Evidence-based practice guidelines for the diagnosis and treatment of lumbar spinal conditions

Abstract: Low back pain remains one of the most common patient complaints. It can exist alone or with the presence of lower extremity symptoms. Review of evidence-based guidelines will assist primary care providers in the identification and treatment of various lumbar disorders in addition to ruling out specific lumbar spinal pathologies.

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Low back pain (LBP) ranks fifth as the reason patients present for healthcare provider visits in the United States and second as the most common chief complaint.[1] It is prevalent among all age groups, ranging from adolescents to older adults.[2] The annual healthcare costs and economic losses associated with LBP in the United States exceed $90 to $100 billion.[3,4] LBP remains the most common reason for disability among patients under age 45.

The prevalence for continued pain or disability from LBP is 60% to 80% after 1 year.[3,4] Patients with a prior history of work absenteeism showed a 40% prevalence for future occurrences.[3,4] Therefore, it is imperative for primary care providers (PCPs) to have a clear knowledge regarding the diagnosis and treatment of a variety of lumbar diagnoses, as patients' LBP treatments typically begin under their care.

Presentation

Nonspecific LBP (NSLBP) is typically described as a mechanical type of pain that varies with patients' physical activity and posture.[2] NSLBP is unrelated to a recognizable pathology, osteoporosis, structural deformity, or radicular syndrome.[2] It may be related to degenerative changes in the intervertebral disk, facet joints, vertebral endplate sclerosis, or presence of osteophytes and is typically seen among working-age patients.[2]

Patients with NSLBP experience back pain that is increased by changes in position, upon flexion, and/or with numbness and weakness.[4] Pain noted with prolonged sitting is a key factor in differentiating it from lumbar stenosis.[5] The association between degenerative disk disease and LBP, based on cross-sectional studies, is significant and typically related to aging and environmental factors.[2,4] One study of retrospective chart reviews indicated that the presence of midline LBP, located directly over the spinous processes, was associated with an 84% accuracy for degenerative disk disease as the source of LBP.[6]

LBP with lower extremity pain exists in approximately 25% to 57% of all lumbar cases.[7] Lumbar stenosis is typically

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acquired through degenerative changes or changes from pathology or prior surgery and is described as narrowing present in the spinal neuroforaminal spaces, lateral recesses, or central canal, but typically refers to narrowing of the central canal.[5,8] Although narrowing of the neuroforaminal canals or central canal is present, there is a poor correlation between the degree of narrowing and symptoms, as some patients remain asymptomatic.[9]

An important factor to consider when discussing patient symptoms is that the canal space increases in flexion and decreases in extension and loading; therefore, patients with lumbar stenosis tend to do better with forward flexion, such as when pushing a grocery cart.[8,10] Degenerative lumbar stenosis is uncommon for individuals under age 50, and the diagnosis and severity are largely dependent on the history and physical exam.[10]

Symptoms of lumbar stenosis include wide base gait, presence or absence of LBP, and neurogenic claudication.[5,9-12] Neurogenic claudication is described as radiating pain into the bilateral or unilateral buttock, anterior thigh, or posterior pain down the leg to the calf and sometimes to the feet that is worsened with standing, walking, or extension and improved with sitting and bending forward.[5,9-12] Neurogenic claudication can include a sensation of weakness and/or heaviness, paresthesias, fatigue, hamstring tightness, and occasional nocturnal cramps.[9] Neurogenic claudication is the most common finding for lumbar stenosis and can severely impact patients' functionality, affecting their quality of life.[12]

Lumbar radiculopathy is defined as pain radiating from the lower back into the legs, which is the result of disk material beyond the disk space margins causing nerve root impingement.[13] Lumbar disk herniations are the primary cause for lumbar radiculopathy. Symptoms of lumbar radiculopathy include radiating pain from the lumbar region, which is primarily unilateral and greater than symptoms of LBP. Some patients with radiculopathy only experience leg pain without any back pain.

