Editorial

# Twenty years of 'insanity' in diagnosing underlying clinically relevant cervical dysfunction using traditional MRI

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*Comment on:* Daimon K, Fujiwara H, Nishiwaki Y, *et al.* A 20-Year Prospective Longitudinal Study of Degeneration of the Cervical Spine in a Volunteer Cohort Assessed Using MRI: Follow-up of a Cross-Sectional Study. J Bone Joint Surg Am 2018;100:843-9.

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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

population.

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

*Conflicts of Interest:* The author has no conflicts of interest to declare.

## References

1. Boden SD, McCowin PR, Davis DO, et al. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am 1990;72:1178-84.
2. Daimon K, Fujiwara H, Nishiwaki Y, et al. A 20-Year Prospective Longitudinal Study of Degeneration of the Cervical Spine in a Volunteer Cohort Assessed Using MRI: Follow-up of a Cross-Sectional Study. J Bone Joint Surg Am 2018;100:843-9.
3. Pang H, Bow C, Cheung JPY, et al. The UTE Disc Sign on MRI: A Novel Imaging Biomarker Associated With Degenerative Spine Changes, Low Back Pain, and Disability. Spine (Phila Pa 1976) 2018;43:503-11.
4. Paul CPL, Smit TH, de Graaf M, et al. Quantitative MRI in early intervertebral disc degeneration: T1rho correlates better than T2 and ADC with biomechanics, histology and matrix content. PLoS One 2018;13:e0191442.
5. Urban JP, Smith S, Fairbank JC. Nutrition of the intervertebral disc. Spine (Phila Pa 1976) 2004;29:2700-9.
6. Lotz JC, Fields AJ, Liebenberg EC. The role of the vertebral end plate in low back pain. Global Spine J 2013;3:153-64.
7. Von Forell GA, Bowden AE. Biomechanical implications of lumbar spinal ligament transection. Comput Methods Biomech Biomed Engin 2014;17:1685-95.
8. Robertson DJ, Von Forell GA, Alsup J, et al. Thoracolumbar spinal ligaments exhibit negative and transverse pre-strain. J Mech Behav Biomed Mater 2013;23:44-52.
9. Singh S, Kartha S, Bulka BA, et al. Physiologic facet capsule stretch can induce pain & upregulate matrix metalloproteinase-3 in the dorsal root ganglia when preceded by a physiological mechanical or nonpainful chemical exposure. Clin Biomech (Bristol, Avon) 2018. [Epub ahead of print].
10. Odonkor CA, Chen Y, Adekoya P, et al. Inciting Events Associated With Lumbar Facet Joint Pain. Anesth Analg 2018;126:280-8.
11. Von Forell GA, Stephens TK, Samartzis D, et al. Low Back Pain: A Biomechanical Rationale Based on "Patterns" of Disc Degeneration. Spine (Phila Pa 1976) 2015;40:1165-72.
12. Bowden JA, Bowden AE, Wang H, et al. In vivo correlates between daily physical activity and intervertebral disc health. J Orthop Res 2018;36:1313-23.
13. Song MJ, Brady-Kalnay SM, McBride SH, et al. Mapping the mechanome of live stem cells using a novel method to measure local strain fields in situ at the fluid-cell interface. PLoS One 2012;7:e43601.
14. Iatridis JC, Kang J, Kandel R, et al. New Horizons in Spine Research: Disc biology, spine biomechanics and pathomechanisms of back pain. J Orthop Res 2016;34:1287-8.
15. Hughes SP, Freemont AJ, Hukins DW, et al. The pathogenesis of degeneration of the intervertebral disc and emerging therapies in the management of back pain. J Bone Joint Surg Br 2012;94:1298-304.
16. Winkelstein BA, Weinstein JN. Pain Mechanisms: Relevant Anatomy, Pathogenesis and Clinical Implications. In: Rlark C. editor. The Cervical Spine. Philadelphia, Lippincott Williams & Wilkins, 2004:122-32.

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