Chapter 8 Minimal Invasive Posterior Dynamic Stabilization: A New Treatment Option for Disc Degeneration (Yoda)

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Introduction

Background

Disorders related to intervertebral disc (IVD) degeneration are widespread causes of morbidity and severe life quality deterioration. In particular, IVD degeneration is a major cause of neck and low back pain (LBP), affecting a large percentage of the population at some point in their lives [1]. The lifetime prevalence of LBP is 70–80 %, and approximately 18 % of the population is suffering from LBP at any time leading to enormous costs due to treatment and work absenteeism [2]. With regard to treatment modalities, there is still an ongoing debate among spine specialists, which patients should be selected for surgical treatment and which operative intervention is superior. Various nonsurgical treatment regimens have shown satisfactory results, especially in short term, but in severe chronic LBP and patients with advanced disc degeneration and segmental instability, studies have shown that fusion is more efficient in reducing pain and disability [3, 4]. Although with modern implants high fusion rates can be achieved [5], one must consider that besides the high costs, approximately every fifth patient requires reoperations in the long term,

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often due to adjacent level disease that develops as a consequence of altered biomechanical stresses and is seen in radiographs of every third patient after fusion [5-8]. Disc arthroplasty and dynamic stabilization techniques have evolved as a result of this frequent complication with the hope that this technology can prevent degeneration of adjacent segments, but up to date the benefit of these newer procedures could not be demonstrated [9, 10]. Although these treatment options are effective in short term, they are associated with high costs and long-term problems and are reserved for advanced degenerated segments only; therefore it would be beneficial to start treatment at an earlier stage of the disc degeneration cascade, prior to the loss of mechanical function and visible segmental instability. These early stages are within the scope of "biological" therapies where regeneration of degenerative changes should be achieved by application of growth factor, gene, or cell therapies. Despite intensive research and promising early results in vitro or in animal models, such regenerative therapies are far from clinical application and might be restricted to very early stages of disc degeneration [11-15]. In the short term there is, besides physical therapy, no adequate treatment option for patients with discogenic low back pain at early stages of disc degeneration that would prevent further deterioration of the disease. This is the background for the YODA concept presented in this chapter, where a minimal invasive technique is investigated with the intention to mechanically stabilize the segments and prevent further degradation resulting from micromotion.

Etiology of Disc Degeneration

The etiology of IVDD is multifactorial, and degenerative changes can be observed to some extent in a majority of adult IVDs [16, 17]. It has been suggested that IVD degeneration may mimic age-related changes but occurs at an accelerated rate [18, 19]. Apart from environmental and biomechanical reasons, genetic predisposition plays a major role in the development of IVD and explains over 70 % of variance in IVDD; these genes at risk are associated with various structures and functions of healthy IVDs as macromolecules (collagens, aggrecans), enzymatic activity, cell senescence, and more [20, 21]. However, the most relevant factor in IVD biology and early degeneration is the limited nutrition as the human IVD is the largest avascular structure in the human as the blood vessels in the cartilaginous endplate obliterate in childhood [22]. Metabolite transport therefore has to occur through small openings in the vertebral endplate (marrow contact channels) via diffusion for smaller molecules and fluid flow coupled for larger molecules [22, 23]. This transport route becomes even more impaired with aging and degeneration where calcifications of the marrow contact channels are observed [24, 25]. This hostile environment with limited metabolite transport and low oxygen tension limits density, viability, and activity of disc cells and explains the reduced capability of the IVD for regeneration and recovery from mechanical injury [22, 26]. In fact, probably as a repairing reaction to destruction and failure of disc matrix, an increase in cell proliferation has been observed in disc degeneration [27]. On the other hand, there is increased cell senescence in degenerated discs [9, 28]. It has been suggested that replicate senescence may naturally occur during aging, while stress-induced premature senescence may be the result of exposure to reactive oxygen species, mechanical load, or inflammatory mediators, contributing to degeneration [29, 30]. Apart from the cell density, viability, and activity, phenotypic changes during aging and degeneration have been extensively studied. As a result of altered phenotype, IVD cells exhibit multiple functional changes, including compromised capability of synthesizing correct matrix components, enhanced catabolic activity, altered synthesis of growth factors and their receptors, and increased levels of inflammatory mediators [29]. Age-related changes in the concentration of matrix macromolecules have recently been documented comprehensively [31]. These changes often result in an insufficiency to maintain a highly hydrated matrix in the NP, which in turn severely affects the mechanical integrity of the IVD. The IVD mechanical function of distributing axial loads and absorbing shock, while providing flexibility, strongly relies on the hydrodynamic capabilities of the NP. Reduced disc pressure in dehydrated, degenerated discs leads to eccentric loading patterns of the endplates, and reduced disc height transfers the load to the posterior elements of the segment, which can initiate annular lesions, herniations, and ultimately facet joint arthrosis [32]. The segment presents an abnormal motion pattern, defined as segmental instability, which is one of the most common causes for LBP [33].

