



REWIRING THE INJURED KNEE: CORTICAL REORGANISATION, MOTOR ENGRAMS, AND THE NEUROBIOLOGY OF RETURN-TO-SPORT SYMMETRY AFTER ACL INJURY"- A CONCEPTUAL REVIEW

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ABSTRACT

BACKGROUND

Anterior Cruciate Ligament (ACL) injury precipitates not only peripheral structural damage but also central neuroplastic reorganisation. Disrupted afferent input alters sensorimotor integration, reduces corticospinal excitability, and destabilises motor engrams governing quadriceps activation and dynamic knee control. These cortical adaptations manifest as persistent arthrogenic inhibition and functional asymmetry despite successful reconstruction. Therefore, effective rehabilitation must integrate neural restoration with tissue healing to achieve true limb symmetry and safe return to sport. Recent research shows that ACL rupture disrupts motor engrams – the brain's stored movement patterns responsible for quadriceps activation, proprioception, and dynamic knee stability. These cortical changes contribute to quadriceps inhibition, altered gait, and delayed functional recovery even after successful reconstruction. Therefore, ACL rehabilitation must target both tissue healing and the restoration of disrupted neural pathways.

AIM:

To explore how motor engram disruption and cortical reorganisation influences recovery after ACL injury and to propose a neuro-integrative rehabilitation as an evidence-supported neuroplasticity technique that can facilitate brain-based rehabilitation and functional symmetry and safe return to sport.

OBJECTIVES:

Primary Objective:

To conceptually analyse the role of motor engram disruption, cortical reorganisation and central neuroplastic changes following ACL injury, and to establish a brain-based framework for rehabilitation.

Secondary Objectives:

To explain the concept of motor engrams and cortical reorganisation role in normal knee motor control and dynamic stability.

1. To synthesise existing evidence on the relationship between altered sensory input, cortical reorganisation, and quadriceps inhibition after ACL injury.

2. To conceptually propose Motor Imagery and cortical reorganisation as an adjunct neuroplasticity-based intervention in ACL rehabilitation for return to sport symmetry.

METHODOLOGY:

This conceptual review synthesises current literature related to neuroplastic changes following ACL injury, motor learning mechanisms, corticospinal excitability changes and the role of Motor Imagery in neuromuscular re-education. Articles describing cortical reorganisation, reduced corticospinal excitability, symmetry deficits and MI-induced neural activation were analysed to develop a theoretical framework for brain-integrated ACL rehabilitation for return to sport symmetry.

RESULTS:

Studies report that ACL injury leads to decreased corticospinal excitability, altered motor cortex activation, and impaired sensorimotor integration. Motor Imagery has been shown to activate similar cortical regions as actual movement, supporting the reactivation of motor engrams. Evidence indicates that Motor Imagery and corticospinal excitability enhances quadriceps muscle activation, improves proprioceptive awareness, and strengthens brain-muscle pathways – especially valuable during early postoperative phases when physical loading is limited and during sports symmetry.



CONCLUSION:

ACL rehabilitation should progress beyond a purely structural approach and incorporate strategies that promote brain healing. ACL Rehabilitation should be evolved from biomechanical model to neuro-biomechanical paradigm that integrates tissue repair and cortical restoration. Motor Imagery is a safe, accessible, and evidence-supported neuroplastic technique that can restore disrupted motor engrams and improve long-term functional outcomes. A brain integrated rehabilitation approach may optimise return to sports outcomes.

LIMITATIONS:

Heterogeneity of included literature, Limited ACL-specific Motor Imagery and Cortical Reorganisation trials, Potential publication bias, Lack of standardized Motor Imagery protocols for return to sport symmetry, Indirect evidence from related knee conditions.

KEYWORDS:

Motor engrams, Motor imagery, Cortical reorganisation, Neuroplasticity, ACL injury, Brain healing, Quadriceps inhibition, Return to sport Symmetry.

