Review Article

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Low Back Pain - Axioms and Controversies

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Low back pain (LBP) is characterized as a pain localized in the lower spine, often with single or double-sided radiation to the buttocks and thighs. LBP is very common symptom that occurs in humans, and its prevalence increases with age. Epidemiological data say about 80-95% of the population who feel, felt, or will feel pain in the lower spine at least once in their life. In the World Health Organization (WHO) report on chronic pain in primary care, 22% of patients reported chronic pain. Taking to account all chronic pain cases, 48% of them concerned the spine. The correlation of the LBP symptoms with depression, smoking, overweight, triglycerides, employment, hereditary tendency are considered. Some researchers support multifactorial ground for this type of back pain, while others state that damaging of one specific structure cause back pain.

Keywords: Low back pain, Intervertebral disc, Connective tissue

Introduction

Low back pain (LBP) is characterized as a pain localized in the lower spine, often with single or double-sided radiation to the buttocks and thighs. LBP is very common symptom that occurs in humans and its prevalence increases with age (1).

The oldest text about LBP, which had survived to our times, is the Egyptian papyrus from 1500 years BC. Unfortunately a description of symptoms only survived, while the part containing methods of the treatment did not endure until our times. Over the next centuries back pain was regarded as a passing ailment or symptom of rheumatoid arthritis (2).

Modern approach to the LBP started in the nineteenth century. These symptoms most often were defined as an irritation of the spine structures through injury. The cure for back pain was resting in bed. After the discovery of degenerative changes in the intervertebral disc it has been stated, that damage of intervertebral discs is the cause of back pain. Therefore treatment was taken up by orthopedic surgeons.

Since Second World War concern on LBP has increased and since than many different therapies arose. However despite the efforts of modern medicine, it doesn't cope much better with LBP than previous generations. Moreover it seems that more and more people suffer from LBP (3).

This descriptive study aims to gather information from literature based on Medline, Pubmed, and Embase on the incidence, risk factors and pathogenesis of non-specific LBP.

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LBP Prevalence

Epidemiological data say about 80-95% of the population who feel, felt, or will feel pain in the lower spine at least once in their life (4-5). In the U.S. back pain affects annually, according to some authors 15-30% of patients, other data say about the maturity up to 45% -50% per year and is the second most common cause of outpatient visits (6-8). In about 15% of adults and 27% of older people lower back pain is chronic (6).

In the World Health Organization (WHO) report on chronic pain in primary care, 22% of patients reported chronic pain. Taking to account all chronic pain cases, 48% of them concerned the spine. Chronic pain is often combined with psychological problems and disabilities (9). The analysis of the National Health Interview Survey (NHIS) from 1997 revealed that 3.2% of the population has experienced limitations in daily activity due to back pain (10), moreover it is the most common cause of disability in persons over 65 years of age (11).

Significant differences between the incidence of back pain in men and women exists (women get sick more often), and also between ethnic groups (Indians and people from Alaska are subjected to it most often; Asian-Americans are least likely to get LBP symptoms). It was found that the prevalence of back pain is related to the degree of education and income. Persons who have not completed high school had an LBP episode in 32%, while only 22% of people who have completed a university ever felt pain in the spine. Persons with higher incomes rarely feel the pain (7).

Another problem discussed in the literature is an adolescent's back pain. It appears that the occurrence of back pain in youth increases with age and occurs earlier in females, which may be associated with maturation. In addition, there is a strong relationship with the pain in youth and symptoms occurrence in adulthood (12).

The number of medical interventions on the low back pain problem has increased over the past two decades. The number of back surgeries, visits to therapists and chiropractors increased (13). The question is whether this increase is due to higher morbidity, or paying visits to the doctor more willingly. In the literature there is no unified opinion on this subject. Some of the studies claim that increase of back pain is alarming (14) while others do not notice the big differences over last years (15).

Such a large scale of the problem has specific social and economic consequences. In the United States alone LBP leads to 149 million days of absence from work (16), resulting in financial losses at 100 - $ 200 trillion per year (17). In the U.S., over the nine years between 1997 and 2005 there was an increase in expenses associated with back pain by 171% (18).