Imaging of Early Lumbar Intervertebral Disc Degeneration

The assessment of advanced lumbar intervertebral disc degeneration is possible with multiple radiologic modalities such as conventional radiography, CT scan, and discography. Thanks to the progress of the magnetic resonance imaging (MRI), this is nowadays the best diagnostic tool to describe and assess not only the advanced lumbar intervertebral disc degeneration (IVDD) but also the first signs of the intervertebral disc degeneration. Conventional clinical MR imaging emphasizes the signal intensity and morphologic changes of intervertebral discs in T2-weighted imaging [34]. Pfirrmann et al. therefore suggested to use a semiquantitative score for the grading of IVDD [35]. Four morphological parameters are evaluated for the Pfirrmann's score: the structure of the disc in T2, the distinction of the nucleus pulposus and annulus, the signal intensity, and the height of the intervertebral disc. Due to its simplicity Pfirrmann's score is frequently used, however, this score shows a lack of correlation between histology and biochemistry or with the clinic [36, 37]. Standard T2-weighted MRI scores are of limited value discriminating early degenerative changes when correlated to biochemical alterations [38]. This is the reason why the use of other sequences to quantitatively evaluate the early degenerative changes on the matrix content of intervertebral discs has recently been suggested. Since the intervertebral disc is the largest avascular structure in the body, its nutrition largely depends on the diffusion of fluid either from the vertebral bodies or

through the annulus fibrosus. Reduction in apparent diffusion coefficient (ADC) has been associated with reduction in nutrient supply in IVDD [39]. A strong correlation between the ADC and a T2 signal/CSF signal ratio has been demonstrated [40]. However, a recent study proved that the diffusion-weighted imaging is less sensitive to detect early morphologic changes in intervertebral disc compared with the T2-weighted imaging [41]. The most significant change that occurs in an early degenerative disc is the loss of proteoglycans [42]. T1rho imaging has the advantage of showing the interaction between water molecules and their macromolecular environment and early biochemical changes in the intervertebral disc [42, 43]. Modern, quantitative MRI techniques as T2 mapping or T1rho imaging have the advantage of showing the interaction between water molecules and their macromolecular environment and early biochemical changes in the intervertebral disc [44-46] (Fig. 8.4). The T1rho sequence was significantly associated with clinical symptoms. It was also more sensitive in order to detect early degenerative changes in the disc in low Pfirrmann score. Furthermore, the signal intensity was weaker in the Pfirrmann grade 2 than in Pfirrmann grade 1. T1rho and T2 were strongly correlated and more sensitive in order to detect the early degenerative changes in the intervertebral disc [36]. The T1rho-weighted sequence has demonstrated a wide range in signal intensity in Pfirrmann's score 1 and 2, and therefore it was suggested to create a quantitative scale in early degenerated discs [47]. It is also important to mention that the T1rho sequence has been proved to reflect the swelling pressure in the disc and therefore its mechanical function [48]. T2 mapping sequences also allow to quantitatively measure water content and are also sensitive for fiber orientation of structures like the annulus fibrosus [45, 46]. Especially on axial images early changes at the nucleus-annulus interface can be observed, as they represent the mucinous infiltration and invagination of the inner annulus fibrosus fibers which is according to Thompsons macroscopic grading one of the first signs for degeneration [49].

