 40, it is about three times the longest trial horizon. Short- to medium-term superiority on patient-reported outcome measures (PROMs) cannot be extrapolated to decades-long preservation. Existing long-term cohorts further compound this by skewing toward the early 30s: Solheim and Volz enrolled participants aged 18 to 50, with Solheim reporting a median age of 32 [5, 6]. This leaves the 18-to-30 subgroup, where decades of preservation matter most, with essentially no direct long-term evidence.

Given this temporal mismatch, cartilage repair in young active patients is reframed as a three-layer, age-aware, mechanics-first decision hierarchy, presented as a hypothesis to be tested. The mechanical environment (Layer 1) and rehabilitation feasibility (Layer 2) sit upstream, while technique selection (Layer 3) is nested beneath them and modulated by lesion morphology.

2. Why technique selection alone is insufficient

2.1. The biological window

A focal defect does not remain static. Within weeks, damage-associated molecular patterns released from the disrupted matrix and apoptotic cells engage pattern-recognition receptors on synoviocytes, macrophages, and chondrocytes. This engagement sustains pro-inflammatory cytokine and matrix-metalloproteinase secretion and subsequently establishes a self-perpetuating catabolic loop, reinforced by abnormal loading [1]. A biological window therefore exists. Early intervention matters primarily because it interrupts this loop, with symptom relief a secondary consequence.

2.2. The surgical technique landscape

Five mainstream techniques span the spectrum from marrow stimulation to structural replacement, namely microfracture, AMIC (marrow stimulation augmented with a type I/III collagen membrane), OATS or mosaicplasty, MACI and OCA. Table 1 summarises each technique's lesion profile within this framework, the longest-term evidence with its time horizon and endpoint type, and the principal limitation or uncertainty in young active patients. Because these endpoints differ in kind, PROM trajectory versus graft or joint survival, the techniques cannot be ranked on a single common scale.

Table 1. Five principal cartilage repair techniques

Technique	Lesion profile (this framework)	Longest-term evidence: horizon and endpoint	Principal limitation / uncertainty in young active patients
Microfracture	Small contained defects (< 2 cm²); fallback only, durability-limited	Horizon 10 y. PROM trajectory: early gain then decline vs AMIC by 10 y (RCT, n = 47) [6]	Fibrocartilage of limited durability over decades [1]
AMIC	Contained defects up to ≈ 5 cm²; donor-sparing alternative	Horizon 10 y. PROM trajectory: stable to 10 y while microfracture declines (RCT, n = 47) [6]	Thinner long-term evidence base than ACI/MACI

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Table 1. (continued)

OATS / mosaicplasty	Small–medium contained defects (2–5 cm^2); structural repair	Horizon 15–17 y. PROM trajectory: Lysholm advantage over microfracture sustained (RCT, n = 40) [5]	Donor-site morbidity; upper size limit
MACI	Larger full-thickness chondral defects (≥ 3 cm^2)	Horizon min 10 y (mean 12.9 y). Survival: 9.0% reoperation, 7.4% conversion to TKA (systematic review, LoE 4) [7]	Two-stage; high cost; centralised service
OCA	Osteochondral defects, bone loss, or failed prior repair	Survival: neutral-alignment benefit significant for ACI but not OCA in the only alignment study (case series, LoE 4, n = 168) [8]	Donor availability; limited OCA-specific long-term data in young patients

Note: Endpoints differ in kind (PROM trajectory versus graft or joint survival) and are not directly comparable across rows. PROM, patient-reported outcome measure; LoE, level of evidence; TKA, total knee arthroplasty.

2.3. Two structural limitations

There are two structural limitations. First, the mechanical mismatch exists. Native hyaline cartilage shows load-dependent biphasic viscoelastic behaviour, with a Young's modulus of about 1.0 MPa and an instantaneous modulus of about 52 MPa under indentation [9]. The fibrocartilage produced by marrow stimulation has a disorganised matrix that does not reproduce these properties [1]. Thus, it fills a defect geometrically, but does not restore load-bearing function.