Risk Factors for Lumbar Spine Pain Syndrome

Studies suggest that the LBP risk factors exist. The correlation of the LBP symptoms with depression is well documented, but the relationship between thosetwo units is not so obvious (19-21). It is still not clear whether depression is the risk factor of LBP or vice versa (22). Some studies show that about 16% of back pain in USA is caused by dysphoric mood. There are even researches that emphasize dominant role of psychological factors in the development of LBP (23).

It appears that LBP is more common among smokers than in non-smokers (24), (25) Moreover cigarette smokers have more severe symptoms than non-smokers (26). Overweight is frequently examined risk factor (27). The results of some studies showed that weight loss reduces pain spine. The proof of this was to be a reduction in intensity of the LBP symptoms in patients after bariatric surgery (28). However not all researchers support this conclusion, thus the correlation of obesity and low back pain is still not confirmed.

The positive correlation between the occurrence of back pain and the triglycerides level and the inverse correlation between the occurrence of back pain with the HDL level was obtained (29).

Factors resulting from employment were also examined. Many researchers have checked the impact of heavy physical work such as lifting, pushing, manipulating heavy objects for the LBP symptoms, but surprisingly many of these studies did not obtain sufficient correlation between LBP and hard physical work (30). Some authors notice hereditary tendency to develop a pain in the lumbar spine (6), (31).

Classifications of LBP

Classification in terms of duration:

1) Acute - lasting up to three months
2) Chronic - lasting longer than three months (32), (33)

Some authors distinguish subacute pain lasting from 6 weeks to 3 months (2).

The simplest and most frequently repeated classification of LBP, divides it into:

1) Mechanical
2) Neuropathic
3) Secondary to another disease

Back pain caused by mechanical substrate is about 80 to 90% of all back pain. It comes from the spine and its supporting structures. Neuropathic pain is caused by irritation of the nerve root by a herniated intervertebral disc or osteofit. Such reasons of LBP as cancer, infections, rheumatoid arthritis, ankylosing spondylitis, reactive inflammation, Paget disease, Scheuerman disease, Bastrup disease comprise about 1-2% of cases, while the pain caused by disease outside the spine occurs in 1-2%. The cause of lumbar pain in 2-4% of cases is fibromyalgia or somatization (1, 34).

The existence of pain in the lumbar spine in the course of other diseases and the oppression of the nerve root are unquestionable. However in most cases of back pain it is not possible to determine precise cause. Some authors call this group, nonspecific pain (35).

Pathogenesis of Nonspecific LBP

Pathogenesis of nonspecific LBP is still submitted to discussion. Some researchers support multifactorial ground for this type of back pain, while others state that damaging of one specific structure cause back pain (36). The researchers, who advocate multifactorial cause of back pain, believe that degenerative changes present in the spine are natural processes occurring in the body (8, 34). For supporting this thesis there are studies, where no correlation was found between the advancement of degenerative changes and the presence or intensity of pain (1, 34, 37, 38).

Scientists, who believe that the disc degeneration is main cause of LBP, explain this discrepancy saying that in the structure of the disk in patients with pain the process of inflammation develops. The evidence for the statement above was the disclosure of the presence the area of vascularized granulation tissue in disk samples taken from patients with pain. This area is stretched from the nucleus pulposus to the outer regions of the annulus fibrosus (39). According to study mentioned above such changes do not occur in people with a healthy intervertebral disc as same as in those who, despite having intervertebral disc damaged, feel no back pain (39, 40). The other difference between painful and not painful discs is the number of nerve fibers (39). In healthy discs nerve fibers are observed only in the most external layers of the annulus fibrosus. In damaged, but unpainful discs, nerves penetrate deeper, but do not occur in the most inner layer, neither in the nucleus pulposus. Deeper penetration of nerves to the intervertebral disc, usually in the area of inflammation foci, can be observed only in the painful disc. The authors of discussed research suppose that the formation of vascularized granulation tissue is a physiological healing mechanism of annulus fibrosus damage. However, due to poor vascularization of these structures, healing process is difficult and for some patients ends with formation of exuberant granulation tissue and increase the number of nerve fibers in the intervertebral disc structures. According to the research above that process correlates with pain (39).