Recently the feasibility of using MR spectroscopy has been examined in cadaveric and bovine spine. A correlation between biochemical reduction in glycosaminoglycan content and N-acetyl/Lac+Lip and N-acetyl/Chol ratios was demonstrated [50]. The feasibility and the clinical application of MR spectroscopy for the demonstration of early IVDD have never been demonstrated. In conclusion, MRI is the gold standard to detect early degenerative changes in the lumbar spine. Currently the Pfirrmann's score is the most used classification scheme; in the near future quantitative and more sensitive sequences as T2 mapping or T1rho imaging are likely to be used in clinical practice. The DWI-weighted sequence has been shown to be less sensitive.

Rational of Interspinous Implants

Discogenic low back pain and spinal stenosis due to hypertrophy of the ligamentum flavum, protrusion of the annulus, and hypertrophic arthrosis of the intervertebral articulations are well-known pathologies in our society. At the beginning of the degenerative process, before alteration of disc height, an increase in range of motion with segmental laxity was demonstrated by Ebara et al. [51] and Mimura et al. [52]. The cascades of disc degeneration begin with loss of disc height and overcharging of the facet joint leading to an intermediate stage of abnormal segment motion in the middle staging before structural lumbar changing appears. This late stage characterized by severe disc degeneration, decrease of disc height and reduced intervertebral mobility, is followed later by structural degenerative deformity and stenosis. The interspinous spacers are implants which are introduced between the spinous processes of the lumbar spine to achieve a segmental distraction. The posterior element tension banding restores the loss of stability missed in the early stage of disc degeneration. This indirect tension, if the implant is correctly positioned, has a positive effect with retention of the posterior annulus and realignment of facet joint without changing the physiological spine balance. The interspinous implants can be classified in two groups depending on the function concept: flexion/extension stabilizer and extension stabilizer. The flexion/extension stabilizer devices have an anchorage in the spinous processes. An example is the DIAM invented by J. Taylor [53]. It is a silicone "bumper" wrapped into a polyester sheath connected to artificial ligaments, positioned in the interspinous space and fixed with the ligament around the superior and inferior spinous processes. The Wallis invented by J. Sénégas [54] is a similar implant with the same fixation system, but the "bumper" is realized in plastic. The surgical technique of the described implants is similar. The SLL (supraspinous lumbar ligament) needs to be detached and the interspinous ligament resected. The difference between these implants is the final stiffness created by the axial load of the "bumper." The DIAM is a less rigid implant than the Wallis. This rigidity has to be considered in the choice of the implant and in the patient selection. These implants have a limited long-term function and efficacy with a tendency of a spontaneous segment fusion. After a long experience with different type of implants, we felt the strong request to develop the YODA (created by SpineArt with the collaboration of G. Maestretti and P. Mangion) a new implant with the capacity to maintain a long-term segment motion and to stabilize the disc.

Implant Description and Surgical Principle

The YODA (Fig. 8.1) posterior dynamic device is realized in Phynox and is designed with a central spring and inferior and superior holding elements, fitting securely between the spinous processes. The interspinous central body is used as a "standalone" device. YODA device exists in two sizes (small and large) and is introduced unilaterally preserving the supraspinous ligament. This supple and dynamic implant is intended to unload the disc and maintain mobility and height between two spinous processes allowing flexibility of the implant to accommodate the natural flexion and extension movements of the spine. The elasticity of the implant is the main difference comparing it to other implants in the market that are placed in the interspinous space. The elasticity of the YODA implant is the key feature, which is



