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The Fatigue-Induced Kinetic Chain Cascade (FIKCC):

A Mechanical Framework for Proximal-to-Distal Coordination Breakdown and Distal Joint Vulnerability in Rotational Sport

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Abstract

Background

Fatigue in rotational sport is often described as a reduction in output capacity, yet its more consequential effect may be mechanical: a progressive reorganization of force transfer, segmental timing, and joint loading across the kinetic chain. Existing literature has examined isolated components of this process — including trunk muscle fatigue, reduced pelvic rotational velocity, altered thoracolumbar sequencing, distal joint stiffening, and increased knee or shoulder loading under fatigue — but these findings are typically reported in fragmented form. A unifying framework explaining how local fatigue evolves into whole-chain mechanical disruption remains underdeveloped.

Objective

To propose the Fatigue-Induced Kinetic Chain Cascade (FIKCC) as an integrative biomechanical framework describing how progressive proximal fatigue alters force-vector control, disrupts proximal-to-distal sequencing, is associated with increased spinal shear-oriented loading, and ultimately shifts compensatory burden toward distal joints in rotational sport.

Methods

This article presents a conceptual mechanical framework developed through integrative evidence synthesis across rotational sport biomechanics, spinal loading mechanics, neuromuscular fatigue, segmental sequencing research, and distal joint injury literature. The model was constructed by organizing recurring fatigue-related biomechanical changes into a staged cascade progressing from proximal load-management decline to distal compensation and joint vulnerability. Emphasis was placed on mechanical variables with translational relevance, including segmental rotational velocity, trunk stiffness regulation, shear-compression redistribution, timing desynchronization, and distal stiffness compensation.

Framework Description

The proposed FIKCC model organizes fatigue-related mechanical deterioration into three progressive stages. Stage I reflects proximal fatigue accumulation, characterized by reduced active trunk stiffness, diminished compressive load tolerance, and early alterations in lumbopelvic force regulation. Stage II reflects coordination disruption, marked by reduced pelvic contribution, increased thoracolumbar compensatory demand, altered phase relationships, and rising shear-oriented spinal loading as proximal-to-distal sequencing deteriorates. Stage III reflects distal compensation, in which unresolved proximal inefficiency shifts mechanical demand toward the extremities, increasing local stiffness, impact concentration, and joint-specific injury vulnerability. The framework is presented as a mechanically reasoned, testable hypothesis architecture rather than finalized empirical doctrine.

Conclusion

The FIKCC framework provides a testable conceptual model for understanding fatigue as a progressive kinetic-chain failure process rather than a simple decline in muscular output. Fatigue is proposed to reorganize kinetic-chain mechanics from efficient proximal-to-distal transfer toward compensatory, increasingly costly mechanical solutions. By linking proximal fatigue, spinal load redistribution, sequencing breakdown, and distal joint compensation into one mechanical cascade, the model offers a translational structure for future research, athlete monitoring, injury prevention, and performance decision-making in rotational sport biomechanics.

Keywords

fatigue biomechanics; kinetic chain coordination; rotational sport mechanics; spinal shear loading; proximal-to-distal sequencing; trunk stiffness regulation; distal joint compensation; movement variability; injury risk biomechanics; force-vector control

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Graphical Abstract

The Fatigue-Induced Kinetic Chain Cascade (FIKCC)

A Mechanical Framework for Proximal-to-Distal Coordination Breakdown and Distal Joint Vulnerability in Rotational Sport

STAGE I: Proximal fatigue accumulation → reduced trunk stiffness regulation → rising stabilization cost

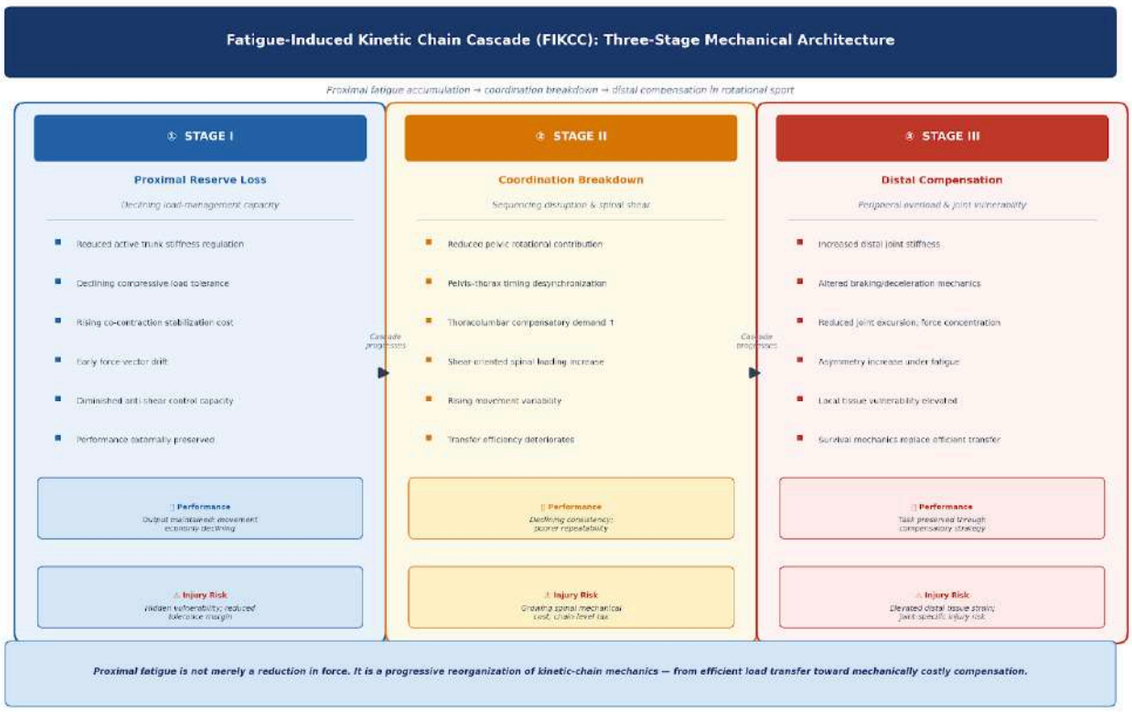
↓

STAGE II: Pelvic contribution declines → sequencing disruption → thoracolumbar compensatory demand → shear-oriented loading

↓

STAGE III: Distal stiffening and compensation → local overload → joint-specific vulnerability

Fatigue is proposed to reorganize kinetic-chain mechanics from efficient load transfer toward mechanically costly compensation — with measurable consequences for performance, coordination, and tissue-level load distribution.



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 1. Conceptual overview of the three-stage FIKCC architecture in rotational sport.

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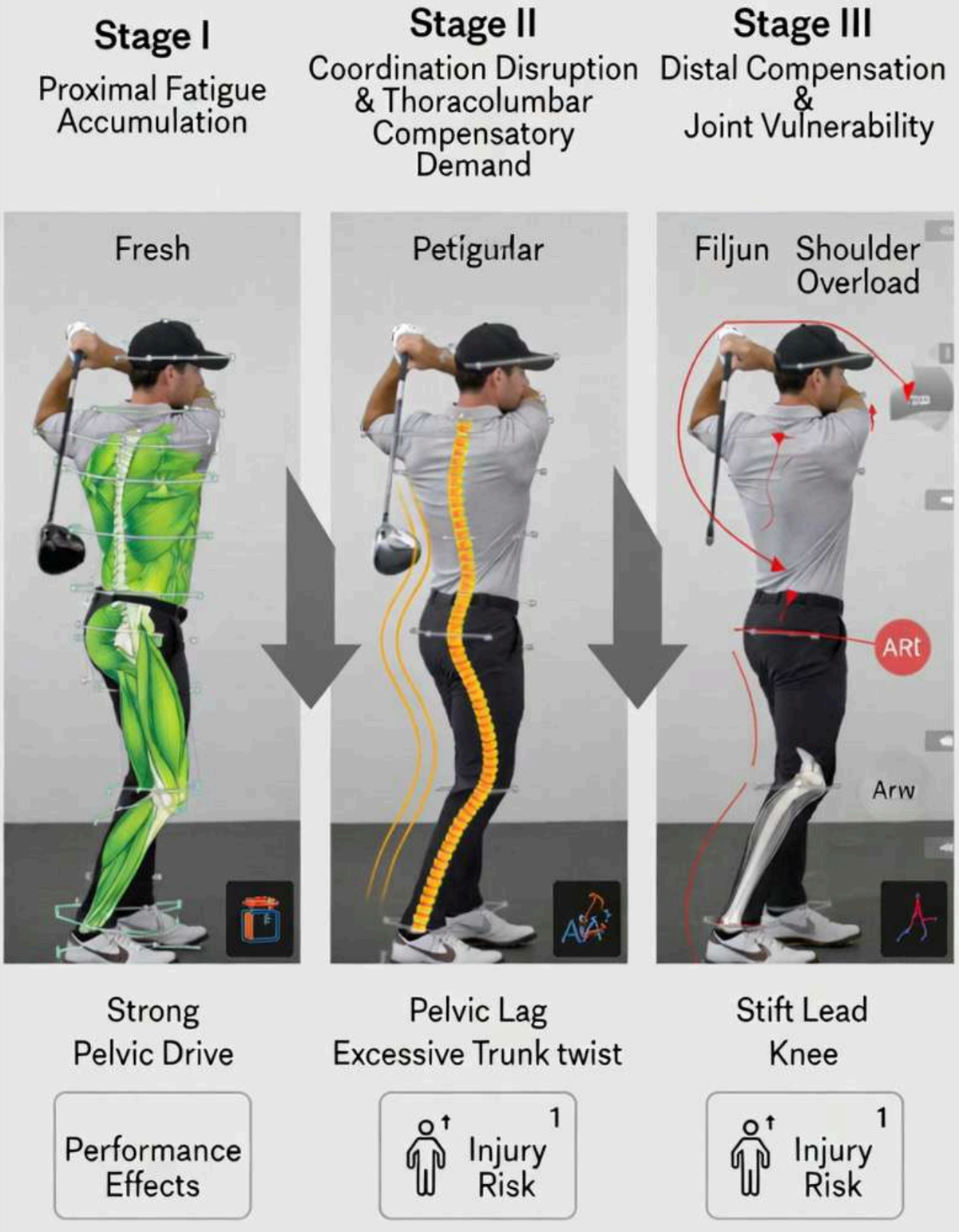
Figure 1. Conceptual overview of the three-stage FIKCC architecture in rotational sport



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1. Introduction

Rotational sport performance is governed by the coordinated sequencing of mechanical forces across the kinetic chain, where energy is transferred from proximal to distal segments to maximize velocity and efficiency (Putnam, 1993; Kibler et al., 2006). This proximal-to-distal sequencing enables effective summation of forces and angular momentum, particularly in activities such as throwing, striking, and rotational lifting (Escamilla et al., 2009). When this coordination is preserved, mechanical efficiency is optimized and unnecessary joint loading is minimized.