Degenerative facet joints were treated as obvious cause of back pain since 1911. It was confirmed by reports of decreased pain after intraarticular blockades (41, 42). Moreover experimental studies shown that stimulation of these joints causes pain in lumbar area and in the front surface of the thigh. According to the authors this effect clearly indicates that a certain percentage of LBP cases is caused by irritation of the facet joints (43). In the other hand, the study comparing the frequency of degenerative changes occurrence in facet joints, did not display correlation with pain in lumbar area (41).

In the literature, one can also find studies, where sacroiliac joints are stated as a source of back pain. Data indicate that these joints can cause back pain even in 15-25% (44-46). Research that were conducted with use of diagnostic blockades have shown existence of a lumbar spine pain from irritation of the nerve endings in the sacroiliac joints (46-48). Other structures that are considered to be a source of pain are fascia and connective tissue structures (49, 50). Structures mentioned above could be the cause of pain as a result of chronic local inflammation associated with restricted mobility. That process could lead to fibrosis of the connective tissue and its stiffness and pain (51).

Some authors claim, that in the pathogenesis of back pain, muscle component is also included (52). This statement could be confirmed by the fact that density differences in paraspinal muscles were found in patients with LBP in computed tomography (53, 54). In studies conducted with use of magnetic resonance spectroscopy (NMRS) it was found that muscle tone during the rest in patients with LBP was higher than in healthy controls. According to the authors these results suggest that back pain is associated with incomplete muscle relaxation (54).

Interpretations of changes occurring in the muscles in the course of LBP are different. Some authors claim that the pain caused by structures damage such as the intervertebral disc or ligament, cause muscle tension increase and consequently the repeated pain. However, there are also authors, who believe that the change in muscle tone is a physiological protection against the pathology (55).

Psychological component, according to some researchers, plays an important role in the development and course of LBP (56-58). The results of experiments showed that fear against pain in healthy persons causes the formation of aberrant patterns of paraspinal muscles activation, the same that occur in patients with lumbar spine pain (59).

An interesting experiment was conducted by Giesecke (60). The results of experimental pain test and test using functional MRI showed greater sensitivity to pain in people with LBP as a result of oppression, than in the control group. In patients with lumbar spine pain exerting pressure on the thumb resulted in a stronger feeling the pain of the oppressed space than it occurred in the control group. From the study above it can be stated that chronic pain of the spine may activate the pathological ways of processing the pain in the brain.

Langevin and Sherman established hypothesis about the role of the central nervous system in the pathogenesis of LBP (51). Their inference relied on studies that have shown that the constant pain is associated with significant changes at many levels of the central nervous system resulting from its neuroplasticity. This happens also in the motor cortex, what allows hypothesized that all factors suspected of causing chronic pain of the spine are involved. It was concluded that the connective tissue remodeling that takes place in the LBP is the result of emotional, behavioral features and motor dysfunction. The first step is a sharp pain. If the pain will lead to the fear of pain development, it will start a cascade leading to chronic symptoms. The person limits ones activity, what causes changes in the muscle innervation and eventually leads to the stronger muscle contraction, microtraumas and muscle inflammation. Each episode of sharp pain may cause a local inflammatory response. When inflammation occurs, sensitization of nociceptors of peripheral and central nervous system arise what reinforces ongoing inflammation in tissues by releasing inflammatory neurotransmitters, such as substance P. The result of these amendments is to tissue fibrosis. In turn, the peripheral and central nervous system sensitivity is increased, what facilitates the development of tissue inflammation, depressed mood, pain and fear of the limitations resulting from the motion.

Each subsequent episode of pain leads to an even greater reduction in mobility and accumulation of fibrosis causing more and more painful ailments. Those patients whose fear of pain does not show up are less likely to develop chronic pain.

