It has been said that the definition of insanity is doing the same thing over and over again, while expecting a different result. Studies dating back several decades [e.g., the article by Boden et al. (1)] have failed to show strong correlation between abnormal magnetic-resonance scans of the intervertebral discs and clinical symptoms. Nonetheless, as a group, we keep trying to make the connection, both in the clinic and in the lab. The recently published 20-year prospective longitudinal study of cervical spine disc degeneration by Daimon et al. (2), is perhaps the strongest confirmation to date affirming that intervertebral discs naturally degenerate with age, and that evidence of degeneration alone is insufficient information with which to make a conclusion regarding the root cause of a patient’s symptoms.

Our most commonly used diagnostic tool for identifying the underlying cause of chronic neck pain continues to be T1, T2, and Turbo spin echo (Fast spin echo) MRI imaging of the intervertebral discs. These tools are commonly available, and helpful in identifying gross structural dysfunction. However, Daimon et al.’s study highlights that these imaging protocols fail to distinguish between symptomatic and asymptomatic patients. Other MR imaging protocols such as ultra-fast time to echo (UTE) (3), T1ρ (4), diffusion imaging, sodium imaging and MR spectroscopy provide superior differentiation of clinically relevant features of the disc and surrounding structures. Some of these modalities also contain information in regards to nutritional state (5) and metabolomics, that may improve both diagnosis and eventually outcomes. However, these imaging modalities have not yet made their way into common clinical practice.

Daimon et al. found that while MRI signal intensity longitudinally decreases across all cervical disc levels, there is a peak in structural degeneration that occurs at the C5–C6 level, with C4–C5 and C3–C4 having progressively lower degeneration rates. Since the C5–C6 level also corresponds with the highest flexion-extension range of motion of the cervical spine, a mechanical component of the degeneration process appears to be highlighted by the study. Once the C5–C6 level has been destabilized due to degeneration, sequential acceleration of degeneration at adjacent levels was observed. This insight has relevance to current discussions regarding adjacent-segment disease subsequent to arthrodesis and arthroplasty.

The authors also observed that 95% of subjects experienced degenerative progression over the 20-year study period, while only 67% developed clinical symptoms. This observation lends strength to the argument that trying to fight all forms of disc degeneration is an insolvable fight against nature, at least for the foreseeable future. Switching to a narrower focus on distinguishing pathological (i.e., pain-inducing) degeneration from asymptomatic disc degeneration represents the more impactful short-term win. The discrepancy between the virtually universal observation of degeneration, versus the smaller symptomatic group also brings up the likelihood that we are missing critical insights from other spinal structures with nociceptive innervation (6-10), which are less-easily imaged but may differentiate the symptomatic patients from the broader asymptomatic...
As a biomechanist, I would be remiss to point out that imaging alone is missing fundamental information regarding the dynamic function of the spine. Spines that look very similar while lying down in the MRI may move very differently while going about activities of daily living—and the consequences can be dramatic for mechanical loading and pain in the discs and adjacent spinal structures (11,12). Efforts towards establishing a “mechanome” (13) for the cells of the spine (or for the broader tissues as a whole), could differentiate healthy motion from destructive or painful motion and yield benefits in the clinic. Of course, none of these insights is particularly new (14-16). Perhaps it is simply another form of repetitive insanity to keep stating it. It requires time and resources to identify and develop alternative diagnostic tools. We’ve made quite a bit of progress over the last 20 years in our understanding of spine function and dysfunction, but we still have far to go. Hopefully, a new way of doing things is around the corner.

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Footnote

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References


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