However, under conditions of fatigue, the neuromuscular system undergoes alterations that extend beyond simple reductions in force output. Fatigue has been shown to impair motor unit recruitment, disrupt intersegmental timing, and alter movement coordination patterns, thereby affecting the integrity of force transmission across the kinetic chain (Gandevia, 2001; Enoka & Duchateau, 2016). These disruptions can lead to compensatory movement strategies, where distal segments are increasingly recruited to maintain task performance despite proximal inefficiencies.

Existing literature has documented fatigue-related changes in movement mechanics, including altered joint kinematics, decreased stability, and increased variability in coordination patterns (Kellis et al., 2014). However, these findings are often interpreted in isolation, without integration into a unified mechanical framework that explains how fatigue propagates through the kinetic chain. This gap limits the ability of clinicians, coaches, and researchers to systematically interpret movement breakdown and its implications for performance and injury risk.

Therefore, this manuscript proposes a conceptual model termed the Fatigue-Induced Kinetic Chain Cascade (FIKCC), which organizes fatigue-related biomechanical changes into a staged mechanical progression. The model aims to provide a structured understanding of how proximal fatigue influences coordination and leads to distal compensatory mechanisms, ultimately affecting movement efficiency and injury risk.

From a mechanical standpoint, fatigue should not be interpreted as a reduction in force capacity alone, but as a redistribution of force, timing, and load across interacting segments of the kinetic chain. Segmental timing may drift. Pelvic rotational contribution may decline. Trunk stiffness regulation may become less precise. Spinal loading may shift toward less favorable patterns. Distal segments may be forced to compensate for proximal inefficiency through increased stiffness, altered joint angles, or sharper local braking demands. The athlete still performs the movement, but the way the body solves the movement problem has changed.

This is where many existing models remain incomplete. A large body of literature has documented components of fatigue-related change in rotational or multiplanar athletic tasks — including reductions in trunk endurance, altered spinal stabilizer behavior, reduced rotational velocity in proximal segments, changes in segmental coordination, and increases in local joint loading. These observations are important, but they are often interpreted in isolation. One paper describes fatigue-related decline in trunk function. Another describes altered sequencing. Another shows elevated distal injury risk. The field still lacks an integrative framework explaining how these findings fit together as parts of a progressive mechanical cascade.

In practical settings, this gap has real consequences. Coaches may identify technical breakdown late, only after distal symptoms appear. Clinicians may treat the painful joint without recognizing the proximal coordination failure that increased its burden. Researchers may quantify isolated variables without locating them within a broader chain-level fatigue logic.

This article proposes that these events can be organized into a coherent, testable framework: the Fatigue-Induced Kinetic Chain Cascade (FIKCC). The model is designed to explain how fatigue progresses from a proximal mechanical regulation problem into a whole-chain coordination failure with distal consequences. Within this framework, Stage I represents proximal fatigue accumulation and declining active stiffness regulation; Stage II represents coordination disruption with rising thoracolumbar compensatory demand and shear-oriented spinal loading; and Stage III represents distal compensation, in which unresolved proximal deficits shift mechanical burden toward peripheral joints and tissues.

The objective of this article is to introduce and develop the FIKCC as an integrative conceptual framework for rotational sport biomechanics. Specifically, the article aims to: (1) synthesize relevant evidence across trunk fatigue, spinal loading, segmental sequencing, and distal joint compensation literature; (2) define a staged proximal-to-distal fatigue cascade; (3) explain the mechanical logic linking each stage; and (4) outline translational implications for athlete monitoring, injury-risk interpretation, and future experimental testing.

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Central Proposition: Fatigue in rotational sport is not merely a decline in force production. It is a progressive reorganization of kinetic-chain mechanics.

2. Conceptual Framework Development and Integrative Evidence Synthesis

2.1 Article Positioning and Framework Purpose

The present article is a conceptual framework article built through integrative evidence synthesis, intended to organize recurring mechanical findings from multiple domains into a single staged model of fatigue-related kinetic-chain deterioration. The purpose of the FIKCC framework is therefore explanatory and translational: to provide a coherent structure through which disparate observations across rotational sport biomechanics may be interpreted as components of one progressive mechanical cascade. The framework is presented as a testable biomechanical hypothesis architecture rather than finalized empirical doctrine.

2.2 Evidence Synthesis Logic

Framework construction was informed by integrative analysis of recurring findings across five overlapping evidence domains:

- Proximal neuromuscular fatigue and trunk stiffness regulation: Literature examining fatigue-related changes in trunk muscle endurance, spinal stabilizer function, active stiffness modulation, and the interaction between passive and active spinal support systems.
- Spinal loading mechanics under repeated or sustained demand: Studies and conceptual work addressing compression–shear relationships, cumulative loading, viscoelastic creep, thoracolumbar torsion, and the mechanical consequences of repeated non-catastrophic loading.
- Proximal-to-distal sequencing in rotational tasks: Research quantifying pelvis–thorax–arm sequencing, rotational velocity order, phase timing, momentum transfer efficiency, and coordination disruption under fatigue.
- Distal compensation and joint-specific vulnerability: Literature describing how proximal control failure is associated with increased demand at distal joints through altered landing mechanics, frontal-plane stiffness, braking strategies, or compensatory movement solutions.
- Performance preservation under changing internal mechanics: Evidence showing that external task completion may remain partially preserved despite deterioration in internal movement economy, suggesting that fatigue reorganizes mechanics before gross output failure becomes obvious.

2.3 Mechanical Assumptions Underlying the FIKCC Model

The FIKCC framework rests on five core biomechanical assumptions (Table 4). These do not eliminate complexity; they organize it into testable propositions.

Table 4. Core Mechanical Assumptions Underlying the FIKCC Framework

Assumption	Description and Mechanistic Rationale
Force-transfer primacy	Rotational sport performance is treated as a force-transfer problem. Segment motions are valuable insofar as they preserve efficient load transfer, timing, and directional force organization across the kinetic chain.
Proximal system as regulatory hub	The pelvis, trunk, and thoracolumbar region must not only generate motion, but regulate torque transmission, manage stiffness, and protect downstream segments from disorganized loading.
Redistribution under fatigue	Fatigue is assumed to alter movement through redistribution, not merely reduction. The body frequently reorganizes task execution by reallocating stiffness, timing, range, and force burden across available structures.
Distal overload as consequence	Distal loading peaks may reflect compensation for failed proximal regulation rather than locally originating pathology in every case. This does not deny local risk factors but acknowledges their upstream context.
Progressive, non-linear cascade	The cascade describes dominant trends in system behavior. Athletes may move between stages at variable rates, show mixed characteristics, and experience overlapping stage transitions.

2.4 Framework Construction Strategy

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The framework was constructed in four sequential steps: (1) identification of recurring mechanical events from the literature (reduced trunk endurance, altered stabilizer behavior, reduced pelvic contribution, timing desynchronization, distal stiffening, joint-specific vulnerability); (2) sorting of events by chain position (proximal regulation, intersegmental transfer, or distal compensation); (3) ordering by likely fatigue progression from subtle proximal reserve loss to visible distal compensatory behavior; and (4) formalization into a three-stage cascade. This process produced the FIKCC structure: Stage I (proximal load-management decline), Stage II (coordination breakdown and spinal compensatory demand), Stage III (distal compensation and joint vulnerability).

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3. The Fatigue-Induced Kinetic Chain Cascade (FIKCC) Model

3.1 Overview of the Three-Stage Cascade

The FIKCC model conceptualizes fatigue as a proximal-to-distal transition in movement strategy. As fatigue accumulates, the movement system shifts through three increasingly costly strategies: Stage I — the athlete begins losing proximal mechanical reserve while still appearing functionally competent; Stage II — the movement sequence becomes less coordinated, with rising thoracolumbar compensatory demand and altered spinal loading; Stage III — distal structures compensate for unresolved proximal deficits, increasing local stiffness, braking demand, and joint-specific tissue risk.

Table 1. Proposed Three-Stage Architecture of the FIKCC

Stage	Primary Mechanical State	Key Biomechanical Features	Likely Performance Effect	Likely Risk Implication
Stage I	Proximal reserve loss	Reduced active trunk stiffness regulation; declining compressive load tolerance; rising co-contraction cost; early force-vector drift; diminished anti-shear control	Output may remain preserved; movement economy declines; rising internal effort	Reduced tolerance margin; early hidden mechanical vulnerability
Stage II	Transfer inefficiency	Reduced pelvic contribution; altered pelvis–thorax timing; thoracolumbar compensatory demand; shear-oriented spinal loading (hypothesized); rising movement variability	Increasing variability; poorer repeatability; reduced sequencing quality	Growing spinal mechanical cost; whole-chain compensation rising
Stage III	Distal compensation	Increased distal joint stiffness; braking compensation; reduced excursion; asymmetry; peripheral joint overload; survival mechanics replace efficient transfer	Task preserved through compensatory strategy rather than efficient transfer	Elevated distal tissue strain; joint-specific injury risk

Stage I. Proximal Fatigue Accumulation and Declining Load-Management Reserve

3.2 Mechanical Definition of Stage I

Stage I represents the earliest mechanically meaningful phase of fatigue progression. The defining feature is not gross performance collapse but declining proximal mechanical resilience. This phase is characterized by subtle reductions in active trunk stiffness regulation, reduced tolerance for repeated compressive demand, diminished anti-shear control capacity, and increased reliance on compensatory co-contraction to preserve stability. The athlete still moves and rotates, and may preserve acceptable external outcomes. However, the proximal system is doing so with less available mechanical margin. Within the FIKCC framework, Stage I is best understood as the beginning of force-governance erosion.

3.3 Trunk Stiffness as a Regulatory Variable

Trunk stiffness should not be interpreted as a universally beneficial quality requiring maximization. In dynamic rotational sport, useful stiffness is task-specific, direction-specific, and time-specific. In the early fatigue state, this regulation becomes less precise. The athlete may still create sufficient trunk tension to continue the task, but the efficiency of that tension changes — stabilizing effort may become more costly, less adaptable, and more dependent on compensatory recruitment rather than coordinated load-sharing. In practical terms, the athlete may not yet appear obviously unstable, but the system is becoming more metabolically and mechanically expensive to sustain.

3.4 Compression Tolerance, Passive Creep, and the Compensatory Stiffness Problem

Under repeated rotational effort or sustained postural demand, passive tissues are exposed to time-dependent mechanical stress. Even when absolute loading remains submaximal, cumulative exposure may alter how effectively passive structures contribute to stability. As these contributions

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become less reliable, the active system must compensate — often through increased co-contraction or segmental bracing. This strategy may initially preserve movement competence, but at higher energetic cost and with reduced adaptability. Stage I is often invisible to coarse observation, which is precisely why it matters: internal deterioration begins before external collapse becomes apparent.