Panjabi also created a hypothesis focusing on the creation of a local pain (61). According to the author trauma or multiple microtraumas to the ligaments of the spine causes damage to the fiber ring mechanoreceptors, which are beginning to generate distorted signals. The result is a disorder of muscle response patterns resulting in impaired muscle coordination. Pathological tension arises in the ligaments and muscles and the articular surfaces are overloaded. The next step is accelerated disc degeneration and a faster rise of osteoarthritis. If the above-described conditions are maintained, this leads to inflammation of the nerve tissue and results in chronic back pain.

After the Panjabi' s theory appeared, other researchers claimed that his approach is incomplete and is missing of role the central nervous system. In addition, it was suggested extending the above theory and the inclusion the thoracolumbar fascia to it (2, 49).

Conclusion

A multitude of hypotheses proved that the nature of back pain is complicated, which is associated with problems in making a diagnosis and implementation of effective treatment. Due to importance of the problem further research are needed to enable proper diagnosis for the individual patient and facilitate the treatment choice.

Reference

1. Domżał T. Neurologiczne postępowanie w bólach krzyża standardy i zalecenia. Pol. Prz. Neurol. 2010; 6(2):59–69.
2. Krismer M, van Tulder M; Low Back Pain Group of the Bone and Joint Health Strategies for Europe Project. Strategies for prevention and management of musculoskeletal conditions. Low back pain (non-specific). Best Pract Res Clin Rheumatol. 2007; 21(1): 77-91.
3. Waddell G. Low back pain: a twentieth century health care enigma. Spine. 1996, 15; 21(24): 2820-2825.
4. Rubin D. Epidemiology and risk factors for spine pain. Neurol Clin. 2007; 25(2): 353-371.
5. Andersson G. Epidemiological features of chronic lowback pain. Lancet. 1999; 354 (9178): 581–85.

