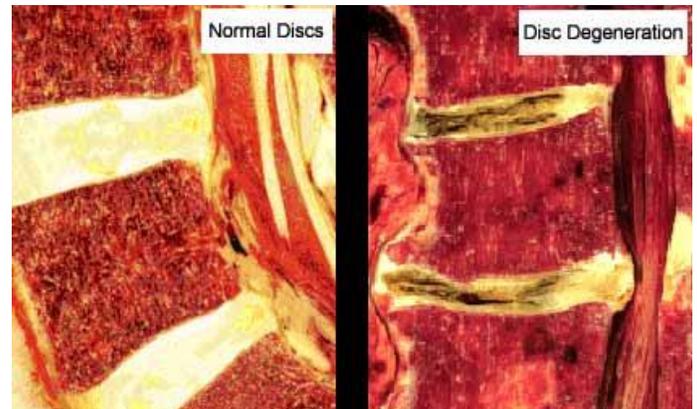


## General Information on DDD and Disc Aging:

Unlike other tissues of the human body, the poorly vascularized intervertebral disc tends to undergo degeneration of its internal structure at a surprisingly early age. In fact, award-winning research has demonstrated such degeneration usually begins within the first decade of life! Thankfully, most of the time this degeneration is harmless and considered just part of the natural aging process. However, in some folks, the degeneration process runs amuck and spells the proverbial "beginning of the end" for the disc as it leads to destruction of the disc and chronic pain. Why some people's discs succumb to such severe disc failure (above right) and pain and others don't continues to be a mystery, although heredity certainly plays a significant role in this process. It is this abnormal accelerated form of pathological disc aging and degeneration that I called Degenerative Disc Disease (**DDD**).

*The figures to the right demonstrate the difference between normal (left) and severely degenerated (right) lumbar discs. Note the moderate to severe loss of disc height (right); a conditions that is called discopathy or (when in combination with arthritic change) discogenic spondylosis. The loss of disc height often causes the secondary problem of stenosis: the closing of the holes where the spinal nerves, nerve roots and spinal cord reside.*



One physical cause of DDD, and natural disc aging for that matter, is poor disc nutrition. The majority of disc tissue has no blood supply (it's avascular). The disc gets its food (nutrients) via diffusion from blood within the upper and lower vertebral endplates. Normally, the nutrients pass from the endplates through tiny pores the into the hungry discs. Arthritis of the endplate (sclerosis) has the negative effect of decreasing the diameter of these pores, which in turn decreases the rate of food-flow to the disc, which in turn causes the disc to die, i.e., DDD. In fact, recent award winning scientific investigations have confirmed the foregoing explanation of disc degeneration.

Therefore, any attempts at injecting new disc material into a degenerated disc (such as live disc cells)--in hope of healing the damaged disc--will only lead to failure, for the new disc material will soon meet the same fate of the original disc tissue: it will starve to death.

Research has also demonstrated that DDD is associated with pain-producing conditions of the spine such as annular tears, disc protrusions and spinal stenosis. In fact, in 10% of the population, DDD will result in permanent chronic pain and life-long disability. Technically it's not the actual process of DDD that results in chronic pain, it's the evil "end-phases" of the disease that cripples these unfortunate few. These end-phases include annular tears (aka: Internal Disc Disruption or IDD); disc protrusions; nerve in-growth ; and stenosis.

### The MRI Appearance:

The diagnosis of DDD is best made on T2-weighted MRI imaging (Fig.#1), although some of the late appearances of DDD (disc collapse, osteophytosis, and sclerosis) may also be seen on CT scan and X-ray. Such MRI appearances are easy to spot and are characterized by a loss of signal intensity (loss of whiteness) of discal tissue, which makes the disc appear **black** instead of bright white. Technically, this blackening of the disc(s) occurs because the disc has dehydrated (lost water content) and is dying. This 'blackening' is called disc **Desiccation**. Since the MRI signal intensity (whiteness) is directly related the disc's water content, any loss of discal water will proportionally decrease the 'whiteness' of that disc on T2-weighted MRI. So, in layman's terms, the dryer the disc, the blacker and more degenerated it will look on MRI.

Figure #1: Here is the classic presentation of DDD as seen in this T2-weighted sagittal (lateral) image. Note the bright white and healthy L3 disc (above the L4 vertebra) in comparison to the 'black' and desiccated L4 and L5 disc. Also note a 4mm herniation at the L4 disc (between L4 and L5 vertebrae) and a 9mm herniation at the L5 disc. Also note there is a loss of disc height at both L4 and L5, in comparison to the thicker L3 disc.

Why some discs prematurely degenerate (DDD) and cause chronic pain and others don't is still somewhat controversial, however, it is becoming clearer that poor genetics; a past history of moderate to severe spinal trauma; or have an occupation that is heavy, and labor-intensive are the main risk factors. These factors will be discussed in depth below.