3.5 Stage I: Performance Expression and Candidate Markers

In many athletes, Stage I does not produce obvious loss of external performance. Club speed, throw velocity, or gross task completion may remain stable. The room for timing error narrows. Tolerance for force-vector drift decreases. Capacity to absorb repeated loading without redistribution begins to decline. Candidate Stage I markers include:

- Declining trunk endurance under repeated task demand
- Reduced quality of active stiffness regulation
- Increased co-contraction requirements to maintain proximal control
- Subtle increases in spinal load-management cost
- Early drift in force-vector orientation without obvious task collapse
- Maintained performance output despite rising internal mechanical effort

Evidence suggests that fatigue of proximal segments, particularly the trunk, significantly alters movement control and stability. Deficits in trunk neuromuscular control have been associated with increased injury risk and altered lower limb mechanics, indicating the importance of proximal stability in maintaining kinetic chain integrity (Zazulak et al., 2007).

Stage II. Coordination Breakdown, Pelvic Contribution Loss, and Thoracolumbar Compensatory Demand

3.6 Mechanical Definition of Stage II

Stage II begins when the proximal system can no longer preserve efficient intersegmental transfer using Stage I compensations alone. The defining feature is coordination disruption: fatigue is no longer simply reducing proximal reserve but changing how motion, timing, and load are transmitted through the chain. The athlete begins to lose clean proximal-to-distal sequencing. Pelvic rotational contribution may diminish or become delayed. Thoracolumbar segments may be forced to bridge the gap between reduced pelvic drive and preserved distal intent.

3.7 Loss of Pelvic Contribution and Timing Desynchronization

Efficient rotational sport mechanics typically depend on the pelvis initiating or strongly contributing to the kinetic sequence — not merely to generate speed, but to create the appropriate temporal and directional conditions for trunk transmission and distal acceleration. When fatigue reduces effective pelvic contribution, three problems emerge: reduced momentum hand-off, increased demand on the trunk to preserve total task output, and timing distortion in which distal segments may begin accelerating under less favorable proximal conditions. This may present as subtle flattening of pelvic velocity, delayed pelvis–thorax separation, or reduced dissociation quality.

3.8 Thoracolumbar Compensatory Demand and Shear-Oriented Loading

Once pelvic contribution becomes insufficient, the thoracolumbar region is hypothesized to absorb the mechanical consequence. The trunk is required not only to transmit force but to rescue sequencing — and this rescue effort may be associated with increased torsional demand, altered rotational timing, and a shift toward shear-oriented loading states. In a well-coordinated system, spinal tissues experience substantial but manageable loading as part of integrated force transfer. In a fatigued and desynchronized system, the pattern of that loading may change: poorly timed rotational transfer under reduced proximal regulation may increase shear-oriented demand relative to useful compressive support. Thus, Stage II is the phase in which the thoracolumbar spine may increasingly function as a compensatory transmission zone rather than an efficiently regulated transfer zone.

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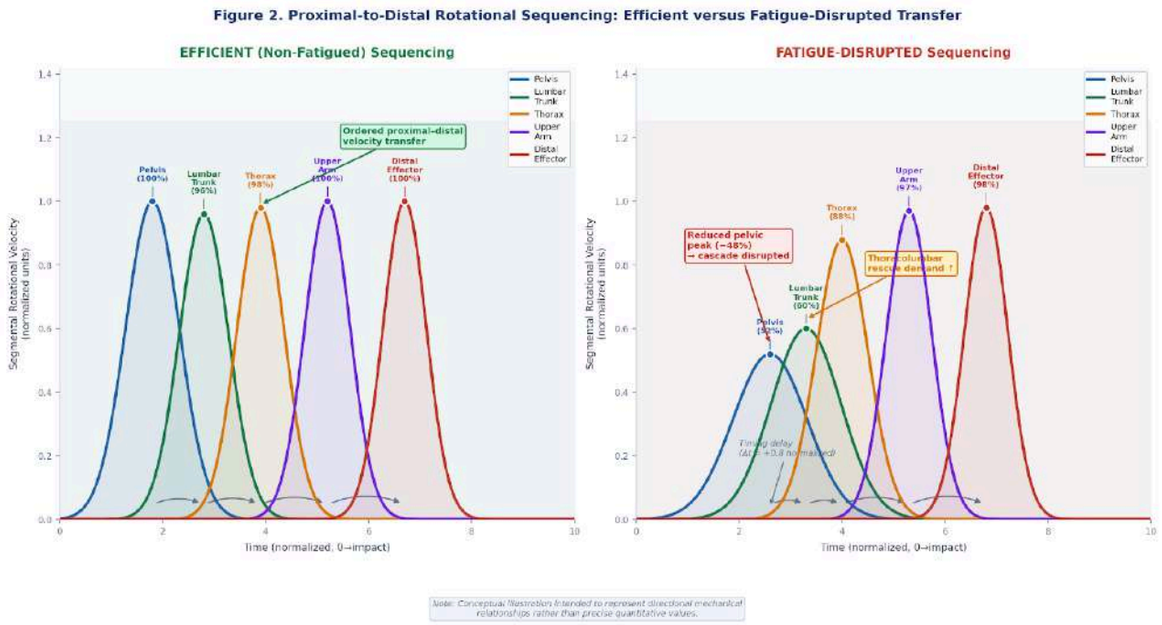

Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 2. Efficient (left) versus fatigue-disrupted (right) proximal-to-distal rotational sequencing. Reduced pelvic contribution and altered timing are associated with increased thoracolumbar rescue demand under fatigue.

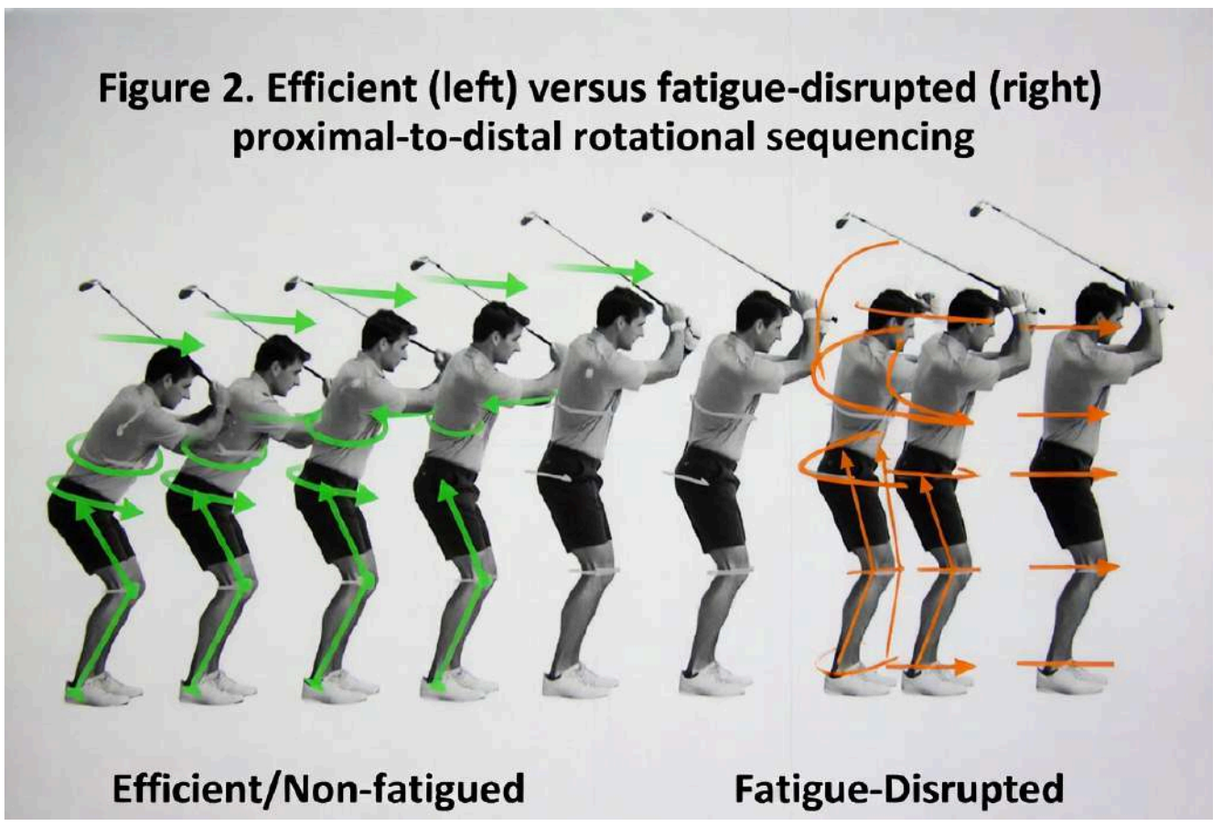

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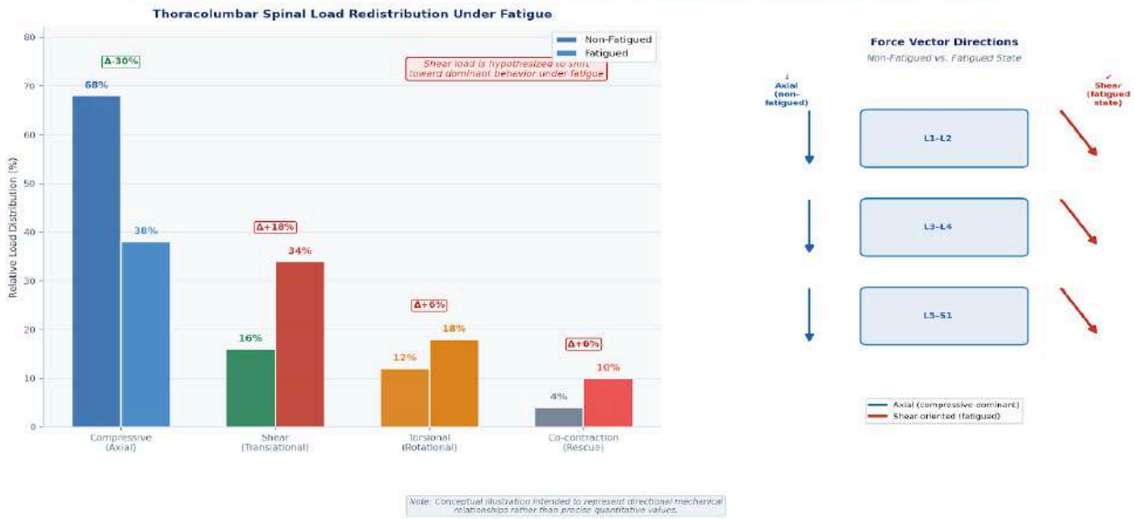
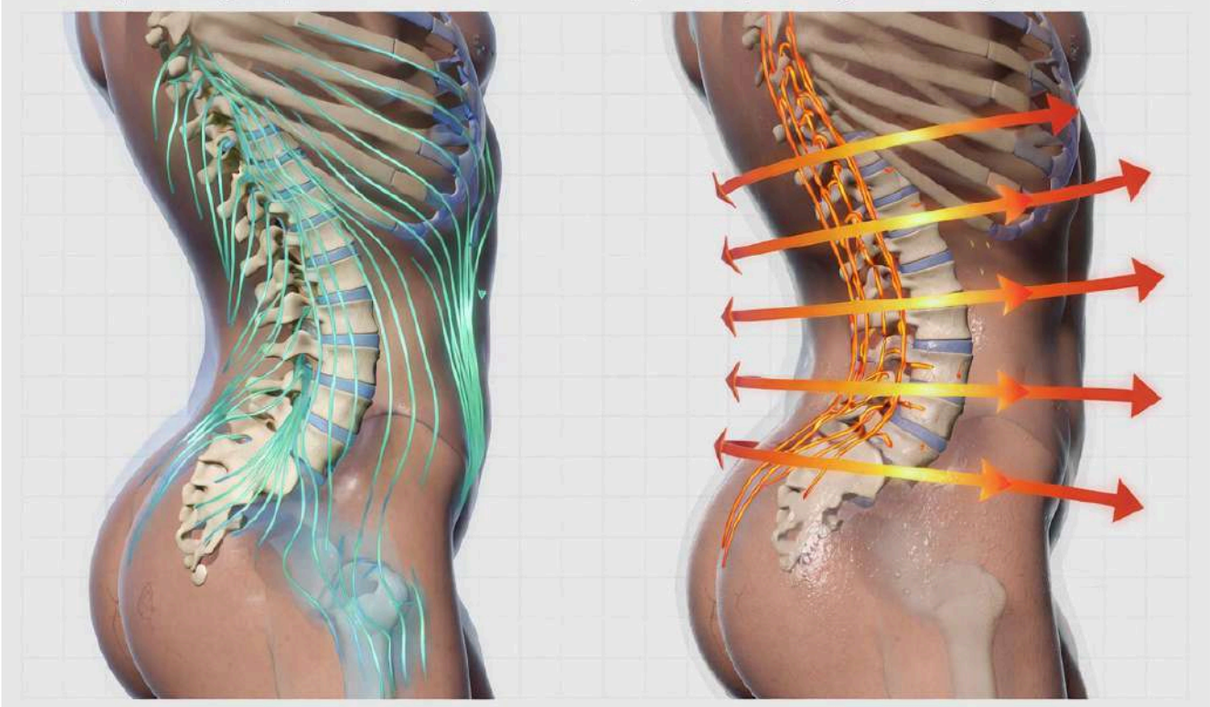
Figure 3. Spinal Load Redistribution: From Regulated Compression Toward Shear-Dominant Behavior Under Fatigue