designed to rebalance and stabilize the spinal segment. A biomechanical study (Kiami cadaveric test with 3D stereotypic measurement) showed that the YODA is an implant which modifies the kinematics of a lumbar segment where the disc has been injured. The insertion of the YODA significantly reduced the deformation of the interspinous space in the lumbar spines tested, showing a stabilizing effect of the implant regarding the segment. The change in the flexion angulations shows that the YODA has a restabilizing function, putting the segment under tension, relieving the intervertebral disc. The stabilizing effect was quantified in flexion with a 7° reduced flexion and increased 4° extension in comparison with a non-instrumented segment. Furthermore, the tests showed an opening of the foramen by an average of 11 %. This is not a simple spacer; it is designed to put the treated segment back under tension by opening the foramen. The YODA is flexible and dynamic and does not behave as an inert rigid interspinous wedge. It combines a posterior "end-stop" effect, due to its obligatory interspinous filling, with a mechanical return in extension and ligament tensioning in flexion. Another advantage of the YODA implant is its minimal invasive fitting technique which retains part of the interspinous ligaments and above all, the entire supraspinous ligament. These ligaments are prestressed and put under tension after fitting the implant by opening the interspinous space. If the more anterior ligaments (anterior and posterior longitudinal ligaments and the ligamentum flavum) are unharmed, they will also be stretched to a lesser degree, because they are farer from the center of the YODA implant. This distraction of the interspinous space, as shown by the reopening of the foramina, occurs within a vertebral unit where the ligaments are all preserved and leads to strain the supraspinous and interspinous ligaments.

Advantages

Technical Advantages

- Minimally unilateral open posterior approach
- Less invasive surgical technique with only consecutive interspinous distraction preserving the muscle
- No detaching or damage of supraspinous lumbar ligament
- Due to the shape and composition in Phynox, the YODA is more elastic than other products in titanium with decrease spinous process stress fracture and future osteolysis
- Two size choices to better fit the interspinous process space
- Simple and compact set instruments to decrease the overall cost

Clinical Advantages

- Restore the missed stability by distracting the posterior elements and posterior annulus relieving low back pain due to degenerative instability
- Indirect disc stabilization with reduced and controlled flexion
- Indirect increase foramen size with reducing root impingement
- Maintain the movement in flexion and extension
- Lateral clamp stabilization to reduce implant migration
- Adapted for multilevel utilization
- Simple surgical technique with reduction of operative time
- Negligible blood loss
- Full reversible procedure with preservation of intact anatomical structures after implant removal

Disadvantages

- No rotational stabilization
- Simple surgical technique with risk of wrong utilization and enlarged indication
- Possible wrong size choice or incorrect implant positioning with risk of secondary displacement

Indications

- Young-middle age patients with history of back pain (>6 months), presenting a disc degeneration with maintained segment mobility
- Disc degeneration grade III and IV following the MRI Pfirrmann classification [35] with ± Modic I change [55]
- In association with the surgical treatment of a voluminous discal hernia or in recurrent discal hernia
- Central, lateral, and foraminal lumbar spinal stenosis with leg, buttock, or groin pain, which can be relieved during flexion due to a large bulging disc
- Topping off pathology adjacent to fusion

Contraindications

- General contraindication for a surgical treatment
- Infection and tumors
- Fractures
- Conus/cauda syndrome
- · Severe structural spinal stenosis lacking a dynamic component
- Degenerative spondylolisthesis at index level of grade >I according to Meyerding
- Spondylolisis
- Scoliotic deformity at index level
- Not mobile or hypomobile segment
- DDD with fixed retrolisthesis
- Spinous process and Baastrup/or lamina dysplasia
- Grade V Pfirmann disc degeneration [35]
- Grade IV Weishaupt facet degeneration [56]
- Nonspecific discogenic low back pain
- Severe osteoporosis
- Morbid obesity (BMI >40)
- Psychological disorders
- Pregnancy

Surgical Technique

Preoperative Planning

The patient's selection is the key of surgery success. The history of patient pain, the plain flexo-extension films, and MRI investigations must be correlated with clinical examination. To confirm the discogenic pain, a discography with double test is

Fig. 8.2 Microscopic intraoperative view of minimal invasive unilateral posterior approach. The image shows the integrity of the supraspinous ligament after YODA implantation



performed. In addition facet joint and sacroiliac joint infiltrations are utilized to find the pain origin. For the soft stenosis, a functional MRI with myelographic sequences or a conventional myelography with CT scan is preformed to assess the clinical indication.