Patients can also experience pain that is worse during rest or in the night, numbness or paresthesias that follows a dermatomal distribution (mostly along L4-S1), muscle weakness typically present below the knee, and possible patellar or Achilles reflex changes.[13,14] Poorer prognoses are noted in patients who have LBP greater than or equal to their radicular symptoms.[15]

Muscle strength testing[18]

Muscle strength is graded on a scale from 0 to 5 following the criteria listed below. Normal strength should be graded as a 5/5, whereas no muscle contraction would be a 0/5.

5	Normal strength (full resistance)
4	Movement possible against some resistance by examiner
3	Movement possible against gravity but not against examiner's resistance
2	Movement possible but not against gravity (test in horizontal plane)
1	Muscle flicker but no movement
0	No muscle contraction

Evaluation of LBP: History and physical exam

Providers should classify NSLBP from back pain with radicular symptoms based on symptoms, including whether the pain is above or below the knee.[2,15-17] An accurate history and physical exam are the most appropriate tools to determine this. The history should include the patient's personal description of symptoms, including specific locations (midline, lateral, bilateral) and the degree of pain on the pain visual analog scale.[2,15-17] It is important for providers to ask patients regarding the presence or absence of leg pain, whether unilateral or bilateral, because approximately a quarter to one-half of lumbar cases present with lower extremity pain.[7]

Determining leg pain can also aid in diagnosing between lumbar stenosis and radiculopathy. Other history items to consider include difficulties with activities, including walking, sitting, standing, flexion, and extension; sensorimotor deficits; aggravating or alleviating factors; and prior beneficial or failed treatments, especially in response to specific medication regimens.[2,15-17]

The physical exam should include inspection and palpation of the spine for alignment, tenderness, and/or erythema or edema.[18,19] Motor assessment should include the patient's gait patterns, including regular, tandem, heel walking, and tiptoe.[18,19] Inability to lift the foot when stepping or dragging it is indicative of probable involvement at L4/L5, as is LBP reproduced on heel walking.[18,19] LBP with tiptoeing typically indicates L5/S1 involvement.[18,19] Muscle strength testing should be performed to evaluate for weaknesses, graded on a scale of 0 to 5 (see *Muscle strength testing*).[18,19]

A tactile sensory exam should be used to detect dermatomal deficits. Neurologic testing includes deep tendon reflexes, Babinski reflex, clonus signs, and straight-leg raise testing, which is indicative of nerve root impingement.[13] A positive straight-leg raise reproduces pain in the lower

extremity, not in the lower back.[20] An important finding in the current evidence is that a positive straight-leg raise should only be used along with other positive findings to determine a diagnosis because the positive straight-leg raise test alone lacks diagnostic utility.[20]

The description of a dermatomal distribution pattern on history is a key factor in determining lumbar radiculopathy, which when combined with one or more positive neurologic findings on physical exam, can confirm the diagnosis (see *Positive neurologic findings on physical exam*).[14] Rectal assessment is needed for a patient complaint of saddle paresthesia or bowel incontinence.[18,19] Cardiovascular assessment should include auscultation for abdominal aorta bruits or evaluation of any lower extremity vascular anomalies, such as decreased pulses or pitting edema.[18,19]

Positive neurologic findings on physical exam[14]

- Numbness, paresthesias, and/or abnormal reflexes that are consistent with the described dermatomal distribution from the patient's history
- Positive straight-leg raise testing (typically unilateral)
- Babinski's sign or clonus sign

Diagnostic exam

Routine use of radiologic imaging, which includes basic X-ray images, is not warranted based on clinical guidelines.[2] This is primarily due to the patient's exposure to radiation, poorer patient outcomes, and pathology identification, which can lead to a perception by some patients that their back pain is a more serious condition (commonly called the "labeling effect").[2] Diagnostic imaging should be reserved for patients with progressive neurologic involvement or if the PCP is suspicious of an underlying pathology.[2]

Patients presenting with concerns for pathology or fracture, such as a history of trauma, should have two-view X-ray imaging (anterior-posterior and lateral) performed immediately, with the addition of flexion/extension views for any concerns of spinal instability.[4] If neurologic involvement or other serious underlying pathology is plausible, the study of choice is magnetic resonance imaging (MRI).[13] This is followed by computed tomography (CT) scan or CT scan with myelogram for patients unable to undergo MRIs; CT scan with myelogram is preferred over CT scan if there is concern for neurologic impingement.[13] Contrast with MRI or CT scan is only needed if patients have had prior lumbar surgery.[21]