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 3. Hypothesized redistribution of thoracolumbar spinal load from compression-dominant transfer (non-fatigued) toward shear-oriented compensatory loading under fatigue. Values are illustrative and directional.

Figure 3. Hypothesized redistribution of thoracolumbar spinal load from compression-dominant transfer (non-fatigued) toward shear-oriented compensatory loading under fatigue



3.9 Stage II Candidate Markers

- Reduced pelvic rotational contribution (flattened velocity peak)
- Altered pelvis–thorax separation timing and phase ratio
- Delayed or reduced proximal segment velocity peaks
- Increased thoracolumbar torsional compensatory demand
- Movement variability index increasing across session
- Shear-oriented loading behavior (modeled or estimated)
- Reduced phase coordination quality under repeated effort

Fatigue-induced disruptions in coordination and timing have been shown to increase movement variability and reduce efficiency in multi-segmental tasks. These alterations reflect a breakdown in the precise sequencing required for optimal force transfer across the kinetic chain (Davids et al., 2003).

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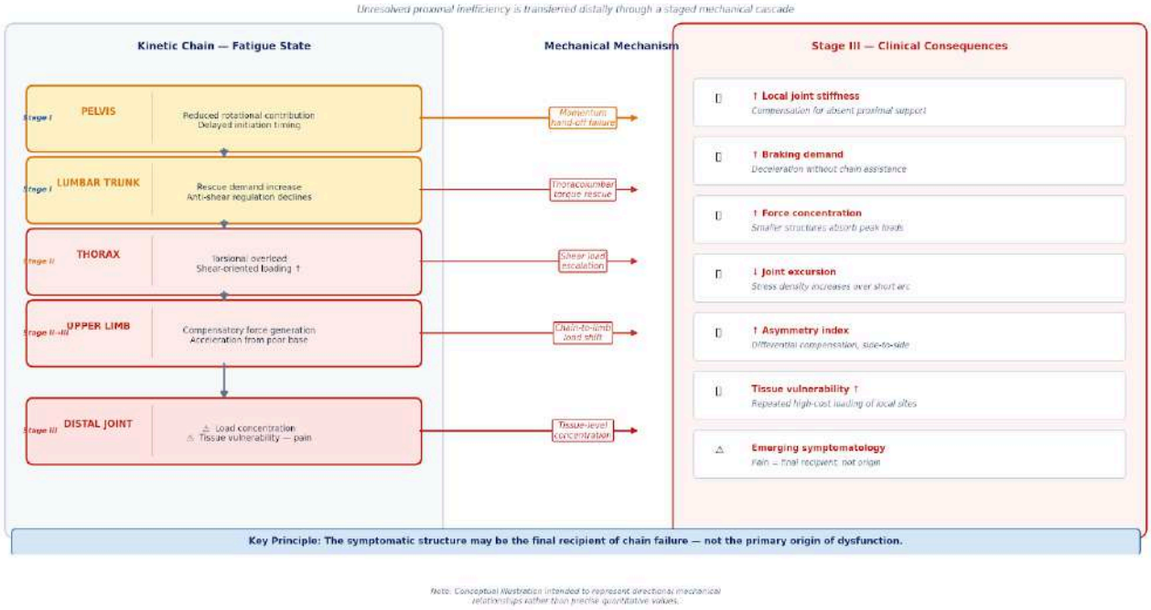
Stage III. Distal Compensation, Local Stiffness Strategies, and Joint Vulnerability

3.10 Mechanical Definition of Stage III

Stage III represents the point at which unresolved proximal inefficiency is transferred decisively to distal structures. To preserve task execution, the body increasingly uses the limbs and peripheral joints as compensatory solutions — involving increased stiffness, altered control strategies, sharper braking demands, higher localized force peaks, reduced excursion, or more abrupt impact handling. The movement still occurs, but now at a distinctly higher distal mechanical price.

Figure 4. Stage III: Distal Compensation as the Final Recipient of Kinetic Chain Failure



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 4. Stage III distal compensation as the final recipient of chain failure. Proximal reserve loss and transfer inefficiency culminate in peripheral joint overload and local tissue vulnerability.

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Figure 4. Stage III distal compensation as the final recipient of chain failure

3.11 Joint-Specific Vulnerability as Final Recipient of Chain Failure

A critical implication of Stage III is that the painful or injured tissue may be the final recipient of chain failure rather than the primary origin of dysfunction. The FIKCC framework does not deny that distal

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tissues can fail due to local weakness, morphology, prior injury, or technique errors. Rather, it argues that under fatigue conditions, distal vulnerability may also reflect a systemic sequence: proximal reserve declines → coordination breaks down → spinal and trunk transfer become less efficient → distal joints compensate → local tissues experience concentrated mechanical demand. This interpretation changes how clinicians and coaches should think about symptom timing and origin.

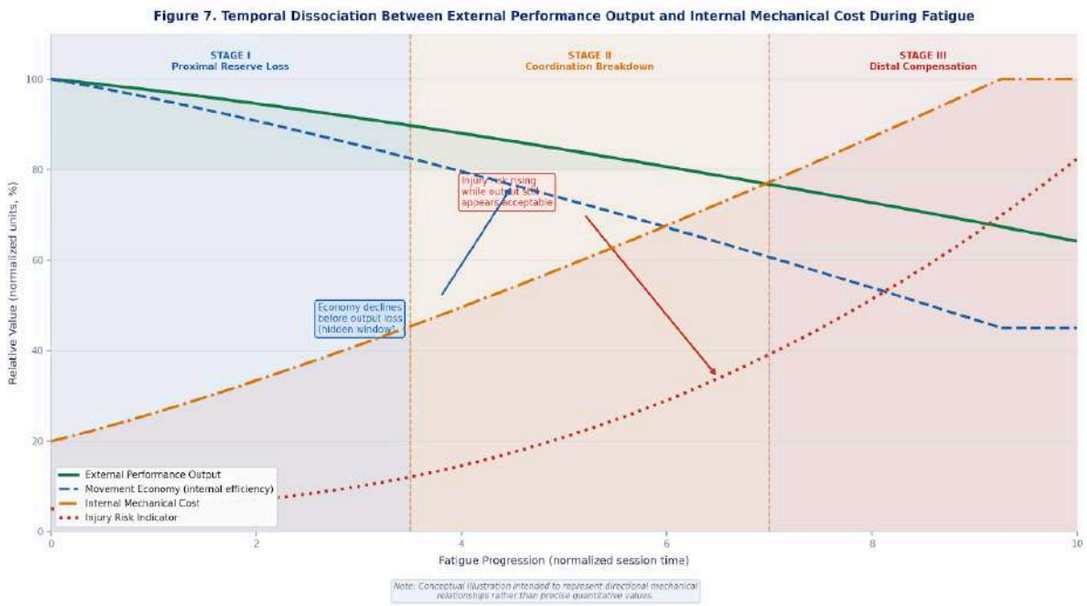
3.12 Stage III Candidate Markers

- Increased distal joint stiffness signatures under fatigue
- Altered lead-limb braking or deceleration mechanics
- Reduced joint excursion with higher local force concentration
- Higher asymmetry index during terminal task phases
- Visible distal compensation despite preserved task completion
- Onset of joint-specific pain, tissue irritability, or strain sensation

As compensation shifts toward distal joints, altered loading patterns may increase mechanical stress and injury risk. Studies have demonstrated that fatigue-related compensatory strategies are associated with increased joint loading and decreased neuromuscular control, particularly in high-demand athletic movements (Hewett et al., 2005).

3.13 Core Architecture of the FIKCC Model

Taken together, the three stages form one coherent proximal-to-distal cascade. Stage I creates the conditions for Stage II by reducing proximal control reserve. Stage II creates the conditions for Stage III by degrading transfer efficiency and increasing compensatory demands. Stage III represents the distal endpoint of unresolved upstream dysfunction. The athlete does not fail all at once — the chain progressively adopts more expensive solutions until the final solution becomes locally costly.



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 7. Temporal dissociation between external performance output and internal mechanical cost during fatigue progression across the three FIKCC stages. Movement economy declines before output collapse; injury risk rises within the 'hidden window'.