6. Manchikanti L, Singh V, Datta S, Cohen SP, Hirsch JA; American Society of Interventional Pain Physicians. Comprehensive review of epidemiology, scope, and impact of spinal pain. Pain Physician. 2009; 12(4): E35-70.
7. Deyo RA, Mirza SK, Martin BI. Back pain prevalence and visit rates: estimates from U.S. nationalsurveys, 2002. Spine. 2006; 31: 2724–2727.
8. Deyo RA, Weinstein JN. Low back pain. N Engl J Med. 2001; 344: 363-370.
9. Gureje O, von Korff M, Simon GE, Gater R. Persistent pain and well-being: A World Health Organization study in primary care. JAMA, 1998; 280(2): 147-151.
10. US Department of Health and Human Services. Healthy people 2010. 2nd ed. With: Understanding and improving health and objectives for improving health. 2 vols. Washington, DC: US Government Printing Office; 2000
11. Chou R, Huffman LH. Guideline for the Evaluation and Management of Low Back Pain Evidence Review. American Pain Society, Publisher Glenview, 2009
12. Jeffries LJ, Milanese SF, Grimmer-Somers KA. Epidemiology of adolescent spinal pain: a systematic overview of the research literature. Spine. 2007; 32(23): 2630 –2637
13. Freburger JK, Holmes GM, Agans RP, Jackman AM, Jane D, Darter JD, Wallace AS, Castel LD, Kalsbeek WD, Carey TS. The Rising Prevalence of Chronic Low Back Pain. Arch Intern Med. 2009; 169(3): 251-258.
14. Palmer KT, Walsh K, Bendall H, Cooper C, Coggon D. Back pain in Britain: comparison of two prevalence surveys at an interval of 10 years. BMJ. 2000; 320(7249): 1577-1578.
15. Hüppe A, Muller K, Raspe H. Is the occurrence of back pain in Germany decreasing? two regional postal surveys a decade apart. Eur J Public Health. 2007; 17(3): 318-322.
16. Guo HR, Tanaka S, Halperin WE, Cameron LL. Back pain prevalence in US industry and estimates of lost workdays. Am J Public Health. 1999; 89(7): 1029-35.
17. Katz JN. Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am. 2006; 88(suppl 2): 21-24.
18. Schwenk TL. Healthcare Expenditures for Back and Neck Pain. Journal Watch. 2008; 7(2), Massachusetts Medical Society 2008.
19. Currie SR, Wang J. Chronic back pain and major depression in the general Canadian population. Pain. 2004; 107(1-2): 54-60.
20. Ramond A, Bouton C, Richard I, Roquelaure Y, Baufreton C, Legrand E, Huez JF. Psychosocial risk factors for chronic low back pain in primary care--a systematic review. Fam Pract. 2011; 28(1): 12-21.
21. Carroll LJ, Cassidy JD, Côté P. Depression as a risk factor for onset of an episode of troublesome neck and low back pain. Pain. 2004 Jan; 107(1-2): 134-139
22. Larson SL, Clark MR, Eaton WW. Depressive disorder as a long-term antecedent risk factor for incident back pain: a 13-year follow-up study from the Baltimore Epidemiological Catchment Area sample. Psychol Med. 2004; 34(2): 211–219.
23. Linton SJ. A review of psychological risk factors in back and neck pain. Spine 2000; 25(9): 1148–1156.
24. Alkherayf F, Wai EK, Tsai EC, Agbi C. Daily smoking and lower back pain in adult Canadians: the Canadian Community Health Survey J Pain Res. 2010; 3: 155–160.
25 Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between smoking and low back pain: a meta-analysis. Am J Med. 2010; 123(1): 87.e7-35
26. Vogt MT, Hanscom B, Lauerman WC, Kang JD. Influence of smoking on the health status of spinal patients: The National Spine Network Database. Spine 2002; 27(3): 313-319.
27. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The Association Between Obesity and Low Back Pain: A Meta-Analysis. Am J Epidemiol. 2010; 171(2): 135-154.
28. Melissas J, Kontakis G, Volakakis E, Tsepetis T, Alegakis A, Hadjipavlou A. The effect of surgical weight reduction on functional status in morbidly obese patients with low back pain. Obes Surg 2005; 15(3): 378–381.
29.Heuch I; Heuch I; Hagen K; Zwart J A. Associations Between Serum Lipid Levels and Chronic Low Back Pain. Epidemiology. 2010; 21(6): 837-841.
30. Bakker EW, Verhagen AP, van Trijffel E, Lucas C, Koes BW. Spinal Mechanical Load as a Risk Factor for Low Back Pain, A Systematic Review of Prospective Cohort Studies. Spine 2009; 34(8): E281-293.
31. Livshits G, Popham M, Malkin I, Sambrook PN, MacGregor AJ, Spector T, Williams FM. Lumbar disc degeneration and genetic factors are the main risk factors for low back pain in women: the UK Twin Spine Study. Ann Rheum Dis. 2011; 70(10): 1740-1745.
32.Domżał T. Przewlekłe nieswoiste bóle krzyża— stara dolegliwość czy nowa choroba neurologiczna? Pol. Prz. Neurol. 2007; 3(4): 216–227.
33. Ehrlich GE. Low back pain. Bulletin of the World Health Organization 2003, 81(9).
34. Cohen SP, Argoff ChE, Carragee EJ. Management of low back pain. BMJ, 2008; 337:a2718.
35. Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffett J, Kovacs F, Mannion AF, Reis S, Staal JB, Ursin H, Zanoli G; COST B13 Working Group on Guidelines for Chronic Low Back Pain. Chapter 4.European guidelines for management of chronic nonspecific low back pain. Eur Spine J. 2006;15 (Suppl 2): S192–S300.
36. Hancock MJ, Maher CG, Latimer J, Spindler MF, McAuley JH, Laslett M, Bogduk N. Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. Eur Spine J. 2007; 16(10): 1539–1550.
37. 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. N Engl J Med. 1994; 331(2): 69-73.
38. Powell MC, Wilson M, Szypryt P, Symonds EM, Worthington BS. Prevalence of lumbar disc degeneration observed by magnetic resonance in symptomless women. Lancet 1986; 2(8520): 1366–1367.
39. Peng B, Wu W, Hou S, Li P, Zhang C, Yang Y. The pathogenesis of discogenic low back pain. J Bone Joint Surg. 2005; 87(1): 62–67.