1. INTRODUCTION

1.1 Clinical Burden of ACL Injury

Anterior cruciate ligament (ACL) injury is one of the most common and functionally disabling knee injuries among athletic populations. Annual incidence rates are particularly high in pivoting and cutting sports, and approximately 20–30% of individuals sustain a second ACL injury within two years of return to sport (RTS) (Arderm et al., 2014; Webster & Feller, 2019). Despite advancements in surgical reconstruction and structured rehabilitation protocols, only nearly half of athletes return to their preinjury level of competitive sport (Arderm et al., 2014). Return-to-sport decision-making commonly relies on time since surgery, quadriceps strength testing, and limb symmetry index ($LSI \geq 90\%$). However, achieving mechanical symmetry does not consistently translate into normalized neuromuscular function or reduced reinjury risk (Grindem et al., 2016). These findings suggest that conventional rehabilitation frameworks may inadequately address underlying mechanisms contributing to persistent deficits.

1.2 Persistent Neuromuscular Deficits After Reconstruction

Persistent quadriceps weakness is one of the most frequently reported impairments following ACL reconstruction. Importantly, this weakness is not solely attributable to muscle atrophy but is strongly influenced by neural inhibition mechanisms (Palmieri-Smith et al., 2008). Arthrogenic muscle inhibition (AMI), a reflexive inhibition of quadriceps motor neuron pools following joint injury, contributes to reduced voluntary activation capacity (Rice & McNair, 2010). Emerging evidence also indicates bilateral neuromuscular alterations following unilateral ACL injury, suggesting central nervous system involvement rather than isolated peripheral dysfunction (Grooms et al., 2017). Athletes who meet traditional RTS criteria may still demonstrate altered landing biomechanics, reduced motor coordination, and compensatory movement strategies under cognitively demanding conditions (Schmitt et al., 2015). These findings highlight the limitations of strength-based RTS assessments and underscore the need for a broader neurophysiological perspective.

1.3 Neuroplastic Consequences of ACL Rupture

The ACL contains mechanoreceptors, including Ruffini endings and Pacinian corpuscles, which contribute to proprioceptive feedback and joint stability (Johansson et al.,

1991). Rupture of the ACL disrupts afferent sensory input to the spinal cord and supraspinal centers, leading to altered sensorimotor integration. Functional neuroimaging studies demonstrate cortical reorganisation in motor planning and sensorimotor regions following ACL injury (Grooms et al., 2015). Specifically, altered activation patterns in the primary motor cortex (M1), supplementary motor area (SMA), and prefrontal cortex suggest compensatory cortical adaptations. Transcranial magnetic stimulation (TMS) studies further reveal reduced corticospinal excitability and increased intracortical inhibition affecting quadriceps motor output (Pietrosimone et al., 2015). These findings indicate that ACL injury should not be conceptualized solely as a ligamentous disruption but as a sensorimotor neuroplastic condition involving central reorganization. Disruption of afferent signaling may degrade previously established motor engrams governing dynamic knee stability, contributing to persistent functional asymmetry.

1.4 Rationale for a Brain-Integrated Rehabilitation Model

Traditional ACL rehabilitation primarily focuses on tissue healing, range of motion, and progressive strength restoration. While these components are essential, they may inadequately address central neuroplastic alterations that influence motor control and quadriceps activation. Motor imagery (MI), defined as the cognitive rehearsal of movement without physical execution, has been shown to activate neural circuits overlapping with those engaged during actual movement (Lebon et al., 2012). MI enhances corticospinal excitability and may facilitate restoration of disrupted motor representations during periods of reduced mechanical loading (Ruffino et al., 2017). Given the evidence of cortical reorganisation and reduced voluntary activation following ACL injury, integrating neuroplastic interventions such as MI, sensorimotor retraining, and cognitive-motor tasks may promote restoration of motor engrams and neural symmetry. A brain-integrated rehabilitation framework may therefore bridge the gap between biomechanical recovery and true return-to-sport readiness.

2. AIM AND OBJECTIVES

2.1 Aim

The primary aim of this conceptual review is to explore the role of motor engram disruption and cortical reorganisation following ACL injury and to propose a neuro-biomechanical rehabilitation framework that integrates neural

restoration with traditional musculoskeletal rehabilitation to optimize return-to-sport symmetry.

2.2 Objectives

1. To examine the neurophysiological consequences of ACL rupture, including altered afferent signaling and corticospinal excitability changes.
2. To analyze the contribution of Motor engram disruption and Cortical Reorganisation to persistent quadriceps inhibition and functional asymmetry.
3. To propose a brain-integrated rehabilitation model aimed at restoring neural and biomechanical symmetry before return to sport.