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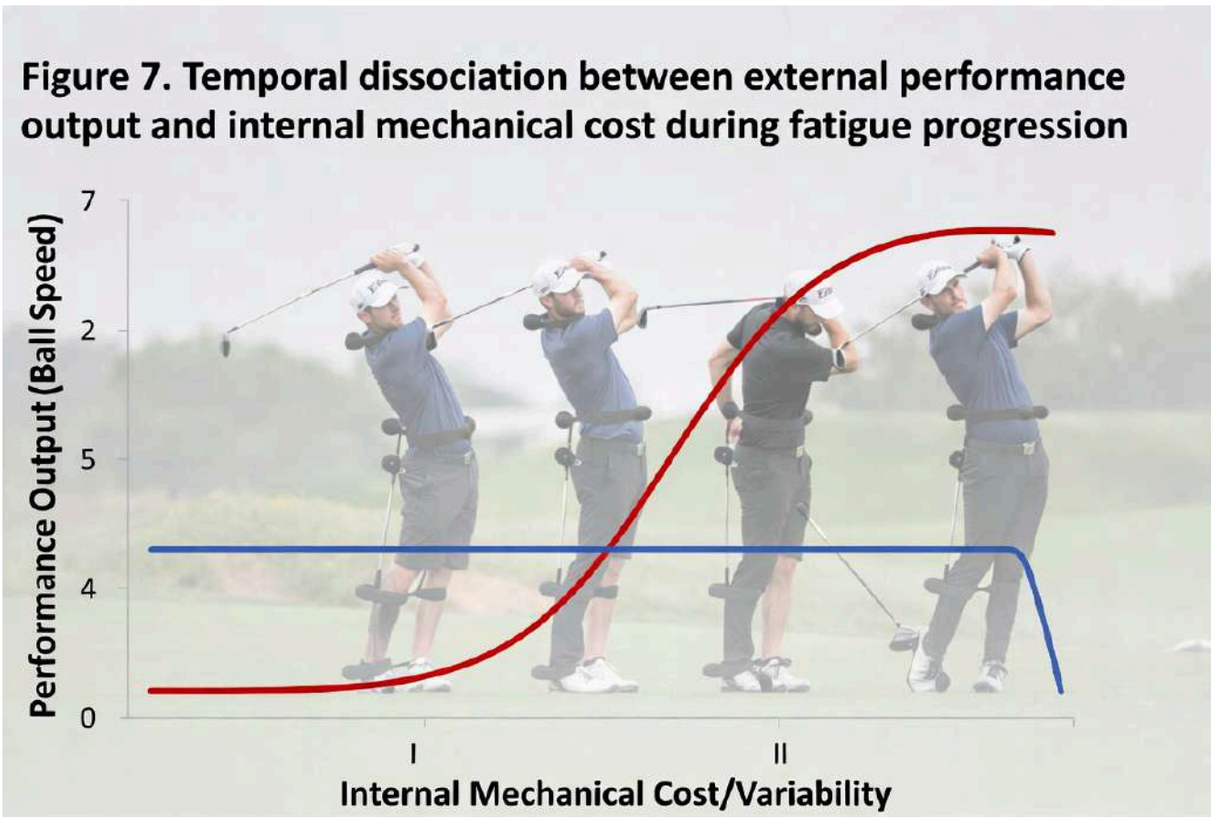
Figure 7. Temporal dissociation between external performance output and internal mechanical cost during fatigue progression



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4. Mechanistic Integration of the FIKCC Model

4.1 From Fatigue to Mechanical Reorganization

The principal value of the FIKCC model lies not merely in naming three stages but in explaining the mechanical continuity between them. In the non-fatigued state, proximal segments contribute to rotational sport through three interrelated functions: (1) generation of useful momentum, (2) regulation of force-vector orientation, and (3) stabilization of transfer conditions for downstream segments. Once fatigue reduces proximal regulation capacity, the system is forced to preserve performance using alternative strategies. Stage I reflects the earliest loss of mechanical reserve; Stage II reflects deterioration in mechanical transfer; Stage III reflects compensation through mechanical redistribution.

4.2 Force-Vector Drift and Segmental Consequence

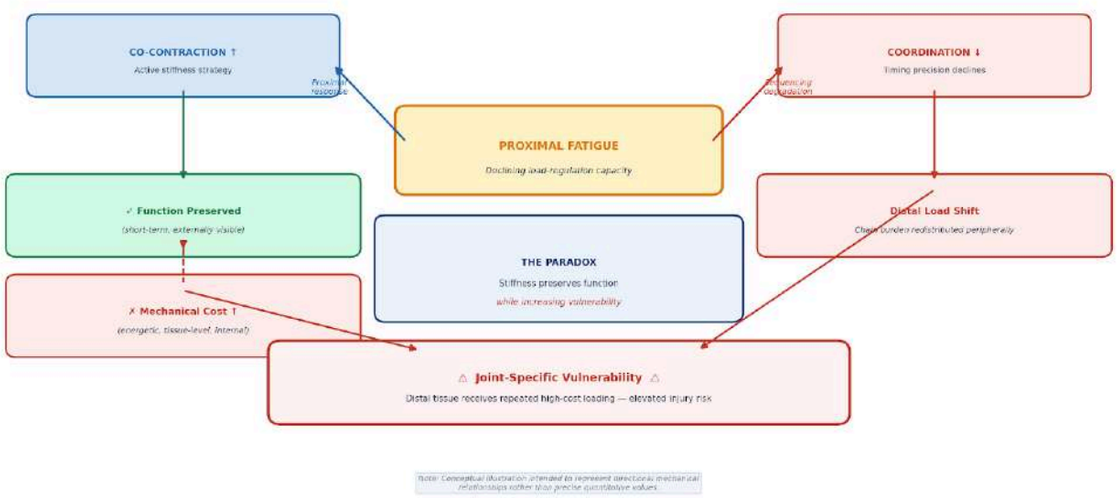
When proximal fatigue accumulates, vector control may begin to drift — presenting as subtle changes in trunk inclination, pelvic contribution, segment timing, or directional force application. Once this occurs, the downstream segments no longer receive the same quality of transfer. The thoracolumbar region increasingly serves as a compensatory bridge: as the cost of that bridge rises, so too may the probability of less efficient loading patterns, including greater shear-oriented demand, altered torsional behavior, or increased co-contraction burden. Force-vector drift is therefore not merely a technical flaw — it is a load-path problem.

4.3 The Compensatory Stiffness Paradox

A recurring theme across the cascade is the compensatory stiffness paradox: under fatigue, the body often responds to declining control by increasing stiffness somewhere in the chain. This may temporarily preserve task completion but does not restore movement efficiency — it relocates the mechanical burden. At the proximal level, increased co-contraction may preserve spinal control but at higher energetic cost and reduced adaptability. At the thoracolumbar level, increased torsional effort may maintain sequence continuity but may also elevate local tissue demand. At the distal level, increased stiffness may preserve directional precision but expose joints to sharper force concentration. The paradox: stiffness may preserve function while simultaneously increasing vulnerability.

Figure 5. The Compensatory Stiffness Paradox

increased stiffness preserves visible task completion while simultaneously increasing local mechanical vulnerability



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values.

Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 5. The Compensatory Stiffness Paradox: increased stiffness preserves visible task completion while accumulating hidden internal mechanical cost and distal tissue vulnerability.

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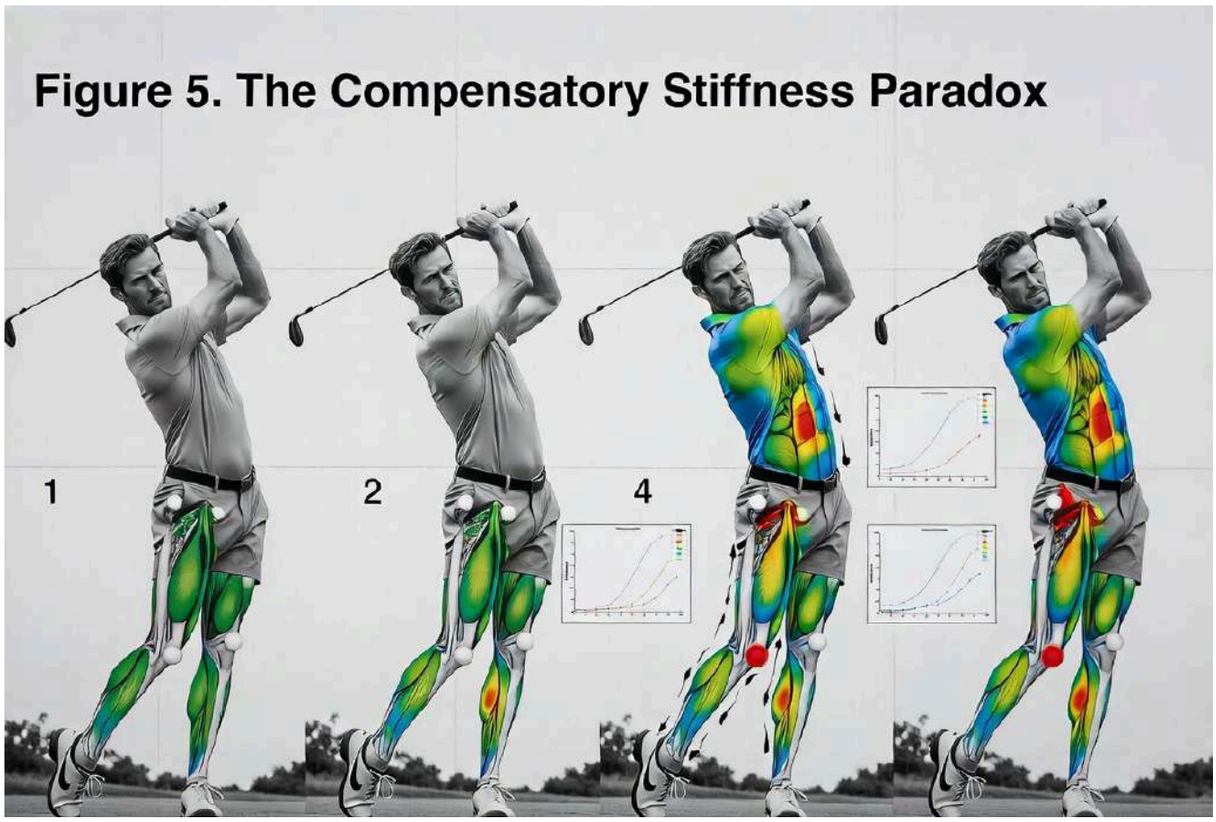

4.4 Proximal-to-Distal Failure as a Continuum

The FIKCC model proposes that fatigue-related breakdown is best understood as a continuum rather than as separate unrelated events. Proximal fatigue, spinal loading change, coordination drift, and distal overload are not independent problems occurring in parallel — they may be sequential expressions of the same unresolved chain-level disturbance. That is why the FIKCC model is not only descriptive but interpretive: it helps reframe where in the sequence a problem may have originated.

5. Performance Implications of the FIKCC Framework

5.1 Performance Loss Does Not Always Begin with Output Loss

A major implication of this model is that performance deterioration should not be defined only by visible decline in external outcome measures. In rotational sport, athletes often preserve gross output for longer than expected by reorganizing internal mechanics. Ball speed may remain adequate; shot distance may remain near baseline. Yet the internal route by which that output is achieved may already be less efficient, more variable, and more mechanically expensive. From a performance-science perspective, the earliest meaningful sign of fatigue may therefore be declining movement economy — not declining absolute outcome.

5.2 Repeatability as a Sensitive Performance Marker

Stage II of the cascade suggests that repeatability may be more sensitive than peak output for identifying fatigue-related deterioration. Once sequencing begins to drift, athletes may still produce isolated high-quality repetitions, but the consistency of those repetitions declines — segmental timing becomes less stable, rhythm becomes less dependable, and mechanical variability increases. In high-skill rotational sports such as golf, baseball, tennis, and striking disciplines, a model treating fatigue as a coordination problem naturally predicts that consistency will erode before total capability disappears.