40. Peng B, Hao J; Hou S,Wu W, Jiang D. Possible Pathogenesis of Painful Intervertebral Disc Degeneration. Spine 2006; 31(5): 560-566.
41. Kalichman L, Li L, Kim DH, Guermazi A, Berkin V, O'Donnell CJ, Hoffmann U, Cole R, Hunter DJ. Facet joint osteoarthritis and low back pain in the community-based population. Spine 2008; 33(23): 2560–2565.
42. Manchikanti L, Boswell MV, Singh V, Pampati V, Damron KS, Beyer CD. Prevalence of facet joint pain in chronic spinal pain of cervical, thoracic, and lumbar regions. BMC Musculoskelet Disord. 2004; 5:15
43. O'Neill S, Graven-Nielsen T, Manniche C, Arendt-Nielsen L. Ultrasound guided, painful electrical stimulation of lumbar facet joint structures: An experimental model of acute low back pain. Pain. 2009; 144(1-2): 76-83
44. Cohen SP. Sacroiliac joint pain: a comprehensive review of anatomy, diagnosis, and treatment. Anesth Analg. 2005; 101(5): 1440-1453.
45. Hansen HC, McKenzie-Brown AM, Cohen SP, Swicegood JR, Colson JD, Manchikanti L. Sacroiliac joint interventions: A systematic review. Pain Physician 2007; 10(1): 165-184.
46. McKenzie-Brown A, Shah RV, Sehgal N, Everett CR. A systematic review of sacroiliac joint interventions. Pain Physician 2005; 8(1): 115-125.
47. Rupert MP, Lee M, Manchikanti L, Datta S, Cohen SP. Evaluation of sacroiliac joint interventions: a systematic appraisal of the literature. Pain Physician. 2009; 12(2): 399-418.
48. Cohen SP, Hurley RW, Buckenmaier CC, Kurihara C, Morlando B, Dragovich A. Randomized placebo-controlled study evaluating lateral branch radiofrequency denervation for sacroiliac joint pain. Anesthesiology. 2008; 109(2): 279-288.
49. Schleip R, Vleeming A, Lehmann-Horn F, Klingler W. Letter to the Editor concerning "A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction" (M. Panjabi). Eur Spine J. 2007;16(10): 1733-1735.
50. Hammer WI, Pfefer MT. Treatment of a case of subacute lumbar compartment syndrome using the Graston technique. J Manipulative Physiol Ther. 2005; 28(3): 199-204.
51. Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. Med Hypotheses 2007; 68(1): 74-80.
52. Bogduk N. On definitions and physiology of low back pain, reffered pain and radicular pain. Pain, 2009;147 (1-3):17-19.
53. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE, De Cuyper HJ. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur Spine J. 2000; 9(4): 266-272.
54. Demoulin Ch, Crielaard JM, Vanderthommen M. Spinal muscle evaluation in healthy individuals and low-back-pain patients: a literature review. Joint Bone Spine 2007; 74(1): 9-13.
55. van Dieen JH, Selen LP, Cholewicki J: Trunk muscle activation in low-back pain patients, an analysis of the literature. J Electromyogr Kinesiol. 2003; 13(4): 333-351.
56. Hurwitz EL, Morgenstern H, Yu F. Cross-sectional and longitudinal associations of low-back pain and related disability with psychological distress among patients enrolled in the UCLA Low-Back Pain Study. J Clin Epidemiol 2003; 56(5): 463-471.
57. Dionne CE. Psychological distress confirmed as predictor of long-term back-related functional limitations in primary care settings. J Clin Epidemiol. 2005; 58(7): 714-718.
58. Pincus T, Burton AK, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/ disability in prospective cohorts of low back pain. Spine 2002; 27(5): E109-120.
59. Moseley GL, Nicholas MK, Hodges PW. Does anticipation of back pain predispose to back trouble? Brain 2004, 127(Pt 10): 2339-2347.
60. Giesecke T, Gracely RH, Grant MA, Nachemson A, Petzke F, Williams A, Clauw DJ. Evidence of Augmented Central Pain Processing in Idiopathic Chronic Low Back Pain. Arthritis Rheum. 2004; 50(2): 613-623.
61. Panjabi MM. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur Spine J. 2006; 15(5): 668–676.

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