

Mechanical Influences in Disc Degeneration

Michael A. Adams, Department of Anatomy, University of Bristol, Southwell St, BS9 8EJ, U.K.
M.A.Adams@bris.ac.uk.

INTRODUCTION It is important to establish what intervertebral “disc degeneration” really is, and to distinguish it from normal ageing. We suggest that disc degeneration should be defined as a cell-mediated response to gross structural disruption. Such disruption is an easily-detected, unambiguous marker of impaired disc function, which does not occur inevitably with increasing age, and which is more closely related to pain than any other feature of ageing discs¹. Structural disruption is irreversible, because adult discs are incapable of repairing gross defects. Furthermore, it naturally *progresses*, by physical and biological mechanisms, and so is a suitable marker for a degenerative process.

MECHANICAL INFLUENCES Certainly, this definition simplifies the issue of causality: excessive mechanical loading disrupts a disc’s structure and precipitates a cascade of non-reversible cell-mediated responses which lead to further disruption. Cadaveric experiments and mathematical models have shown how various combinations of compression, bending, and torsion can cause all of the major structural features of disc degeneration, including endplate defects, radial fissures, radial bulging, disc prolapse, and internal collapse of the annulus². Damage can be created by a single injury, or by wear-and-tear “fatigue” loading. Animal experiments confirm that structural disruption to disc or endplate *always* leads to cell-mediated degenerative changes³.

OTHER INFLUENCES: Other features of degenerated discs can be considered as predisposing factors for, or consequences of, structural disruption. Genetic inheritance and impaired metabolite transport can make the disc matrix physically weaker and so vulnerable to injury. So too can age-related changes in collagen cross-linking, and loss of water and proteoglycans from the nucleus. Elevated levels of cytokines and MMP’s may represent attempted repair⁴, as in other connective tissues, and they could be triggered by the abnormal matrix stresses which follow structural

disruption. Ingrowth of blood vessels and nerves doubtless represent a late consequence of altered mechanics and biochemistry in severely disrupted tissues. Defining disc degeneration in terms of structural disruption therefore leads to a simple conceptual framework which incorporates all known features of degenerated discs.

DISCUSSION It is important to realise that “excessive” loading does not mean high loading. The fact that disc degeneration is common even among sedentary people with no history of spinal injury suggests that an unfavourable inheritance, middle age, inadequate metabolite transport and accumulating fatigue damage can weaken some discs to such an extent that physical disruption occurs during the activities of daily living. This speculation is supported by the very wide range of tissue strengths reported in cadaveric experiments.

CONCLUSIONS Disc degeneration should be defined as a cell-mediated response to progressive structural disruption. The underlying cause is tissue weakening arising primarily from genetic inheritance, ageing, and nutritional compromise. The precipitating cause is structural disruption arising from injury or fatigue failure.

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