

# A Theoretical Framework Including The Kirkaldy-Willis Model And The Three-Joint Complex For The Degenerative Cascade Of The Lumbosacral Spine

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## Abstract

**Background:** One of the main causes of disability worldwide is low back pain (LBP), which is mostly caused by degenerative changes in the lumbosacral area. Even though magnetic resonance imaging (MRI) is the gold standard for diagnosis, there is frequently a theoretical gap in the relationship between specific imaging results and the overall course of spinal deterioration.

**Objective:** To create a thorough theoretical framework for the Degenerative Cascade by combining the Kirkaldy-Willis pathophysiological model with the biomechanical "Three-Joint Complex" hypothesis, particularly in the context of radiological evaluation.

**Theoretical Framework:** The interdependence of the paired facet joints and the intervertebral disc is integrated in this work. Pfirrmann and Weishaupt grading systems are used as markers to chart the development from biochemical dysfunction to mechanical instability and finally biological stabilization.

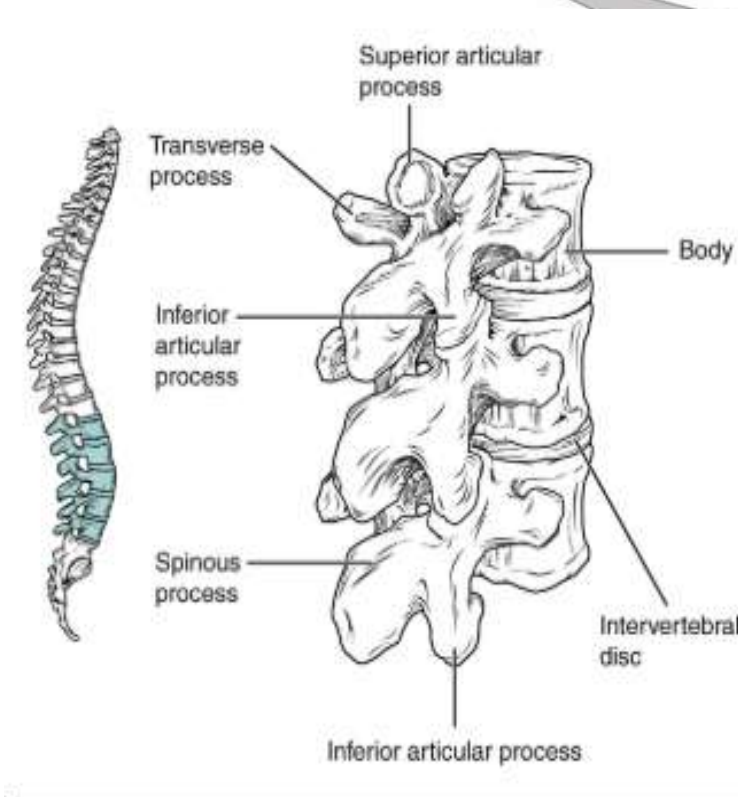
**Conclusion:** The degenerative process is a predictable cascade that is not linear. The "Clinical-Radiological Paradox" requires a correlation-based approach for management planning, while MRI offers the granularity required to pinpoint the precise stage of this cascade.

**Keywords:** Lumbosacral Spine, Degenerative Cascade, Three-Joint Complex, Spinal Stenosis, and Facet Joint Arthropathy.

## 1. Introduction

One of the most common musculoskeletal conditions in the world, low back pain affects people of all demographic backgrounds and is a major contributor to years spent disabled <sup>[1,2]</sup>. Because the L4–L5 and L5–S1 segments of the lumbosacral spine endure the majority of human axial stress and mobility, they are particularly vulnerable to wear and tear <sup>[3,4]</sup>. Degenerative spine disease is a progressive, multi-phasic disintegration of the spinal structures, including the facet joints, intervertebral discs, and supporting ligaments <sup>[5,6]</sup>. Because of its improved soft-tissue contrast and lack of ionizing radiation, magnetic resonance imaging (MRI) has become the gold standard in modern radiology for evaluating these changes <sup>[7,8]</sup>.