Anesthesia

The procedure is performed with the patient in general or in spinal anesthesia depending on the indications and on the patient. An antibiotic prophylaxis in single shoot is administered at the induction.

Patient Positioning

The patient is placed in prone position on a radiolucent table. An increased flexion of the spine is useful to optimize the implant placement and does not increase the risk a postoperative segmental lumbar kyphosis because, against other products, the implant is flexible.

Surgical Steps

Minimally Invasive Posterior Approach

Mini open approaches centered at the interspinous and associated approach for disc herniation decompression are utilized (Fig. 8.2).

Fig. 8.3 Tridimensional drawing of inferior spinous process preparation using the dedicated instrument to detach the muscular insertion



Fig. 8.4 Drawing of the insertion of the YODA implant in the interspinous space



- 1. Interspinous process preparation (Fig. 8.3) This consists in different two instruments inserted directly in the interspinous space for the spinous process preparation.
- Implant size selection With a special footprint insertion instruments, the size of implant is decided.
- 3. Implant insertion (Fig. 8.4) After implant placement on the implant holder, it is inserted between the spinous processes and released.



Fig. 8.5 Postoperative flexo-extension X-rays of a case of L3-L4 YODA implantation

Postoperative Care

Free mobilization is allowed since first postoperative day with extreme flexo-extension and heavy (weight-lifting) limitations. Physiotherapy for spine rebalancing is necessary in the first postoperative months in patients with discogenic pain. X-rays are performed at the first mobilization and in the follow-up at 6 weeks and 3, 12, and 24 months to assess the long-term result in association at the clinical score (Fig. 8.5). The sport activity can be early restarted after few weeks depending on the axial loading stress. A book publication from Calvosa and Dubois [57] presents the rehabilitation program in detail after dynamic stabilization with Dynesys. The main of the therapy are similar and finally are adapted for all dynamic stabilization systems including YODA.

Results and Discussion

Over 150 cases of YODA have been performed all over the world, and in these cases the postoperative evaluation demonstrates promising clinical results. So far 28 patients have been treated in our institution since the end of 2009. From February 2010, 20 patients (11 men and 9 females) with mean age of 44 years (19–77) were enrolled in a prospective study. In 17 patients we treated one level, in 2 patients two levels, and in 1 patient three levels were treated. The treated levels were especially L3–L4 and L4–L5. The indication was in 18 patients a back pain history (>6 months) associated to radiological disc degeneration (>Pfirrmann IV) and a deficitary disc herniation or a dynamic stenosis due to a large bulging disc. Two patients presented chronic discogenic pain and radiological evidence of a two levels discopathy. Patients presenting disc herniation underwent to a microscopic disc decompression at the same time. The average of our patients presented, for the different indications, a leg and back VAS higher than 6 with pathological Oswestry rate. At 1-year preliminary data analysis, the leg and back VAS and Oswestry were improved. The preliminary clinical and radiological analysis shows promising good short-term results. In patients with pure discogenic pain, results seemed to be better than expected but necessitate a postoperative rehabilitation program to restore the spinal balance. These patients presented persistent muscular tension pain due to the preexisting history of back pain. Only after 3 months with physiotherapeutic treatment, a clinical benefit was observed.