3. METHODOLOGY

3.1 Study Design

Conceptual narrative review with theoretical synthesis.

3.2 Literature Search Strategy

- Databases searched (e.g., PubMed, Scopus, Web of Science)
- Keywords used (ACL injury, cortical reorganisation, motor engrams, motor imagery, corticospinal excitability, quadriceps inhibition)
- Time frame (e.g., 1990–2025)

3.3 Inclusion Criteria

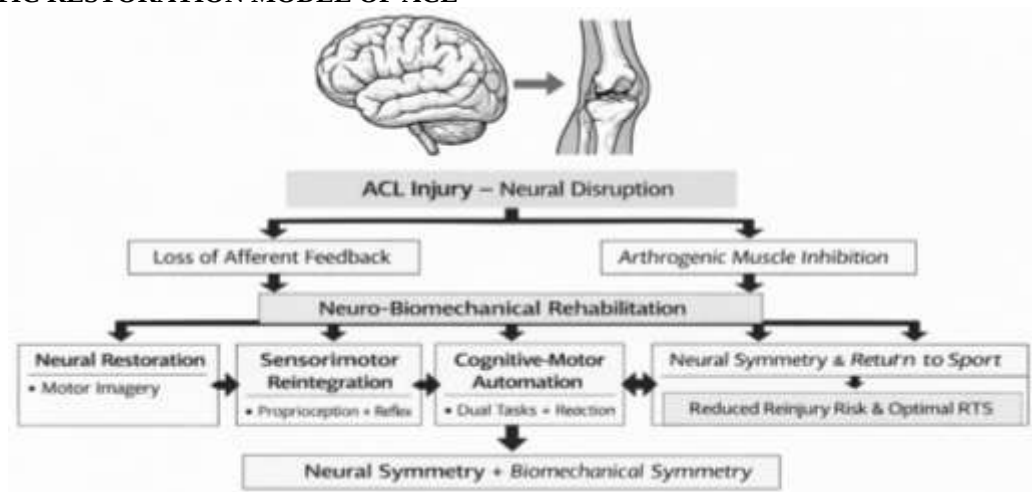
- Studies involving ACL injury or reconstruction
- Articles addressing cortical reorganisation or neuroplasticity
- Studies measuring corticospinal excitability
- Research on motor imagery in musculoskeletal rehabilitation
- Peer-reviewed English-language articles

3.4 Exclusion Criteria

- Case reports
- Editorials or opinion pieces without empirical support
- Non-human studies (unless mechanistic relevance)
- Studies unrelated to knee joint neurophysiology

5. DISCUSSION

NEUROPLASTIC RESTORATION MODEL OF ACL



3.5 Outcome Measures

Corticospinal excitability
Quadriceps activation failure
Limb Symmetry Index (LSI)
Neurocognitive motor performance

3.6 Statistical Analysis

Descriptive synthesis of available literature

3.7 Data Extraction and Theoretical Synthesis

Eligible studies were reviewed using a structured narrative framework targeting neurophysiological and biomechanical constructs, including corticospinal excitability, cortical activation, voluntary quadriceps activation, proprioception, limb symmetry, and motor imagery-induced modulation. Findings were thematically synthesized into three mechanistic domains: disrupted afferent signaling, supraspinal reorganisation with corticospinal inhibition, and motor engram degradation contributing to persistent quadriceps inhibition and asymmetry. This synthesis informed development of a systems-based neuro-biomechanical model linking ACL injury to central neuroplastic adaptation and impaired return-to-sport symmetry.

4. RESULTS

Evidence indicates that ACL injury induces central neuroplastic alterations characterized by cortical reorganisation and reduced corticospinal excitability (Grooms et al., 2015; Pietrosimone et al., 2015). Altered activation within motor planning regions reflects compensatory supraspinal control (Grooms et al., 2017). Persistent quadriceps weakness is largely neurogenic, driven by arthrogenic muscle inhibition and voluntary activation deficits despite restored muscle mass (Rice & McNair, 2010; Palmieri-Smith et al., 2008). Limb symmetry indices may overestimate functional recovery due to bilateral neural impairments (Wellsandt et al., 2017). Motor imagery enhances corticospinal excitability and supports preservation of motor representations during rehabilitation (Lebon et al., 2012; Ruffino et al., 2017). Collectively, findings support a neuro-biomechanical pathway linking ACL rupture to motor engram disruption and persistent functional asymmetry.