However, the interpretation of MRI results frequently takes place in a vacuum, concentrating on discrete anomalies such as "disc bulge" or "facet hypertrophy" <sup>[9]</sup>. The "Degenerative Cascade," a model first made popular by Kirkaldy-Willis that explains the successive stages of spinal degeneration, is the theoretical framework that this research suggests using to interpret these data. By incorporating the biomechanical idea of the "Three-Joint Complex" <sup>[11]</sup>, we may gain a clearer understanding of how a failure in one component, like disc desiccation, invariably triggers disease in other components, resulting in the intricate clinical presentations seen in individuals with symptoms <sup>[12,13]</sup>.



**Figure 1:** Facet Joints (Reference: McMINN. Lasts Anatomy Regional and Applied. CHURCHILL LIVINGSTONE).

## 2. THEORETICAL FRAMEWORK: THE THREE-JOINT COMPLEX

The "motion segment," which is conceptualized as a Three-Joint Complex in theory, is the basic functional unit of the lumbosacral spine<sup>[11,14]</sup>. The two posterior synovial facet joints and the sizable anterior intervertebral disc make up this complex<sup>[11,15]</sup>. This "tripod" configuration permits flexion, extension, and rotation while providing biomechanical stability<sup>[16]</sup>.

The Three-Joint Complex functions in a state of synergy, according to the theoretical framework first put forward by Kirkaldy-Willis and subsequently developed by others<sup>[11,17]</sup>. The intervertebral disc distributes loads uniformly and serves as the main shock absorber in a healthy state<sup>[18]</sup>. However, disc height decreases as disc degeneration sets in, which is marked by a loss of proteoglycans and water content<sup>[19]</sup>. The mechanical load is transferred posteriorly onto the facet joints as a result of this height reduction<sup>[20]</sup>. This increased stress causes rapid articular wear, cartilage thinning, and ultimately hypertrophy because the facet joints are not intended for primary weight-bearing<sup>[21, 22]</sup>.

This theoretical connection explains the statistical correlation between facet joint arthropathy and radiographic indications of disc degeneration (evaluated by the Pfirrmann scale)<sup>[23,24]</sup>. When the front "leg" of the tripod fails, the posterior "legs" must compensate, starting a series of anatomical changes that characterize the aging spine<sup>[25]</sup>.

## 3. PATHOPHYSIOLOGICAL STAGES OF THE DEGENERATIVE CASCADE

The Degenerative Cascade is traditionally divided into three sequential clinical and radiological stages: Dysfunction, Instability, and Stabilization<sup>[10, 26]</sup>.

### 3.1 The Stage of Dysfunction

Biochemical changes in the disc and mild facet joint synovitis are the hallmarks of this early stage<sup>[10,27]</sup>. Disc desiccation, a loss of high T2 signal intensity in the nucleus pulposus (Pfirrmann Grade II–III)<sup>[23]</sup>, is how this appears on MRI. The disc starts to lose its capacity to transfer hydrostatic pressure, resulting in tiny circumferential tears in the annulus fibrosus, even if structural integrity is mostly maintained<sup>[28]</sup>.

### 3.2 The Stage of Instability

Segmental laxity results from the cumulative effect of annular tears and disc height loss as the cascade advances <sup>[10,29]</sup>. The motion segment moves abnormally or excessively during this "Instability" phase <sup>[30]</sup>. Disc protrusions, internal disc disturbance, and early facet joint subluxation are radiological indicators of this <sup>[22,31]</sup>. Due to temporary nerve root irritation, patients are most likely to have acute episodes of low back pain and radicular symptoms during this phase <sup>[32]</sup>.



**Figure 2:** MRI demonstrating L4-L5 and L5-S1 degenerative disc disease with height loss and desiccation  
(Reference:DSc SSP. Gray's  
Anatomy. Churchill Livingstone. (2011) ISBN:0443066841.)

### 3.3 The Stage of Stabilization

The body starts hypertrophic modifications as a biological attempt to restore stability <sup>[10,33]</sup>. Large osteophytes, facet joint hypertrophy, and ligamentum flavum thickening (hypertrophy) are also part of this latter stage <sup>[5,34]</sup>. These alterations simultaneously intrude on the spinal canal and neural foramina, even while they successfully "stiffen" the motion segment and may lessen the pain related to instability <sup>[35]</sup>. This stage, which often presents as neurogenic claudication in the elderly, is conceptually associated with the development of central and foraminal stenosis <sup>[36]</sup>.