Conclusions

The YODA is not indicated to treat degenerative stenosis in the aging spine, because of the difference in the design compared to other interspinous implants. In our opinion if the rare cases of dynamic stenosis are excluded, the indication should be reserved for patients with large herniated disc with low back pain. Ouite different is the concept for patients suffering from discogenic chronic low back pain. The less invasiveness, the early mobilization, and the short rehabilitation time offer a concrete temporary alternative for the patient suffering from low back pain. These reversible solutions do not compromise any future treatments. The mini invasive open approach offers the possibility to treat patients with multilevel lumbar disc pathology. The difficulty in this special treatment remains the patient selection. The results after 1 year in this demanding category of patients demonstrate a positive clinical outcome similar to other more invasive surgical techniques (fusion, total disc replacement). The efficacy of the implant to protect the adjacent segment against accelerated disc degeneration needs years of follow-up to establish whether this theoretical advantage is actually achieved. G. Dubois, J. Sénégas, and J. Taylor observed in some patients at the follow-up MRI a certain capacity of the degenerated black disc to rehydrate after dynamic stabilization. N. Specchia presented cases with histological disc amelioration after Dynesys implantation. These observations are very promising for the future and may be a prospective treatment for young patients suffering from chronic discogenic low back pain. The new biological cellular stem cell or fibroblast research can be one of the more promising future disc treatments. For those eventual possibilities, it is important to maintain the natural patient scaffold (disc) with the interspinous spacers. At the moment with a too short follow-up, it is impossible to answer if the YODA is a really good solution to avoid future disc degeneration at the target level or if it has the same capacity like other interspinous implants to give a sufficient posterior disc stabilization and to provide disc rehydration.

References

- 1. Freemont AJ, Watkins A, Le Maitre C, et al. Current understanding of cellular and molecular events in intervertebral disc degeneration: implications for therapy. J Pathol. 2002;196(4):374–9.
- Andersson GB. Epidemiological features of chronic low-back pain. Lancet. 1999;354(9178): 581–5.
- Mannion AF, Muntener M, Taimela S, et al. A randomized clinical trial of three active therapies for chronic low back pain. Spine. 1999;24(23):2435–48.

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- Fritzell P, Hagg O, Wessberg P, et al. 2001 Volvo Award Winner in Clinical Studies: Lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. Spine. 2001;26(23):2521–32.
- 5. Fritzell P, Hagg O, Wessberg P, et al. Chronic low back pain and fusion: a comparison of three surgical techniques: a prospective multicenter randomized study from the Swedish lumbar spine study group. Spine. 2002;27(11):1131–41.
- 6. Martin BI, Mirza SK, Comstock BA, et al. Reoperation rates following lumbar spine surgery and the influence of spinal fusion procedures. Spine. 2007;32(3):382–7.
- 7. Etebar S, Cahill DW. Risk factors for adjacent-segment failure following lumbar fixation with rigid instrumentation for degenerative instability. J Neurosurg. 1999;90(2 Suppl):163–9.
- Kumar MN, Jacquot F, Hall H. Long-term follow-up of functional outcomes and radiographic changes at adjacent levels following lumbar spine fusion for degenerative disc disease. Eur Spine J. 2001;10(4):309–13.
- 9. Grob D, Benini A, Junge A, et al. Clinical experience with the dynesys semirigid fixation system for the lumbar spine: surgical and patient-oriented outcome in 50 cases after an average of 2 years. Spine. 2005;30(3):324–31.
- van den Eerenbeemt KD, Ostelo RW, van Royen BJ, Peul WC, van Tulder MW. Total disc replacement surgery for symptomatic degenerative lumbar disc disease: a systematic review of the literature. Eur Spine J. 2010;19(8):1262–80. Review.
- 11. Masuda K. Biological repair of the degenerated intervertebral disc by the injection of growth factors. Eur Spine J. 2008;17 Suppl 4:441–51.
- 12. Nishida K, Suzuki T, Kakutani K, et al. Gene therapy approach for disc degeneration and associated spinal disorders. Eur Spine J. 2008;17 Suppl 4:459–66.
- 13. Sakai D. Future perspectives of cell-based therapy for intervertebral disc disease. Eur Spine J. 2008;17 Suppl 4:452–8.
- Hohaus C, Ganey TM, Minkus Y, et al. Cell transplantation in lumbar spine disc degeneration disease. Eur Spine J. 2008;17 Suppl 4:492–503.
- Alini M, Roughley PJ, Antoniou J, Stoll T, Aebi M. A biological approach to treating disc degeneration: not for today, but maybe for tomorrow. Eur Spine J. 2002;11 Suppl 2:S215–20. Review.
- Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain [see comments]. N Engl J Med. 1994;331:69–73.
- Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am. 1990;72(3):403–8.
- 18. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? Spine. 2006;31(18):2151–61.
- Le Maitre CL, Freemont AJ, Hoyland JA. Accelerated cellular senescence in degenerate intervertebral discs: a possible role in the pathogenesis of intervertebral disc degeneration. Arthritis Res Ther. 2007;9(3):R45.
- Battié MC, Videman T. Lumbar disc degeneration: epidemiology and genetics. J Bone Joint Surg Am. 2006;88 Suppl 2:3–9. Review. PubMed PMID: 16595435.
- Chan D, Song Y, Sham P, Cheung KM. Genetics of disc degeneration. Eur Spine J. 2006;15 Suppl 3:S317–25. Review.
- 22. Urban JP, Smith S, Fairbank JC. Nutrition of the intervertebral disc. Spine. 2004;29(23): 2700–9.
- Ferguson SJ, Ito K, Nolte LP. Fluid flow and convective transport of solutes within the intervertebral disc. J Biomech. 2004;37:213–21.
- 24. Bernick S, Cailliet R. Vertebral end-plate changes with aging of human vertebrae. Spine. 1982;7(2):97–102.
- Benneker LM, Heini PF, Alini M, Anderson SE, Ito K. 2004 Young Investigator Award Winner: vertebral endplate marrow contact channel occlusions and intervertebral disc degeneration. Spine (Phila Pa 1976). 2005;30(2):167–73.