Differential diagnoses for LBP

The primary goal in the evaluation of LBP from evidence-based guidelines is to first rule out serious pathology or underlying conditions, which are present in about 5% of cases. These conditions or pathologies consist of spinal cancer, spinal fracture, spinal infection, or cauda equina syndrome.[17,21] Spinal cancer is suggested by age over 50, prior history of cancer, insidious onset, unexplained weight loss, night pain, pain at multiple sites (which can occur at rest), urinary retention, and unresponsiveness to prior care.[2,17]

Spine fracture is suggested by age over 50, osteoporosis, trauma, and chronic corticosteroid use.[2,17] Spine infection is suggested by fever, history of I.V. drug use, prior or current infections (possibly from prior lumbar epidural injections), and immunocompromise.[19] Cauda equina syndrome is suggested by acute or worsening radicular symptoms, sensorimotor deficits (including impaired foot dorsiflexion [foot drop]), saddle paresthesias, and bladder and/or bowel incontinence.[16,17]

Providers should classify patients' LBP as radicular or nonradicular to assist in determining an appropriate diagnosis.[2,15-17] The major causes of nonradicular LBP include lumbar strain/sprain, myofascial pain, and lumbar disk degeneration. Major causes of LBP with radiculopathy include lumbar disk protrusions and lumbar stenosis. Radicular pains from lumbar stenosis are typically bilateral, helping to differentiate it from radiculopathy stemming from disk protrusions, which are typically unilateral.[10]

Providers should also be able to differentiate neurogenic claudication from vascular claudication to aid in the diagnosis of lumbar stenosis. Patients whose symptoms are relieved upon standing alone, located typically below the knee, unchanged by flexion of the spine, and have a fixed duration of walking typically have vascular claudication.[10,22] Other possible causes of LBP include ankylosing spondylitis, aortic aneurysm, pancreatitis, or renal calculi.[4,17]

Treatment options

Once the LBP has been identified as radicular or nonradicular, the provider can develop a treatment plan. Most cases of NSLBP, and even lumbar radiculopathy, resolve after a few weeks of treatment.[8] Current treatment options for LBP have a minimal impact on outcomes, are typically short term, and rarely change the longer-term prognostic path for patients.[3]

The goal of treatment for LBP includes pain relief, improvement of function, reduced work leave of absence, and prevention of chronicity.[8] Common treatment options include the use of oral or topical medications, exercise, spinal

manipulation therapy, traction, transcutaneous electrical nerve stimulation (TENS), heat, back supports, acupuncture, biofeedback, spinal injections, and lastly, surgery.[1-3,8,17]

Pharmacologic management

Medication management of NSLBP should be decided after weighing the factors of allergies or adverse reactions of the medication; the duration and severity of the symptoms; the expected benefits; the prior response to various medications; comorbidities; and the medication's cost-efficiency.[1] Medication management remains one of the most recommended treatments for NSLBP, either acute or chronic.[23]

The first choice of treatment consists of short-term use of acetaminophen due in part to its decreased incidence of gastrointestinal (GI) adverse reactions and myocardial infarction (MI). Due to the risk of hepatotoxicity from acetaminophen, patients should be screened for any hepatic contraindications.[1-3,17]

This should be followed by the short-term use of traditional nonsteroidal anti-inflammatory drugs (NSAIDs) with the understanding that these medications carry an increased risk for gastric bleeding and ulceration, MI, and renal adverse reactions (see *Common medications for LBP management*).[1-3,8,17,24,25] Naproxen has the lowest risk of cardiac events.[1] NSAIDs should be avoided in patients over age 75 due to their risks.[1] If used, NSAIDs can be administered with misoprostol or a proton pump inhibitor to reduce GI risks.[1]