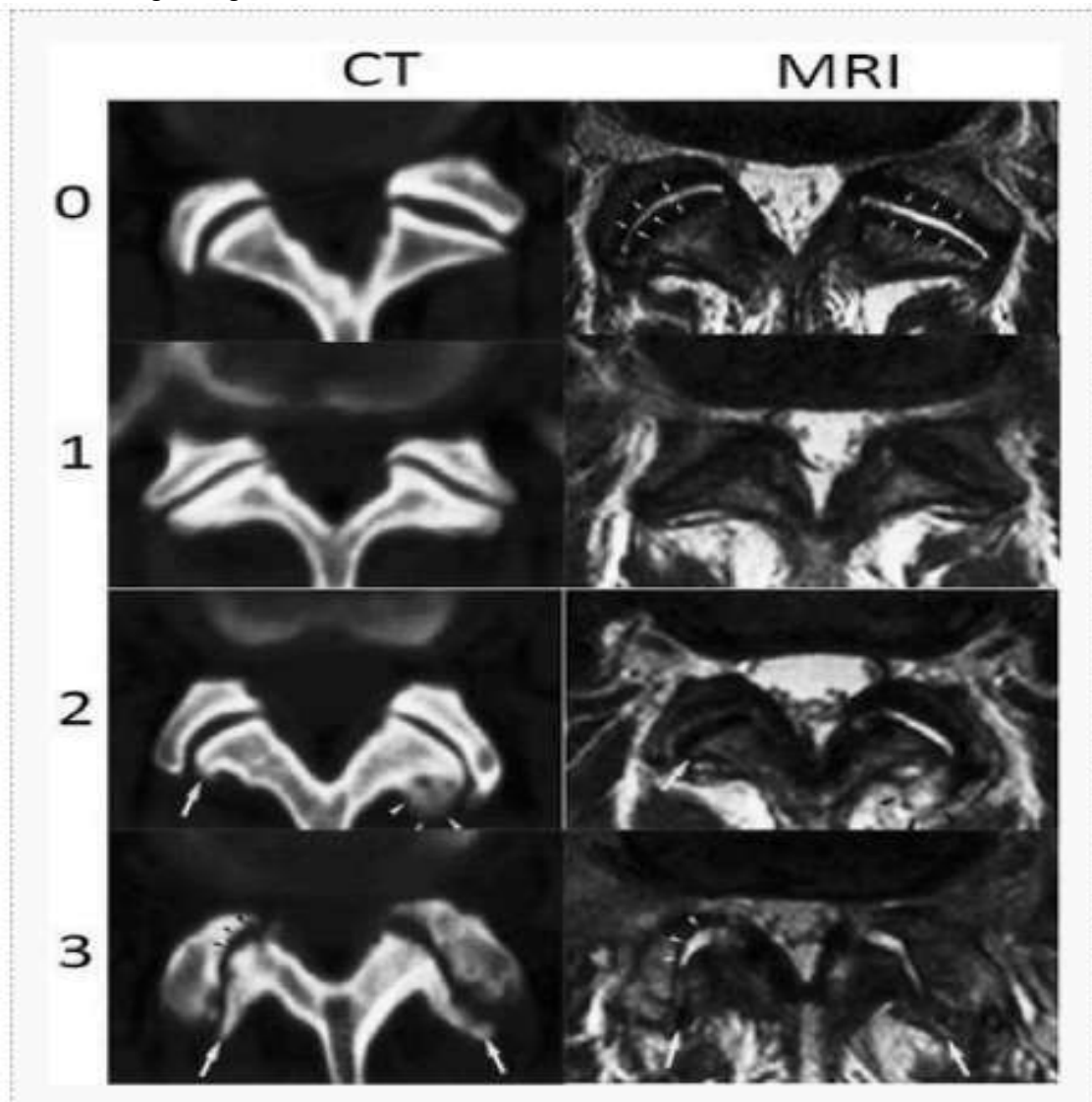
## 4. RADIOLOGICAL MARKERS OF THEORETICAL STAGES

Radiologists use standardized grading systems to precisely map a patient's location within the degenerative cascade <sup>[23,37]</sup>. These systems provide theoretical constructs an objective language <sup>[2]</sup>.