- Horner HA, Urban JP. 2001 Volvo Award Winner in Basic Science Studies: Effect of nutrient supply on the viability of cells from the nucleus pulposus of the intervertebral disc. Spine. 2001;26(23):2543–9.
- Sakai D, Mochida J, Yamamoto Y, et al. Transplantation of mesenchymal stem cells embedded in Atelocollagen gel to the intervertebral disc: a potential therapeutic model for disc degeneration. Biomaterials. 2003;24(20):3531–41.
- Roberts S, Evans EH, Kletsas D, et al. Senescence in human intervertebral discs. Eur Spine J. 2006;15 Suppl 3:S312–6.
- 29. Zhao CQ, Wang LM, Jiang LS, et al. The cell biology of intervertebral disc aging and degeneration. Ageing Res Rev. 2007;6(3):247–61.
- Kim KW, Chung HN, Ha KY, et al. Senescence mechanisms of nucleus pulposus chondrocytes in human intervertebral discs. Spine J. 2009;9(8):658–66.
- Singh K, Masuda K, Thonar EJ, et al. Age-related changes in the extracellular matrix of nucleus pulposus and annulus fibrosus of human intervertebral disc. Spine (Phila Pa 1976). 2009;34(1):10–6.
- McNally DS, Shackleford IM, Goodship AE, et al. In vivo stress measurement can predict pain on discography. Spine. 1996;21(22):2580–7.
- Kirkaldy-Willis WH, Farfan HF. Instability of the lumbar spine. Clin Orthop Relat Res. 1982;165:110–23.
- 34. Modic MT, Ross JS. Lumbar degenerative disk disease. Radiology. 2007;245:43-61.
- Pfirmann CWA, Metzordf A, Zanetti M, et al. Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine. 2001;26:4873–8.
- 36. Blumenkrantz G, zuo J, Li X, et al. In vivo 3.0 Tesla magnetic resonance T1rho and T2 relaxation mapping in subjects with intervertebral disc degeneration and clinical symptoms. Magn Reson Med. 2010;63:1193–200.
- Buirski G, Silberstein M. They symptomatic lumbar disc in patients with low-back pain. Magnetic resonance imaging appearances in both a symptomatic and control population. Spine. 1993;18:1808–11.
- Benneker LM, Heini PF, Anderson SE, Alini M, Ito K. Correlation of radiographic and MRI parameters to morphological and biochemical assessment of intervertebral disc degeneration. Eur Spine J. 2005;14(1):27–35.
- 39. Nguyen-minh C, Riley 3rd L, Ho KC, et al. Effect of degeneration of the intervertebral disk on the process of diffusion. AJNR Am J Neuroradiol. 1997;18:435–42.
- Niinimäki J, Korkiakoski A, Ojala O, et al. Association between visual degeneration of intervertebral discs and the apparent diffusion coefficient. Magn Reson Imaging. 2009;27:641–7.
- 41. Niu G, Yang J, Wang R, Dang S, Wu EX, Guo Y. MR Imaging assessment of lumbar intervertebral disk degeneration and age-related changes: apparent diffusion coefficient versus T2 quantification. AJNR Am J Neuroradiol. 2011;32:1617–23.
- 42. Raj PP. Intervertebral disc: anatomy-physiology-pathophysiology-treatment. Pain Pract. 2008;8:18–44.
- Antoniou J, Pike GB, Steffen T, Baramki H, Poole AR, Aebi M, Alini M. Quantitative magnetic resonance imaging in the assessment of degenerative disc disease. Magn Reson Med. 1998;40(6): 900–7.
- 44. Johannessen W, Auerbach JD, Wheaton AJ, Kurji A, Borkhatur A, Reddy R, Elliott DM. Assessment of human disc degeneration and proteoglycan content using T1-rho weighted magnetic resonance imaging. Spine. 2006;31:1253–7.
- Watanabe A, Benneker LM, Boesch C, Watanabe T, Obata T, Anderson SE. Classification of intervertebral disk degeneration with axial T2 mapping. AJR Am J Roentgenol. 2007;189(4):936–42.
- 46. Hoppe S, Quirbach S, Mamisch TC, Krause FG, Werlen S, Benneker LM. Axial T2* mapping in intervertebral discs: a new technique for assessment of intervertebral disc degeneration. Eur Radiol. 2012;22(9):2013–9.
- 47. Zobel BB, Vadalà G, Vescovo RD, Battisti S, Martina FM, Stellato L, Leoncini E, Borthakur A, Denaro V. T1rho magnetic resonance imaging quantification of early lumbar intervertebral disc degeneration in healthy young adults. Spine (Phila Pa 1976). 2012;37(14):1224–30. PAP.