Second, there is a temporal convergence. A 15-year RCT (n = 80) found no significant difference between autologous chondrocyte implantation (ACI) and microfracture in PROMs, failure rates (42.5% vs 32.5%; p = 0.356), or radiographic osteoarthritis [4]. A network meta-analysis of 19 RCTs found no difference in PROMs among microfracture, AMIC, OATS, and ACI at any timepoint [10]. The absence of inter-technique differences weakens a strongly technique-centred view, but does not by itself establish a positive role for the mechanical environment. This role rests on independent positive evidence, such as the alignment–survival association in Section 3.1.

3. A three-layer mechanics-first framework

The three layers interact rather than form rigid gates, although the mechanical environment and rehabilitation capacity represent the dominant, upstream decisions (Figure 1). Whenever an upstream layer is unmet, the Layer 3 intervention is deferred, downgraded, or replaced by a non-operative option. This is a point of diagnostic deferral that a size-based pathway lacks. Age operates across all three layers rather than as a separate step. Because remaining joint lifespan increases as age falls, an early-declining repair costs the youngest patients the most. With decreasing age, the framework favours fuller correction of the mechanical environment and the most durable reconstruction the lesion permits.

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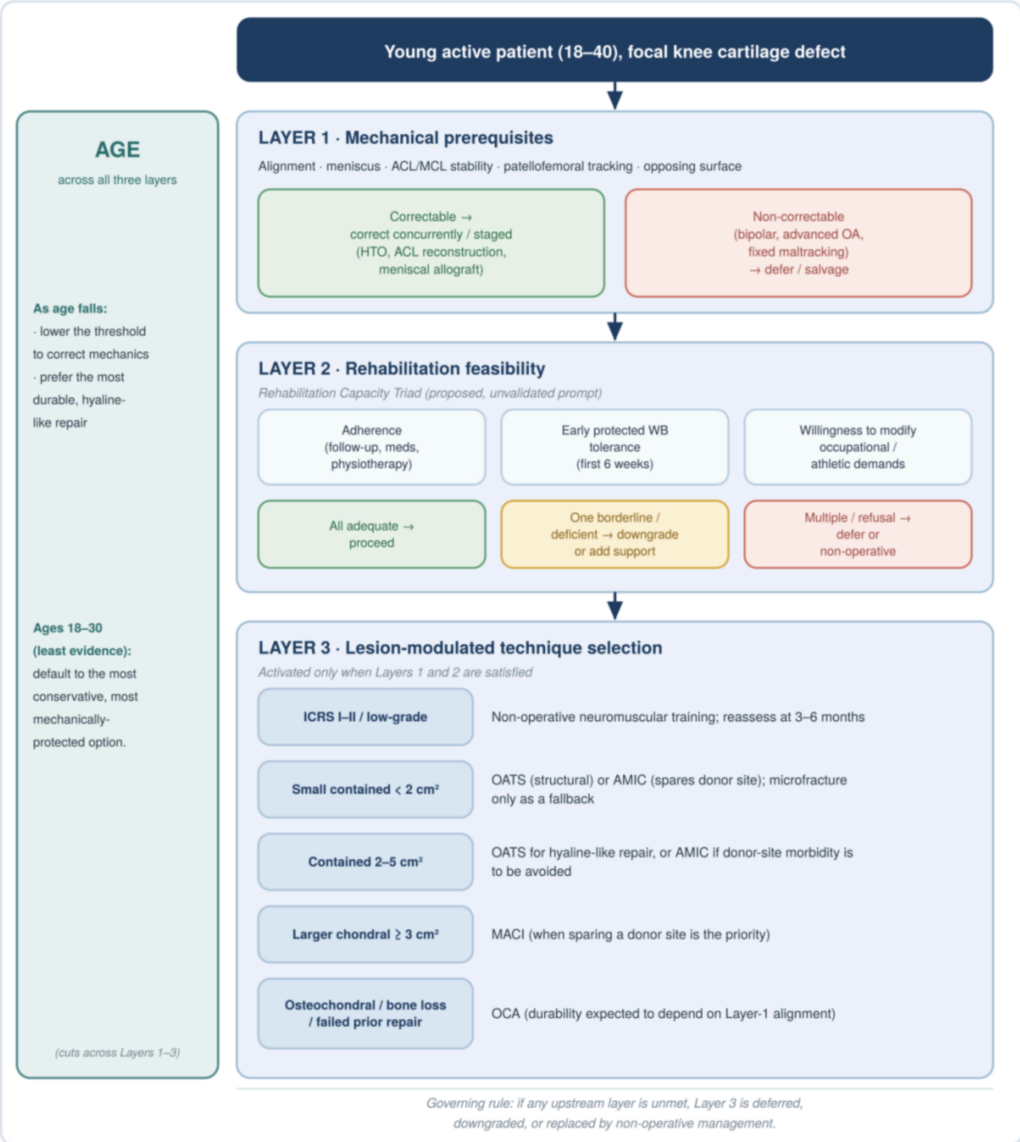