5.3 Efficiency, Timing, and the Cost of Preserved Output

The FIKCC model suggests that a preserved performance metric can hide an increased biological cost. A session appearing successful on outcome measures alone may still be mechanically unfavorable if those outcomes are achieved through unstable or increasingly costly movement solutions. Accordingly, one of the key practical uses of the FIKCC framework is to distinguish between

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performance preserved through efficient mechanics and performance preserved through compensatory mechanics. These are not equivalent states.

6. Injury-Risk Interpretation Through the FIKCC Lens

One of the strongest contributions of the FIKCC model is that it reframes injury risk as a possible endpoint of unresolved mechanical redistribution rather than as an isolated local event. Fatigue-related injury may arise because the chain progressively transfers greater burden toward regions never intended to carry that burden repeatedly under fatigue. The model suggests stage-specific risk interpretation: Stage I risk is largely hidden and relates to reserve loss and rising stabilization cost; Stage II risk relates to repeated transfer inefficiency and thoracolumbar compensatory loading; Stage III risk relates to visible distal compensation and concentrated tissue demand. This stage-specific view encourages earlier intervention — shifting from symptom detection toward stage detection.

A common clinical trap is to equate the painful structure with the primary origin of dysfunction. A distal symptom may be real and locally important but still represent the end-stage consequence of proximal coordination failure. For example, a knee may become a visible site of stress because trunk and pelvic control no longer provide an efficient deceleration base. A shoulder may become symptomatic because the proximal chain fails to sequence and transfer momentum cleanly, forcing the upper extremity to generate or brake more force than intended. This does not eliminate the need for local assessment — it contextualizes it.

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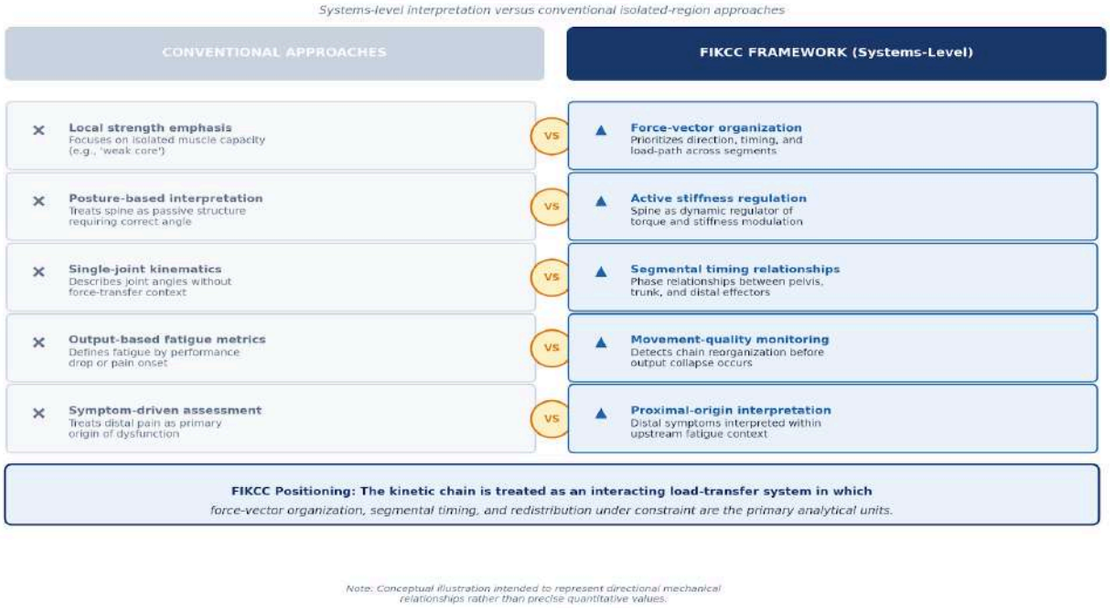
7. Biomechanical Positioning of the FIKCC Framework

The FIKCC framework is positioned within a systems-level interpretation of movement mechanics, in which the kinetic chain is treated as an interacting load-transfer system rather than a collection of isolated joints or muscles. Unlike models that emphasize local strength, posture, or isolated joint kinematics, the present framework prioritizes:

- (1) Force-vector organization — the direction, magnitude, and timing of forces transmitted through the chain;
- (2) Segmental timing relationships — phase coordination between the pelvis, trunk, thorax, and distal effectors;
- (3) Load redistribution under constraint — how the system reallocates mechanical burden in response to declining proximal reserve;
- (4) The mechanical consequences of fatigue-driven compensation — how compensatory strategies alter tissue-level loading at both proximal and distal sites.

In this context, the spine and trunk are not treated as passive structures or posture-dependent stabilizers, but as active regulators of load transfer, stiffness modulation, and torque propagation across the system. This systems-level positioning distinguishes the FIKCC framework from models that examine fatigue through isolated physiological or kinematic lenses, and provides the conceptual basis for its staged cascade logic.

Figure 8. Biomechanical Positioning of the FIKCC Framework



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 8. Biomechanical positioning of the FIKCC framework relative to conventional isolated-region approaches. The FIKCC model prioritizes force-vector organization, segmental timing, and load redistribution as primary analytical units.

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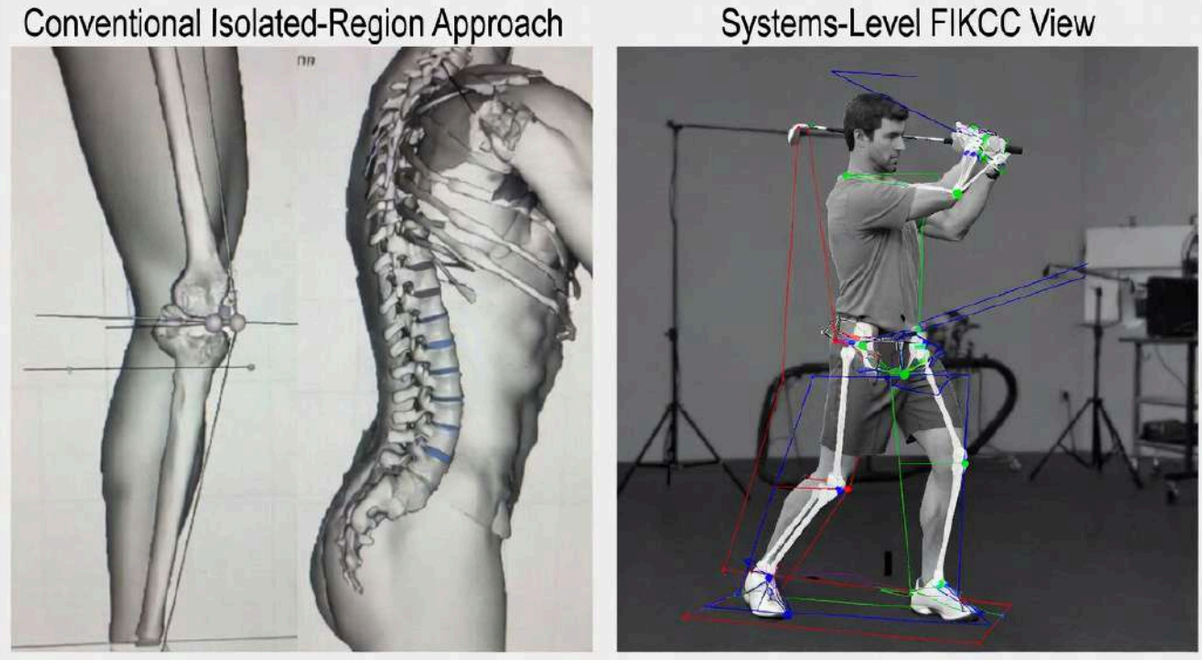
Figure 8. Biomeclanical positioning of the FIKCC framework relative to conventional isolated-region approaches



8. Clinical and Applied Translation of the FIKCC Model

8.1 Practical Translation by Stage

Table 3. Translational Interpretation of the FIKCC Model for Applied Practice

Stage	Athlete Presentation	Practitioner Suspicion	Immediate Practical Response
Stage I	Looks mostly normal; effort cost rising; output preserved; movement qualitatively intact	Hidden proximal reserve decline; rising stabilization cost; narrowing error margin	Modify exposure volume; monitor trunk fatigue index; compare early vs. late repetition quality
Stage II	Rhythm and repeatability declining; variability increasing; late-session rhythm loss	Transfer failure; thoracolumbar rescue strategy activation; chain desynchronization	Assess sequencing under fatigue; reduce high-cost repetitions; retrain proximal timing quality
Stage III	Visible compensation strategies; distal joint symptoms emerging; asymmetry visible	Distal tissues receiving unresolved upstream mechanical burden	Reduce session load; protect symptomatic structure; restore proximal contribution and transfer quality

8.2 Return-to-Play and Progression Logic

The FIKCC model has important implications for progression decisions. An athlete should not be considered fully restored merely because pain is reduced or isolated strength benchmarks are met. If the chain still enters Stage II or Stage III behavior under repeated effort, the athlete may remain mechanically underprepared for real competition. A more robust return-to-play model would therefore assess whether the athlete can: (1) preserve proximal control under repetition, (2) maintain segmental sequencing under fatigue, and (3) avoid distal survival strategies during high-intent efforts. This supports a capacity-under-fatigue model of readiness rather than a simple pain-free-at-rest model.

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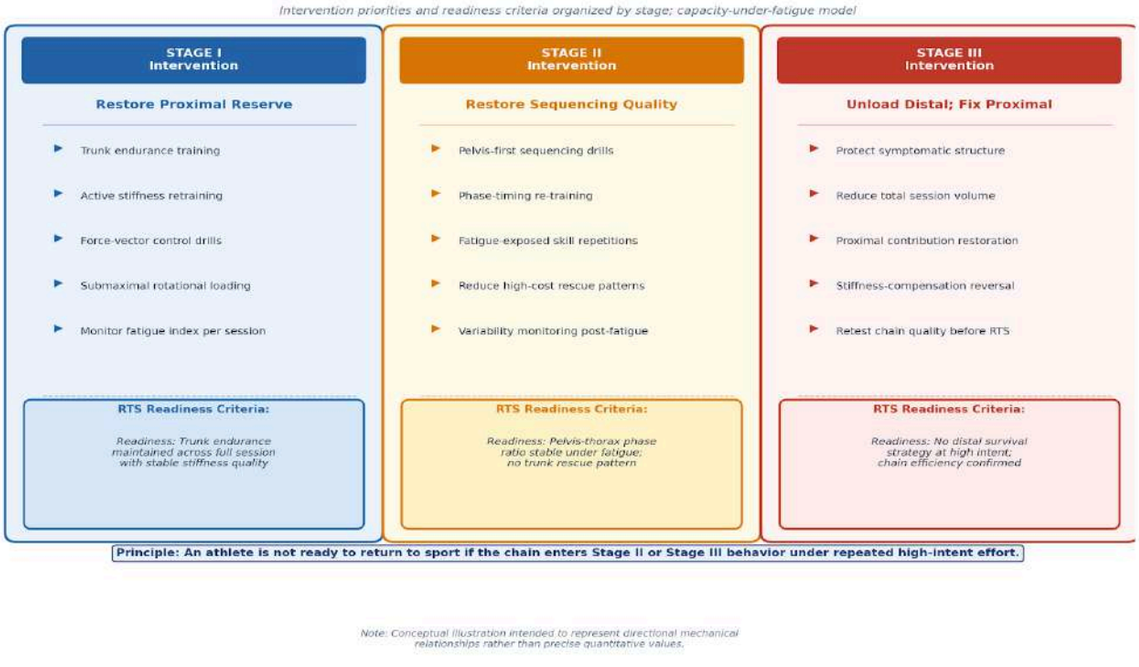
Figure 9. FIKCC-Informed Return-to-Sport and Intervention Decision Framework



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 9. FIKCC-informed return-to-sport and intervention decision framework. Stage-specific intervention priorities and readiness criteria organized around the capacity-under-fatigue principle.