Weak opioids may be administered in select cases where patients are unresponsive to the first two recommendations or have a high risk for use of NSAIDs with moderate-to-severe pain. Their use remains controversial with limited evidence of benefits and serious adverse reactions, such as respiratory depression, abuse, and addiction potential.[1,2,8,16,17] The use of tramadol is contraindicated with concurrent use of a selective serotonin or norepinephrine reuptake inhibitor due to the risk of serotonin syndrome.[1]

Some other classes of medications employed in the treatment of NSLBP include skeletal muscle relaxants and antiepileptic medications.[1,25] Although utilized by many providers, evidence-based studies show insufficient evidence for the recommendation of these medications for NSLBP.[1] They may be more useful for patients with LBP and radicular symptoms. Skeletal muscle relaxants, as an adjunct to analgesic medications, have been shown in trials to be more effective than analgesia alone.[1]

Little evidence exists in the literature for the use of oral or systemic corticosteroids in the treatment of NSLBP due to the risk of infection, avascular necrosis, and various other metabolic, endocrine, cardiovascular, or ophthalmologic changes.[1,2,26] Corticosteroids are best reserved for severe acute LBP and should be used cautiously in chronic back pain conditions.

Common medications for LBP management[1,25]

Acetaminophen

Traditional NSAIDs
- Etodolac
- Diclofenac
- Naproxen

COX-2 inhibitor NSAID
- Celecoxib

Weak opioids
- Tramadol

Muscle relaxants
- Baclofen (FDA off-label use for LBP)
- Tizanidine
- Methocarbamol
- Cyclobenzaprine

Antiepileptics
- Gabapentin (FDA off-label use for LBP)
- Topiramate (FDA off-label use for LBP)

Nonpharmacologic management

Nonpharmacologic recommendations for persistent NSLBP include spinal manipulative therapy (SMT), supervised exercise, acupuncture, and cognitive behavioral therapy (CBT).[2,3,8,27] SMT, although supported as beneficial by some studies for the short-term treatment of acute pain, has no clinical benefit after a 6-month duration, with some stating withdrawal of failed treatment after 8 weeks.[16,27]

Other reviews of SMT showed no further benefit from this treatment over any other treatments.[5] Exercise programs are not recommended for patients with acute LBP; however, they were found to be effective in both the prevention and treatment of chronic LBP, with no evidence found that one type of exercise is more beneficial than another.[27]

Acupuncture showed evidence to be beneficial for short-term relief of back pain in conjunction with other treatments; however, when used alone, it was not beneficial except in patients suffering from depression.[28] Many patients question the use of lumbar corsets for reduction in pain. Although they can assist in reducing pain during episodes, prolonged use may cause a reduction in spinal function and deconditioning of the paraspinal and abdominal muscles.[8]

Clinical evidence states that other procedures, such as the use of TENS, intradiscal electrothermal therapy, radiofrequency

facet joint denervation, or percutaneous intradiscal radiofrequency thermocoagulation, lack recommendation.[2,3,16] There is limited data supportive of surgery for NSLBP, and it has been widely overutilized and criticized.[2]

Patient education and prevention

Evidence-based practice for NSLBP, lumbar stenosis, and lumbar radiculopathy indicates that all treatment should begin with basic patient advice regarding the diagnosis. This promotes self-management, provides reassurance that the condition is not a serious disease, and provides encouragement to remain active through activities, such as walking, cycling, or swimming. Steady activity progression is recommended. Discouraging bed rest or employing no more than 2 days duration of bed rest is recommended due to the possibilities of the loss of bone mineral density, muscle wasting, and joint stiffness.[2,3,8,16,17]

The inclusion of the patient in shared decision-making is important in the treatment of LBP, which allows informed choices in their care. Benefits of shared decision-making include patient autonomy, a therapeutic relationship, improved patient satisfaction, and improved patient participation.[29] Although underutilized in most care settings, shared decision-making is a useful tool in the treatment of back pain, especially when it comes to decisions regarding surgery.[29]

In order to prevent the progression of acute NSLBP to chronic back pain, all providers, most important those in primary care settings, should recognize and evaluate patients for psychosocial risk factors (see *Psychosocial risk factors*).[2,16] Providers should employ the use of CBT, which is a cost-efficient option for both the patient and provider with documented sustained results on pain.[2,3,16]