Figure 1. The age-aware, mechanics-first decision hierarchy. Each upstream layer can divert a patient to the deferral pathway; age modulates thresholds across all three layers

3.1. Layer 1 — mechanical prerequisites

Layer 1 evaluates limb alignment, meniscal function, ACL and MCL stability, patellofemoral tracking, and the opposing articular surface, distinguishing correctable from non-correctable factors. Correctable factors are addressed concurrently or in stages. A medial-compartment lesion with varus exceeding 3° can be combined with high tibial osteotomy (HTO). ACL insufficiency can be reconstructed and meniscal deficiency can be restored by an allograft. The direct evidence anchoring this layer is an alignment–survival association. In 168 patients (retrospective case series) undergoing medial-femoral-condyle ACI or OCA, neutral mechanical alignment conferred significantly longer ACI graft survival than overcorrection into valgus; among patients who underwent concomitant HTO, 5-year survival was 92% with neutral alignment versus 55% with overcorrection [8].

These factors share a common mechanism. An uncorrected adverse environment concentrates load abnormally across the repair, so the graft is prone to fail by mechanical overload regardless of

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its cellular sophistication. Correction should therefore target a neutral, physiologically loaded axis in which a Layer 3 repair can heal protected, not maximal correction for its own sake. By contrast, non-correctable factors, including bipolar disease, advanced osteoarthritis, or fixed complex maltracking, enter the deferral pathway toward non-operative or salvage management.

3.2. Layer 2 — rehabilitation feasibility (an unvalidated clinical prompt)

Layer 2 assesses whether the patient can carry out protected weight-bearing and progressive rehabilitation over 6 to 12 months. This is a non-surgical precondition for any cell- or scaffold-based technique. A lightweight Rehabilitation Capacity Triad is proposed with explicit assessment points. The triad combines two preoperative elements with one that can only be assessed postoperatively. Preoperatively, adherence is inferred from the prior record of follow-up, medication use and physiotherapy, and willingness to modify occupational or athletic demands is established by discussion. Postoperatively, early protected weight-bearing tolerance is confirmed from the response of pain and effusion within the first 6 weeks, so it guides rehabilitation intensity rather than the initial technique choice. Numeric cut-offs, which the evidence cannot yet support, are avoided; each element is instead graded as adequate, borderline, or deficient. Where all are adequate the patient may proceed. A single borderline or deficient element prompts technique downgrade or additional rehabilitation support, whereas multiple deficiencies, or refusal to adjust, defer Layer 3 or replace it with non-operative care. Although the individual constructs are plausible, their integration into this zoned scheme is what is proposed and has not been validated.

3.3. Layer 3 — lesion-modulated technique selection

Layer 3 is activated only once Layers 1 and 2 are satisfied. The technique is then mapped to lesion morphology, with tie-breakers where size ranges overlap.

- **ICRS grade I–II or low-grade lesions:** non-operative management with neuromuscular training, reassessed at 3 to 6 months.
- **Small contained defects below 2 cm²:** because durability is the goal, microfracture is not preferred as its fibrocartilage declines by 10 years relative to AMIC [6]. OATS (structural) or AMIC (donor-sparing) is preferred, whereas microfracture is a fallback only.
- **Contained defects of 2 to 5 cm²:** OATS or mosaicplasty for hyaline-like structural repair, where the Lysholm advantage over microfracture was sustained to 15–17 years [5]. AMIC where avoiding donor-site morbidity is the priority. The tie-breaker is donor-site tolerance and whether structural repair is sought.
- **Larger chondral defects of 3 cm² or more, once the size exceeds the practical donor-site limit for OATS/AMIC:** MACI; a minimum-10-year review reported approximately 9.0% reoperation and 7.4% conversion to TKA [7].
- **Osteochondral defects, bone loss, or failed prior repair:** OCA, whose durability is expected on mechanical grounds to depend on the Layer 1 alignment correction.