5.1 Neuroanatomy of the ACL and Sensorimotor Integration

The ACL contains mechanoreceptors (Ruffini endings, Pacinian corpuscles, Golgi-like receptors) that provide proprioceptive input to spinal and supraspinal centers (Johansson et al., 1991). Rupture disrupts afferent signaling, altering reflex excitability and contributing to quadriceps inhibition (Rice & McNair, 2010). Neuroimaging demonstrates cortical reorganisation within M1, SMA, and prefrontal regions, indicating central adaptation affecting knee motor control (Grooms et al., 2015).

5.2 Motor Engrams and Movement Memory

Motor engrams are distributed cortical-subcortical representations of learned movement patterns (Krakauer et al., 2019). ACL-induced sensory disruption degrades these stored motor programs, leading to compensatory strategies that may become maladaptively encoded, perpetuating asymmetry.

5.3 Cortical Reorganisation After ACL Injury

Post-ACL reconstruction, fMRI studies show increased reliance on higher-order cortical regions, reflecting compensatory motor planning (Grooms et al., 2017). TMS evidence indicates reduced corticospinal excitability and increased intracortical inhibition affecting quadriceps output (Pietrosimone et al., 2015). These supraspinal adaptations may persist despite mechanical recovery.

5.4 Neurobiology of Quadriceps Inhibition

Arthrogenic muscle inhibition (AMI) reflects reflex-mediated suppression of quadriceps activation following joint injury (Rice & McNair, 2010). Altered afferent discharge enhances presynaptic inhibition at the spinal level and increases cortical inhibitory drive (Pietrosimone et al., 2015), rendering post-ACL weakness predominantly neurogenic rather than purely muscular.

5.5 Motor Imagery and Cortical reorganisation as a Neuroplastic Intervention

Motor imagery activates neural substrates overlapping with physical execution, including M1 and SMA (Lebon et al., 2012). MI enhances corticospinal excitability and preserves motor representations during reduced loading (Ruffino et al., 2017). Early integration may facilitate engram reactivation and improve quadriceps activation (Maddison et al., 2012).

5.6 Reconsidering Return-to-Sport Symmetry

Limb symmetry indices may overlook bilateral deficits and neural readiness (Wellsandt et al., 2017). Persistent impairments under cognitive load suggest incomplete cortical recovery (Grooms et al., 2017). True RTS readiness likely requires restoration of both biomechanical and neurophysiological symmetry.

6. CLINICAL IMPLICATIONS

ACL rehabilitation should address corticospinal inhibition and motor engram disruption, not strength alone. Early motor imagery and sensorimotor retraining may restore

neural drive. Return-to-sport decisions should include neural readiness beyond limb symmetry to reduce reinjury risk.

7. FUTURE RESEARCH

Standardized motor imagery protocols and longitudinal neuroimaging are needed to define cortical recovery. TMS and EEG may serve as biomarkers of neural symmetry. Randomized trials comparing biomechanical versus neuro-biomechanical rehabilitation are warranted.

8. LIMITATIONS

Evidence is heterogeneous, with limited ACL-specific neuroplastic trials and small neurophysiological samples. As a conceptual synthesis, no quantitative aggregation was performed.

9. CONCLUSION

ACL injury induces central neuroplastic adaptations that may underlie persistent asymmetry. Rehabilitation should integrate neural and biomechanical restoration to achieve true return-to-sport readiness and minimize reinjury.

10. CONFLICT OF INTEREST STATEMENT

The authors declare that there are no conflicts of interest related to this manuscript.

11. FUNDING STATEMENT

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12. AUTHOR CONTRIBUTIONS

Madhumitha Varadarajan: Conceptualization, Literature review, Theoretical framework development, Writing – original draft preparation.

Dr. Sivakumar S, Dr. Prakash Jayabalan.: Supervision, Methodological guidance, Critical revision of manuscript.

R. Edward Thainess Durai: Neurophysiological content review, Writing review & editing.

R. Divya: Literature screening, Data extraction support, Manuscript formatting.

All authors have read and approved the final manuscript.

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