Psychosocial risk factors[2]

- Inappropriate attitudes and beliefs about back pain
- Fear-avoidance behavior
- Anxiety
- Depression
- Workers' compensation claim status
- Litigation status
- Socioeconomic factors
- Malingering pain
- Persistent request for opioid medications when inappropriate for treatment

Specific treatment options for lumbar stenosis and radiculopathy

In relation to the treatment of lumbar spinal stenosis and lumbar radiculopathy, the treatment options remain similar. For spinal stenosis, conservative management should remain the first option. The use of medication management, corresponding to those for NSLBP, is one option.[5,10] Limited evidence exists for the benefit of SMT in patients with stenosis.[5] Exercise therapy consisting of spinal stretching and strengthening should be employed to prevent deconditioning.[10] Invasive therapies, including 30% of all epidural spinal injections, are employed for patients with lumbar stenosis, with only limited, short-term benefit of no more than 3 weeks based on evidence-based guidelines for patients with neurogenic claudication.[5,10]

Prior to beginning invasive interventions, such as spinal injections or surgery, correlation with spinal imaging is recommended. For patients who have failed conservative treatments, referral to a specialist for consideration of surgical decompression, typically with lumbar laminectomy, has been shown to be supported by the guidelines based on long-term follow-up studies.[5,6,10]

For lumbar radiculopathy, conservative treatments are recommended for the first 6 to 8 weeks beginning with patient education and avoidance of bed rest.[5] There is limited evidence supporting the use of analgesics, no evidence supporting muscle relaxants, and no support of the use of antidepressants for patients with lumbar radiculopathy but typically medication management is provided following the same guidelines for NSLBP.[13] In addition, traction, corset use, acupuncture, physical therapy, and SMT have little or no evidence for or against their recommendation based on best practice guidelines, although the North American Spine Society stated a short-term structured exercise program can be presented to patients with mild-to-moderate radiculopathy.[8,13] Secondary treatment of an epidural corticosteroid injection was recommended for short-term benefit for patients with lumbar radiculopathy, but there was a lack of evidence supporting more than a series of three repeated injections.[8,13,16,17]

Once again, prior to beginning invasive interventions, such as epidural injections or surgery, correlation with spinal imaging is recommended. Clinical evidence indicates insufficient data to support the use of intradiscal electrothermal annuloplasty, plasma disk decompression/nucleoplasty, intradiscal high-pressure saline injection, or low-power laser for the treatment of lumbar radiculopathy.[13] For patients who have failed conservative treatments, referral to a specialist for consideration of surgical discectomy has been shown to be a cost-effective treatment supported by the guidelines, especially for those with progressive muscle weakness or altered bladder or bowel function.[13]

New research on causal factors for LBP

New research is discovering various types of treatment options evaluating possible causal factors for NSLBP and back pain with radicular symptoms. An increase in tumor necrosis factor-alpha is one probable cause.[2] Genetic predisposition has been documented involving changes to interleukin-1; aggrecan, the vitamin D receptor; genes responsible for various collagen fibers, including I, IX, XI; matrix metalloproteinase 3; and many proteins.[2,4]

Obesity has been recently studied as a causal factor in relation to increased mechanical load, systemic chronic inflammation, association of increased abdominal obesity causing metabolic syndrome changes that may affect the disk material, and a decrease in spine mobility.[10,30] Lastly, a small association between smoking status and NSLBP has been identified in cohort studies, with many relating this to changes in the vascular supply to the intervertebral disk.[2,4,10]

Educating patients

Because LBP is one of the most common diagnoses presenting to primary care offices, NPs can see the importance in performing excellent history and physical exam to assist in clarifying pathology from back pain with or without radicular symptoms. In addition, with the variety of pharmacologic and nonpharmacologic treatments employed for LBP, providers will need to make educated decisions on how to best manage these conditions.

