Haughton et al. ([i]) postulated that disc contents leaking out through a torn annulus may have an inflammatory effect.

They found that the contents of the central part of the disc, the nucleus pulposus, chondroitin (a component of the disc) and lactic acid produced by processes within the disc could cause inflammation, as could synovial fluid from degenerating facet joints.

Looking at monkeys, the team found that nucleus pulposus produced "significant fibrosis in the arachnoid and epidural spaces."

The authors therefore suggested that this was a factor in the inflammatory response.

Saal ([ii])described the high levels of the inflammatory enzyme phospholipase A2 present in herniated or degenerating discs and the consequent discogenic pain.

Goupille et al. in 1998 ([iii]) suggested that involvement of inflammatory mediators in causing radiculopathy had yet be proven, but suggested a hypothesis that leakage of inflammatory agents such as prostaglandins and interleukins may produce an excitation of nociceptors, a direct neural injury, nerve inflammation or enhancement of sensitisation to other pain-producing substances (such as bradykinin).

Although these authors believe this effect to be transitory as a part of the early stage of disc herniation, it seems quite feasible that these inflammatory processes may in susceptible individuals become prolonged and progress to chronic problems.

It may be that, as Frank and Mayfield suggested ([iv]) the immune capabilities of the arachnoid membrane which were demonstrated in vitro are responsible for initiating and maintaining an inflammatory response to the presence of disc material.

[i] Haughton VM, Nguyen CM, Ho KC. *Spine* 1993 Jul; 18(9):1193-8 The etiology of focal spinal arachnoiditis. An experimental study.

[ii] Saal JS *Spine* 1995; 20 (16): 1821-1827 The Role of Inflammation in Lumbar Pain

[iii] Goupille P, Jayson MI, Valat JP, Freemont AJ. *Semin Arthritis Rheum* 1998 Aug; 28(1):60-71 The role of inflammation in disk herniation-associated radiculopathy.

[iv] Frank F, Mayfield F, Presented at 1982 Congress of Neurological Surgeons Annual Meeting