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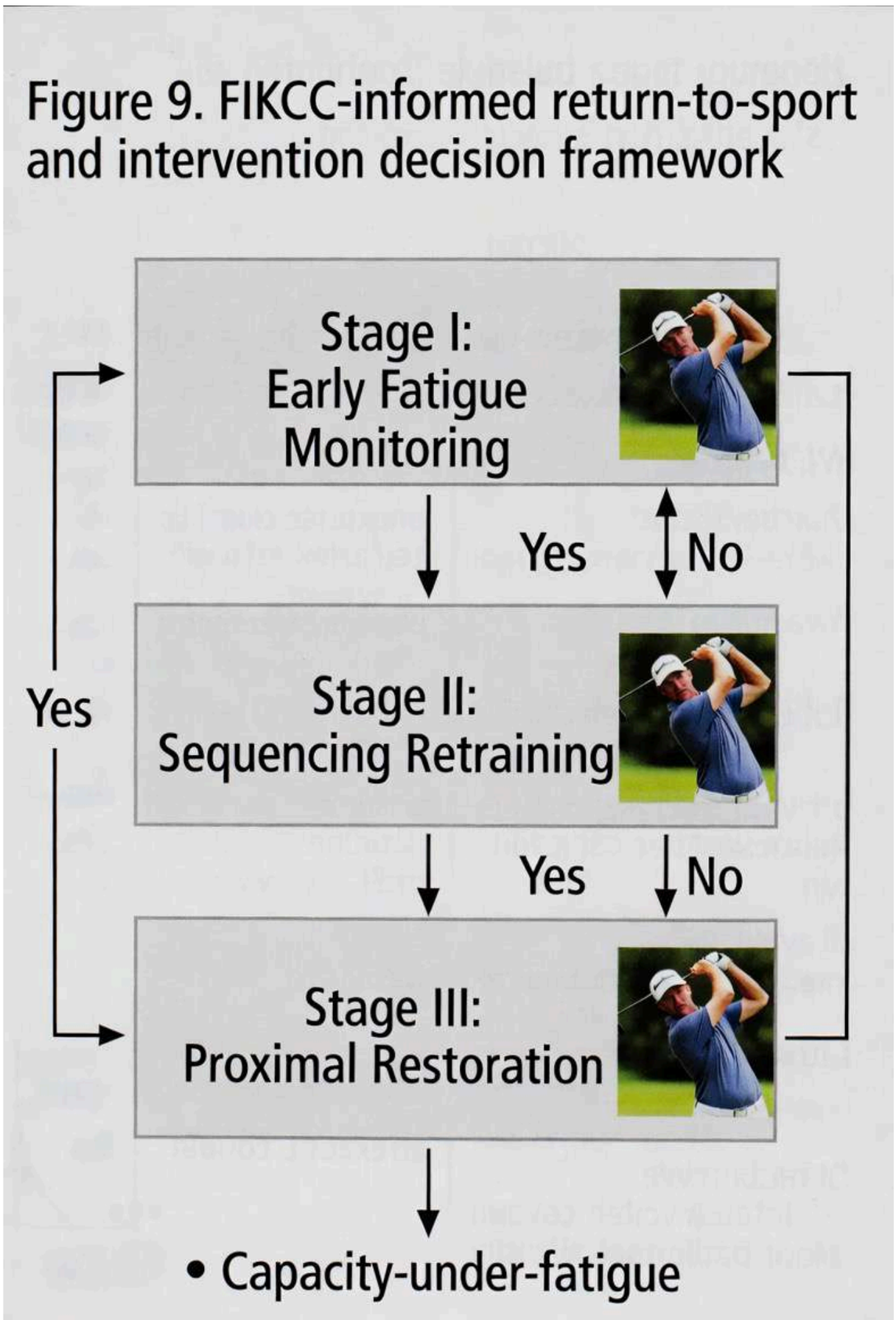
Figure 9. FIKCC-informed return-to-sport and intervention decision framework



9. Applied Monitoring Model

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9.1 Monitoring Fatigue as a Mechanical State

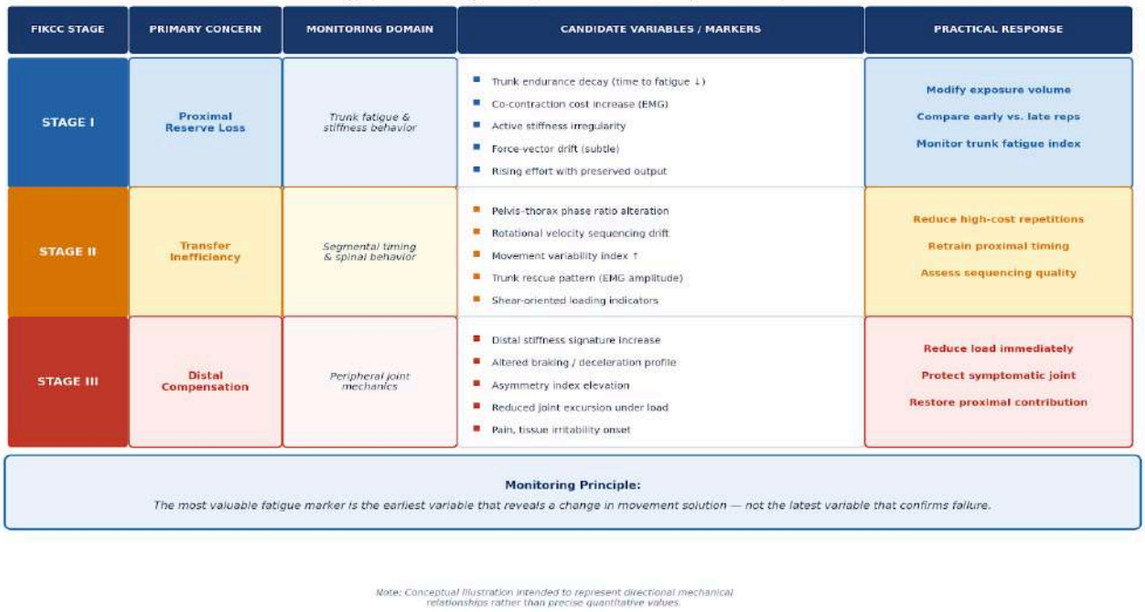
The FIKCC framework supports an important shift in monitoring philosophy: fatigue should not be assessed only by workload totals, perceived exertion, heart rate, or output decline. It should also be assessed as a changing mechanical state — asking whether the athlete is still using the same transfer strategy, whether the proximal system is still regulating load effectively, whether segmental timing has become noisier, and whether the distal chain has begun compensating for upstream decline.

Table 2. Candidate Monitoring Markers Aligned to Each FIKCC Stage

Stage	Monitoring Domain	Candidate Monitoring Variables / Markers
Stage I	Proximal fatigue reserve	Trunk endurance decay (time-to-fatigue ↓); rising co-contraction cost (EMG); altered active stiffness behavior; early force-vector drift; increased effort with preserved output metric
Stage II	Transfer quality	Pelvis–thorax phase ratio disruption; reduced rotational velocity sequencing quality; increased trunk rescue effort amplitude (EMG); movement variability index ↑; thoracolumbar shear-oriented load indicators (modeled)
Stage III	Distal compensation	Increased local stiffness signatures; altered braking/deceleration profile; asymmetry index elevation; reduced joint excursion under repeated load; pain, swelling, or tissue irritability onset

Figure 6. FIKCC-Aligned Applied Monitoring Framework

Stage-specific monitoring domains, candidate variables, and practitioner response



Note: Conceptual illustration intended to represent directional mechanical relationships rather than precise quantitative values. Figure 6. FIKCC-aligned applied monitoring framework: stage-specific monitoring domains, candidate variables, and practical responses for coaches, clinicians, and sport scientists.

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Figure 6. FIKCC-Aligned Applied Monitoring Framework

Stage I	Stage II	Stage III
Monitoring Domain	Candidate Variables	Practical Responses
Monitoring Domain	EMG traces velucity-time graphs asymmetry indices	EMG traces velucity-indices asymmetry indices
Monitoring Domain	asymmetry indices (e., (e.,, ^t) asymmetry indices	Rigatice for Responeses

Monitoring Principle: The most valuable fatigue marker is the earliest variable that reveals a change in movement solution — not the latest variable that confirms failure.

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10. Practical Implications for Performance and Rehabilitation

The FIKCC framework suggests that intervention strategies should prioritize restoration of proximal load-management capacity before addressing distal symptoms. Training approaches focused solely on local joint strengthening may fail to address the underlying chain-level disturbance — a distal joint cannot be adequately protected while the proximal system that was meant to support it continues to fail under fatigue.

Effective intervention, informed by the FIKCC model, may require:

- (1) Improving trunk fatigue resistance — through endurance-based trunk training that emphasizes sustained regulation quality, not merely peak force;
- (2) Restoring proximal-to-distal sequencing — through drills that reinforce pelvis-first initiation, pelvis–thorax phase relationships, and timing quality under increasing fatigue exposure;
- (3) Reducing compensatory stiffness strategies — by identifying and targeting the stiffening patterns that preserve visible function at the cost of internal efficiency;
- (4) Monitoring coordination quality under repeated effort — integrating stage-sensitive metrics (movement variability, sequencing phase ratios, trunk effort signatures) into ongoing athlete surveillance.

For rehabilitation specifically, this framework challenges the convention of resolving local pain as the primary endpoint of treatment. An athlete who has recovered pain-free range of motion but continues to enter Stage II or Stage III movement solutions under fatigue has not yet restored the chain-level capacity needed for safe, effective return to rotational sport. The FIKCC model therefore supports the integration of fatigue-exposed movement assessment into standard rehabilitation progression criteria.

This manuscript presents a conceptual biomechanical framework derived from established literature in fatigue, motor control, and kinetic chain dynamics. The proposed FIKCC model is intended as a hypothesis-generating structure that integrates existing evidence into a staged mechanical cascade. While grounded in prior research, the framework requires empirical validation through experimental studies, including motion capture analysis, electromyography, and force plate assessments, to establish its predictive and clinical utility.