**Table 1:** Integration of Radiological Grading with the Degenerative Cascade

| Cascade Stage        | Intervertebral Disc (Pfirrmann Grade)                                | Facet Joint (Weishaupt Grade)                         | Ligamentous/Canal Changes                |
|----------------------|----------------------------------------------------------------------|-------------------------------------------------------|------------------------------------------|
| <b>Dysfunction</b>   | Grade II: Clear nucleus/annulus distinction, high signal.            | Grade 0: Normal joint space (2-4 mm)                  | Minimal or no thickening.                |
| <b>Instability</b>   | Grade III-IV: Blurred distinction, intermediate signal, height loss. | Grade 1-2: Narrowing (<2mm), small osteophytes.       | Early Ligamentum Flavum thickening       |
| <b>Stabilization</b> | Grade V: Black disc, collapsed space, no signal.                     | Grade 3: Large osteophytes, severe hypertrophy, cysts | Severe Stenosis, Nerve root compression. |

The most dependable method for determining the extent of disc degeneration is still the Pfirrmann grading system <sup>[23,24]</sup>. Simultaneously, the inflammatory and fatty replacement phases of the cascade are indicated by Modic changes, which are variations in signal intensity in the vertebral endplates <sup>[38]</sup>. Type 1 (inflammatory) Modic alterations are frequently linked to the shift from dysfunction to instability, whereas Type 2 (fatty) modifications are more stable and long-lasting <sup>[39,40]</sup>.



**Figure 3:** Expression of adiponectin in the subchondral bone of lumbar facet joints with different degrees of degeneration (Reference <https://www.researchgate.net/figure/The-Weishaupt-grade-of-CT-and-MRI-images-of-the-lumbar-facet-joint-Grade-0-normal-facet>)

## 5. THE CLINICAL-RADIOLOGICAL PARADOX

The lack of a linear relationship between the severity of imaging findings and clinical symptoms—a problem known as the "Clinical-Radiological Paradox"—presents a major theoretical issue in spine radiology<sup>[12,41]</sup>. While some patients with crippling pain only have minor "Dysfunction" stage signs, many asymptomatic people have substantial Pfirrmann Grade IV disc degeneration or facet arthropathy<sup>[13,23]</sup>.

Individual differences in pain thresholds, the existence of chemical radiculitis (inflammation without substantial mechanical compression), and the psychosocial components of chronic pain are some theoretical explanations for this discrepancy<sup>[42,43]</sup>. Although the "cascade" is a universal anatomical process, the precise spatial interaction between degenerative tissues and neural systems determines how it manifests clinically<sup>[44]</sup>.

## 6. CONCLUSION

The degenerative alterations of the lumbosacral spine are components of an organized pathophysiological sequence called the Degenerative Cascade rather than isolated events<sup>[10]</sup>. Disc degeneration is the result of using the Three-Joint Complex hypothesis as the *primum movens*<sup>[11]</sup>, which alters the biomechanics of the spine and ultimately calls for ligamentous thickening and facet joint hypertrophy as compensatory stabilizing measures<sup>[5,34]</sup>.

The key instrument for staging this cascade is magnetic resonance imaging (MRI), which enables the detection of early dysfunction through signal loss and late stabilization through stenotic indicators<sup>[7,8]</sup>. However, clinical correlation must always be used to balance the theoretical framework<sup>[12]</sup>. These changes will become more common as the population ages, thus uniform reporting of these stages—rather than just isolated findings—is essential for efficient management and surgical planning<sup>[9,41]</sup>. In order to detect the "Stage of Dysfunction" even earlier and possibly enable preventative interventions before the cascade reaches the irreversible "Stabilization" phase, future research should concentrate on sophisticated MRI sequences, such as T2 mapping<sup>[45]</sup>.

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