Human Cartilage Endplate Compressive Modulus Is Altered by Degeneration and Is Location-Dependent

John F. DeLucca, Daniel H. Cortes, Ph.D., Randall L. Duncan, PhD, Dawn M. Elliott, PhD.
University of Delaware, Newark, DE, USA.


Introduction: The cartilage endplate (CEP) is a thin layer of hyaline cartilage located between the nucleus pulposus (NP) and the vertebral endplate that functions to contain the NP, transmit compressive loads, and control water and nutrient diffusion into the disc. Changes in these biomechanical functions may contribute to a CEP-driven class of degeneration [1]. While degenerative changes in the human NP and the annulus fibrosus are well documented, human CEP mechanics and changes with degeneration have not been measured. Further, the prevalence of CEP pathology is not symmetric. For example, Schmorl’s nodes (herniation through the vertebral endplate) occur more frequently across the superior CEP and in the lower thoracic. Therefore, the objective of this study was to quantify the compressive modulus of the human CEP as a function of disc and spinal location and how this modulus changes with degeneration.

Methods: Human spine segments (n=16) were acquired, imaged with MRI, and graded for degeneration with the Pfirrmann scale. Healthy discs were defined by grades of 1-2 and degenerate discs were defined by grades of 3-5. Superior and inferior endplates were removed from lower thoracic (T7-T12, n=7) and lumbar (L1-L4, n=20) vertebra and any remaining NP was removed. The central CEP was sectioned using a microtome on a freezing stage to create two parallel flat surfaces and punched to create cylindrical samples 4 mm diameter by 0.84 mm +/- 0.17 mm thick. The sample was tested in confined compression with the CEP side that was adjacent to the vertebral body placed in contact with the porous platen. A tare load of 0.1 N was applied to hold the sample in place and the sample was swelled to equilibrium in a bath of 0.15 M PBS. Samples were compressed at a quasi-static rate of 1%/min to 5%, 10%, and 15%, then held until equilibrium. The equilibrium modulus was calculated as the change in stress divided by the change in applied strain at each increment. The effect of degeneration and effect of thoracic versus lumbar was analyzed using a two-way ANOVA, with Bonferroni post-hoc comparisons. The relationship between superior and inferior modulus was analyzed using linear regression against a slope of 1. Significance was set at p<0.05. Adjacent CEP samples to those tested in confined compression were dehydrated and analyzed using a 1,9-dimethylmethylene blue assay to measure water and sulfated glycosaminoglycan content.

Results: The CEP modulus in degenerated discs was 2X greater than that of healthy discs for inferior endplates (p < 0.05), while degeneration did not alter the modulus in superior endplates (Figure 1A). Lower thoracic CEP equilibrium modulus was 30-50% larger than lumbar CEP equilibrium modulus (p < 0.0005; 1000-1820kPa for thoracic CEP; Figure 1B). The modulus of the superior CEP was linearly correlated with the inferior CEP with a slope of 0.73, significantly different than a slope of 1 (p = 0.05; Figure 1C). The superior CEP modulus was 50% larger than the inferior. There was no significant relationship of modulus with either water or glycosaminoglycan.

Discussion: Compressive mechanical properties of the human cartilage endplate were measured for the first time. Human CEP modulus (0.6 - 2 MPa) was consistent with reported ranges for articular cartilage (0.6-0.85 MPa) and baboon CEP (0.44 MPa) [2,3]. While the physiological consequences of the higher modulus with degeneration and the differential CEP modulus in the thoracic versus lumbar and the superior versus inferior CEP remain to be determined, these differences could affect cell-level stresses and consequent mechanobiologic response. These mechanical property differences can also be considered in light of the damage and failure of the CEP, including the higher prevalence of Schmorl’s nodes in the lower thoracic region compared to the lumbar region and in the superior CEP compared to the inferior endplate [4]. While the specific mechanisms from changes in sub-failure moduli to damage and failure are unknown, they are almost certainly related. The location-specific mechanical properties of the CEP are consistent with, and likely related to, the mechanics of the vertebral endplate [5]. No significant relationship was found between glycosaminoglycan and CEP modulus, suggesting that the collagen content, the minor constituents, or the structural organization of the constituents may contribute to differences in mechanical response observed here.

Significance: The functional role of the CEP to distribute disc loads and contain the NP has qualitatively been understood but has not been explored quantitatively. The CEP mechanics were dependent on the location within the disc and spinal level, both conditions which are consistent with the location- and region-specific nature of endplate damage in the form of Schmorl’s nodes. The damage associated with a Schmorl’s node may alter the load-bearing and structural roles the CEP plays in disc function.

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Figure 1: Compressive equilibrium modulus of the human cartilage endplate was measured. (A) Interior CEFPs are more greatly impacted by degeneration than surface CEFPs. Interior endplate modulus increases 122% with degeneration. (B) Modulus of CEFPs of the lower lumbosacral region was 509% greater than in the lumbar regions (503%) applied strain. (C) Equilibrium compressive modulus varies linearly with strain and is significantly different than a slope of 1 (p<0.05).

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