11. Discussion

11.1 Conceptual Contribution of the Present Framework

The present article proposes the Fatigue-Induced Kinetic Chain Cascade as a unifying mechanical framework for understanding how fatigue alters movement in rotational sport. The central argument is that fatigue should not be conceptualized merely as a decrement in local force-generating capacity. Rather, it should be understood as a staged reorganization of kinetic-chain mechanics. This interpretation provides a bridge across several bodies of literature that are often discussed separately: trunk fatigue, spinal loading, segmental sequencing, and peripheral joint vulnerability.

11.2 Why the Framework Is Mechanically Useful

The FIKCC model is useful because it changes the level at which fatigue is interpreted. Rather than locating fatigue only in a muscle group or physiological metric, the framework locates it in the movement system's strategy of load regulation — a more biomechanically meaningful lens for rotational sport. A system-level view helps reconcile why athletes may retain isolated strength or skill capability while showing rising inconsistency, altered rhythm, or emerging joint-specific symptoms.

11.3 Key Implication: Mechanical Inefficiency Precedes Observable Performance Decline

A key implication of the FIKCC model is that mechanical inefficiency may precede observable performance decline. This challenges conventional fatigue models that rely primarily on output-based metrics and supports a shift toward movement-quality and coordination-based monitoring approaches. Clinicians, coaches, and performance scientists may therefore gain greater predictive value by tracking chain-level reorganization markers — such as pelvis–thorax phase relationships, trunk rescue patterns, and movement variability indices — rather than waiting for external output collapse or distal symptom onset.

11.4 Relevance to Rotational Sport Biomechanics

Rotational sports are an especially appropriate setting for this framework because they depend on efficient sequential transfer across multiple linked segments. Small disruptions in pelvic initiation,

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trunk stiffness regulation, or timing relationships can produce disproportionately large effects downstream. This may help explain why some athletes remain apparently competitive while moving into progressively less economical and less tissue-tolerant patterns — the body is willing to pay a greater internal price to maintain external output.

11.5 Implications for Research Design

The framework provides a clearer structure for future research. Rather than studying fatigue through isolated local variables, future work can test stage-linked predictions: whether proximal reserve markers change before sequencing metrics, whether sequencing metrics deteriorate before distal stiffness signatures, and whether distal overload markers are more likely when Stage II transfer inefficiency is already present. This staged logic encourages longitudinal and repeated-effort paradigms rather than single-time-point assessments.

11.6 Conceptual Strength, Necessary Caution, and Falsifiability

The FIKCC model is evidence-informed, not fully experimentally proven within a single unified protocol. Some stage transitions remain inferred from convergent evidence. The model would be weakened or challenged if future data consistently showed that: (1) distal overload emerges without any preceding proximal reserve loss or transfer disruption; (2) pelvic contribution decline does not alter thoracolumbar demand or sequencing quality; (3) athletes sustain efficient distal mechanics under fatigue despite substantial proximal control loss; or (4) stage-linked deterioration consistently occurs in a different order than proposed. These falsifiability conditions preserve the framework's scientific integrity.

From a clinical perspective, the FIKCC model may assist in identifying early indicators of movement dysfunction and guide targeted rehabilitation strategies focused on restoring proximal stability and coordination. From a performance standpoint, the framework provides a basis for monitoring fatigue-related mechanical changes and optimizing training interventions. Future research should aim to validate this model through experimental and longitudinal studies to establish its applicability across different athletic populations and movement tasks.

12. Limitations

- The evidence domains informing the framework arise from studies differing in sport type, participant level, fatigue protocol, instrumentation, and outcome variables. This heterogeneity limits direct one-to-one comparison across studies and was managed through convergent thematic synthesis rather than quantitative aggregation.
- The proposed stage transitions remain partly inferential. The exact temporal boundaries between Stage I, Stage II, and Stage III have not yet been established prospectively and should be understood as dominant mechanical tendencies rather than rigid categorical boundaries.
- The framework does not directly quantify spinal loading or tissue-level stress in vivo. Therefore, interpretations related to shear and compression behavior should be understood as mechanically inferred rather than directly measured. All references to spinal shear loading are presented as hypothesized mechanical relationships, not as confirmed biomechanical measurements.
- The framework is presently most developed for high-level rotational sequencing sports, particularly golf and analogous rotational athletic tasks. Sport-specific validation is essential before wider generalization across all rotational disciplines is justified.
- Several proposed monitoring markers remain hypothesized variables rather than validated diagnostic criteria. Clinical usefulness will depend on future work establishing reliability, responsiveness, and interpretive thresholds across populations.
- The FIKCC model emphasizes proximal-to-distal mechanical propagation but does not deny the importance of local factors. Prior injury history, anatomy, skill level, exposure volume, surface characteristics, equipment demands, and sport-specific technical habits may all modulate cascade expression and should be considered in applied interpretations.
- Because this paper is intentionally translational, some mechanical concepts are framed at a systems level rather than reduced to isolated variables. Individual subcomponents of the model must now be operationalized and tested experimentally.

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13. Future Research Directions

The primary strength of the FIKCC framework is that it generates a clear experimental agenda. Table 5 summarizes the priority research directions.

Table 5. Priority Future Research Directions Derived from the FIKCC Framework

Research Priority	Primary Research Question	Proposed Methodological Approach
Prospective stage validation	Does fatigue-related movement deterioration follow the proposed proximal-to-distal sequence?	Repeated-effort protocols with multi-segment kinematic and kinetic tracking across full session duration
Multi-segment time-series analysis	When and how do fatigue-related changes propagate across the kinetic chain?	Synchronized motion capture, IMU arrays, force platforms, and EMG across pelvis, trunk, thorax, limb segments
Spinal shear-compression redistribution	Does repeated rotational effort shift spinal loading from compressive toward shear-oriented behavior?	Musculoskeletal modeling with inverse dynamics under progressive fatigue; subject-specific spinal load estimation
Distal compensation signatures	Do distal joints show elevated stiffness signatures only after proximal sequencing deteriorates?	Sport-specific repeated-task protocols with distal force, stiffness, and joint excursion metrics
Preserved output vs mechanical cost	Can athletes maintain task output while internal mechanical cost and variability rise?	Dual-outcome monitoring: external performance metrics + internal biomechanical cost per repetition
Cross-sport validation	Does the cascade order generalize across rotational sports (golf, baseball, tennis, cricket)?	Comparative biomechanical studies with standardized fatigue protocols across sport populations
Sex, age, and training-status effects	Does the cascade unfold differently across demographic and developmental populations?	Longitudinal and cross-sectional comparison studies across novice/expert, youth/adult, sex-stratified groups
Intervention trials	Can targeted proximal interventions delay or interrupt cascade progression?	Randomized controlled trials: trunk endurance training, sequencing retraining, stage-specific load management
Computational and modeling approaches	Can musculoskeletal simulation predict cascade-linked load redistribution?	Subject-specific inverse dynamics, fatigue-state modeling, and computational joint load estimation

13.1 Prospective Stage Validation

Future studies should use repeated-effort or fatigue-exposure protocols capable of tracking changes in proximal trunk endurance and active stiffness behavior, pelvis–thorax sequencing, thoracolumbar load signatures, and distal stiffness or compensatory joint strategies. Such work should aim to determine whether the proposed sequence — proximal reserve loss followed by transfer inefficiency followed by distal compensation — is consistently supported across repeated-task environments.

13.2 Multi-Segment Time-Series Research

A major limitation of existing fatigue research is that many studies isolate one region or one variable. The FIKCC model requires multi-segment longitudinal analysis with synchronized measures across the pelvis, trunk, thorax, upper extremity, and distal joints to identify when and how fatigue-related changes propagate through the chain.

13.3 Shear-Compression Redistribution Under Fatigue

Future studies should examine how repeated rotational effort changes spinal load-sharing behavior, whether altered trunk stiffness regulation predicts greater translational demand, and whether segmental timing disruption is associated with measurable changes in estimated thoracolumbar shear-oriented loading. This area connects rotational sport performance research with spinal mechanics in a clinically meaningful way.

13.4 Distal Compensation Signatures

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Key research questions include: Do distal joints show increased stiffness signatures only after proximal sequencing deteriorates? Can late-session joint-risk patterns be predicted by earlier proximal markers? Does restoring proximal contribution reduce distal overload even when local tissue pain is present?

13.5 Performance Preservation Versus Mechanical Cost

Future research should test whether athletes can maintain ball speed, throw speed, or task success while sequencing deteriorates — and whether such preserved output is associated with higher movement variability, trunk rescue effort, or distal stiffening. This line of work has direct value for elite performance monitoring.

13.6 Cross-Sport and Population Adaptation

Although golf provides an excellent model system, future work should examine the framework across baseball, tennis, cricket, throwing tasks, combat striking, and rotational field sports. Sex-specific, age-related, developmental, and skill-dependent differences may influence how proximal reserve is lost and where compensation appears first.

13.7 Intervention Trials

The strongest validation of FIKCC will come from interrupting the cascade. Future trials should test whether targeted interventions — trunk endurance enhancement, rotational force-control training, fatigue-exposed sequencing drills, proximal stiffness-modulation training, and stage-specific load management — can delay progression from Stage I to Stage II or reduce Stage III distal compensation.

13.8 Computational Modeling

Musculoskeletal simulation, inverse dynamics, fatigue-state modeling, and subject-specific spinal load estimation may help test how changes in pelvic contribution or trunk control alter downstream mechanical demand — especially where direct in vivo quantification is difficult.

14. Conclusion

The present article introduced the Fatigue-Induced Kinetic Chain Cascade (FIKCC) as a conceptual mechanical framework for understanding how fatigue reorganizes movement in rotational sport. Rather than treating fatigue as a simple decline in muscular output, the FIKCC model proposes that fatigue progresses through a staged proximal-to-distal cascade beginning with declining proximal load-management reserve, advancing through coordination breakdown and thoracolumbar compensatory demand, and culminating in distal compensation and joint-specific vulnerability.

The principal contribution of this framework is integrative. It places trunk fatigue, force-vector drift, spinal loading changes, segmental sequencing disruption, and distal overload within one coherent chain-level model — offering a more complete explanation for why athletes may preserve visible performance while accumulating hidden mechanical cost, why symptoms may appear distally after upstream deterioration has already begun, and why fatigue monitoring should move beyond workload quantity toward movement-system organization.

The FIKCC framework is not presented as finalized doctrine. It is presented as a testable biomechanical architecture whose value now depends on whether future research confirms, refines, or challenges its proposed stages, markers, and mechanistic links. Even in its current conceptual form, the model provides an important translational lens — encouraging researchers to study fatigue as a chain-level transfer problem, clinicians to interpret distal symptoms in the context of upstream fatigue mechanics, and coaches to recognize that preserved output does not always mean preserved efficiency.

Fatigue in rotational sport is not merely a reduction in force capacity. It is a progressive reorganization of kinetic-chain mechanics, with measurable consequences for performance, coordination, and tissue-level load distribution.

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