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- Nguyen AM, Johannessen W, Yoder JH, et al. Noninvasive quantification of human nucleus pulposus pressure with use of T1rho-weighted magnetic resonance imaging. J Bone Joint Surg Am. 2008;90:796–802.
- 49. Thompson JP, Pearce RH, Schechter MT, Adams ME, Tsang IK, Bishop PB. Preliminary evaluation of a scheme for grading the gross morphology of the human intervertebral disc. Spine (Phila Pa 1976). 1990;15(5):411–5.
- Zoo J, Saadat E, Romeno A, Look K, Li X, Link TM, Kunhanewicz J, Majumdan S. Assessment of intervertebral disc degeneration with magnetic resonance single-voxel spectroscopy. Magn Reson Med. 2009;62:1140–6.
- Ebara S, Harada T, Hosono N, et al. Intraoperative measurement of lumbar spinal instability. Spine. 1992;17:44–50.
- Mimura M, Panjabi M, Oxland TR, et al. Disc degeneration affects the multidirectional flexibility of the lumbar spine. Spine. 1994;19:1371–80.
- 53. Taylor J, Ritland S. Technical and anatomical consideration for the placement of a posterior interspinous stabilizer. In: Mayer HM, editor. Chapter 50: Minimally invasive spine surgery. 2nd ed. Berlin/Heidelberg: Springer; 2006.
- 54. Sénégas J. Mechanical supplementation by non rigid fixation in degenerative intervertebral lumbar segment: the Wallis system. Eur Spine J. 2002;11 suppl 2:164–9.
- Modic M, Pavlicek W, Weinstein M, et al. Magnetic resonance imaging of intervertebral disc disease. Radiology. 1984;152:103–11.
- Weishaupt D, Zanetti M, Boos N, Hodler J. MR imaging in osteoarthritis of the lumbar facet joints. Skeletal Radiol. 1999;28:215–9.
- 57. Calvosa G, Dubois G. Rehabilitation in the dynamic stabilization of the lumbosacral spine, vol. 7. Heidelberg: Springer cap; 2008. p. 21–5.