Age is the primary tie-breaker across these bands. Where more than one option is defensible, age 18–30 not only strengthens the preference for structural or hyaline-like repair over marrow stimulation, but also lowers the threshold for correcting alignment, instability or meniscal deficiency. A generational caveat applies throughout. Much long-term and alignment evidence derives from first- or early-generation ACI and is applied to current MACI with caution, since the techniques differ in fixation and failure mode.

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4. Discussion

4.1. Contribution

The principal contribution is to move alignment, ligamentous stability, meniscal function, and rehabilitation feasibility ahead of technique selection. Preoperative assessment thereby broadens from "is the lesion large enough, and is the technique suitable" to "can the mechanical environment be corrected, and can rehabilitation be carried out". It shows a shift from a procedure-driven to a joint-preservation-driven paradigm.

4.2. Evidentiary basis

Trials that controlled the mechanical environment and rehabilitation at enrolment reported durable results. Solheim et al. [5] excluded malalignment over 5°, instability, and inability to follow the protocol, and mosaicplasty retained its Lysholm advantage at 15 to 17 years. Volz et al. [6] excluded axis deviation and unresolved instability, and AMIC remained stable at 10 years. By contrast, trials without such control showed no consistent inter-technique advantage [4, 10]. These observations are consistent with durability depending more on mechanical protection than on cellular complexity, but the comparison is indirect and confounded by era, centre, and selection. The only direct positive signal is the alignment–survival association [8]. Accordingly, the inference is hypothesis-generating rather than established.

4.3. Considerations for application

Three considerations bear on the application. First, no RCT has compared a framework-first with a technique-first pathway. Such a trial remains an open need. Second, the Rehabilitation Capacity Triad is a clinical heuristic, not a validated scale. It requires prospective development with reliability and predictive testing. Third, the framework positions mechanics as necessary but not sufficient. Cell quality, scaffold properties and biological augmentation retain an independent contribution. Placing mechanics first reflects its persistent neglect within size-driven pathways, not a claim of exclusivity.

4.4. The under-evidenced subgroup: ages 18–30

The framework is built around age, yet the patients it most concerns are those the evidence covers least. Long-term and alignment data concentrate in cohorts with a median age in the early 30s [5, 6]. The 18-to-30 band has essentially no direct long-term evidence, even though these are the patients with the most years of knee function still ahead of them — and therefore the most to lose if a repair declines early. In the absence of subgroup data, the framework defaults for these patients to its most conservative posture. This posture involves a lower threshold for correcting the mechanical environment, a stronger preference for durable structural or hyaline-like repair, and reluctance to spend an irreversible option early. This is offered as principled extrapolation under uncertainty, to be resolved only by registries and trials that report the 18-to-30 band separately, with joint survival and functional durability as endpoints.

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4.5. Constraints and future directions

Several constraints remain. MACI is limited by cost and centralisation. Its long-term survival estimates should be interpreted with caution. The pooled minimum-10-year evidence lost about 44.5% of patients to follow-up [7], which tends to flatter reoperation and survival figures. OCA is constrained by donor availability. Future work should include framework-comparison trials and the prospective validation of a rehabilitation-capacity tool.

5. Conclusion

Long-term management of focal cartilage defects in young active patients calls for a decision framework matched to their decades-long joint preservation rather than to the limited horizon of the available evidence. The three-layer hierarchy proposed here places the mechanical environment and rehabilitation feasibility ahead of technique selection, while allowing age to modulate the choice, defaulting for the youngest and least-evidenced patients to the most durable, most mechanically protected option. The reasoning rests on a single direct signal — the alignment–survival association — reinforced by convergent but indirect evidence, while the rehabilitation-capacity prompt remains unvalidated. Accordingly, we advance the framework as a testable hypothesis rather than a standard of care. It is to be confirmed by trials whose follow-up matches this population's joint lifespan and that report the 18-to-30 subgroup separately.

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