Educating patients on their diagnosis and maintenance of an active lifestyle, including them in the decision-making and assessing for psychosocial risk factors, can assist in preventing the progression from acute to chronic pain. Through the review of evidence-based practice, extracting the recommendations from the research, and employing these recommendations into everyday practice, providers can improve outcomes for patients with LBP. NP

REFERENCES

1. Chou R. Pharmacological management of low back pain. *Drugs*. 2010;70(4):387-402.
2. Balagué F, Mannion AF, Pellisé F, Cedraschi C. Non-specific low back pain. *Lancet*. 2012;379(9814):482-491.
3. Foster NE. Barriers and progress in the treatment of low back pain. *BMC Med.* 2011;9:108.
4. Taher F, Essig D, Lebl DR, et al. Lumbar degenerative disc disease: current and future concepts of diagnosis and management. *Adv Orthop*. 2012;2012:970752.
5. Kreiner DS, Shaffer WO, Baisden JL, et al. An evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis (update). *Spine J.* 2013;13(7):734-743.
6. Depalma MJ, Ketchum JM, Trussell BS, Saullo TR, Slipman CW. Does the location of low back pain predict its source? *PM R.* 2011;3(1):33-39.
7. Shultz S, Averell K, Eickelman A, Sanker H, Donaldson MB. Diagnostic accuracy of self-report and subjective history in the diagnosis of low back pain with non-specific lower extremity symptoms: a systematic review. *Man Ther.* 2015;20(1):18-27.
8. Walker J. Back pain: pathogenesis, diagnosis and management. *Nurs Stand.* 2012;27(14):49-56.
9. de Schepper EI, Overdevest GM, Suri P, et al. Diagnosis of lumbar spinal stenosis: an updated systematic review of the accuracy of diagnostic tests. *Spine.* 2013;38(8):E469-E481.
10. Costandi S, Chopko B, Mekhail M, Dews T, Mekhail N. Lumbar spinal stenosis: therapeutic options review. *Pain Pract.* 2015;15(1):68-81.
11. Cook C, Brown C, Michael K, et al. The clinical value of a cluster of patient history and observational findings as a diagnostic support tool for lumbar spine stenosis. *Physiother Res Int.* 2011;16(3):170-178.
12. Ammendolia C, Stuber KJ, Rok E, et al. Nonoperative treatment for lumbar spinal stenosis with neurogenic claudication. *Cochrane Database Syst Rev.* 2013;(8):CD010712.
13. Kreiner DS, Hwang SW, Easa JE, et al. An evidence-based clinical guideline for the diagnosis and treatment of lumbar disc herniation with radiculopathy. *Spine J.* 2014;14(1):180-191.
14. Iversen T, Solberg TK, Romner B, et al. Accuracy of physical examination for chronic lumbar radiculopathy. *BMC Musculoskelet Disord.* 2013;14:206.
15. Kongsted A, Kent P, Albert H, et al. Patients with low back pain differ from those who also have leg pain or signs of nerve root involvement—a cross-sectional study. *BMC Musculoskel Dis.* 2012;13:236
16. Herndon CM, Zoberi KS, Gardner BJ. Common questions about chronic low back pain. *Am Fam Physician*. 2015;91(10):708-714.
17. Dagenais S, Tricco AC, Haldeman S. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. *Spine J.* 2010;10(6):514-529.
18. Hoppenfeld S. *Physical Examination of the Spine and Extremities.* East Norwalk, CT: Appleton-Century-Crofts; 1976.
19. Hoppenfeld S. *Orthopaedic Neurology: A Diagnostic Guide to Neurologic Levels.* Philadelphia, PA: Lippincott Williams and Wilkins; 1997.
20. Scaia V, Baxter D, Cook C. The pain provocation-based straight leg raise test for diagnosis of lumbar disc herniation, lumbar radiculopathy, and/or sciatica: a systematic review of clinical utility. *J Back Musculoskelet Rehabil.* 2012;25(4):215-223.
21. Wassenaar M, van Rijn RM, van Tulder MW, et al. Magnetic resonance imaging for diagnosing lumbar spinal pathology in adult patients with low back pain or sciatica: a diagnostic systematic review. *Eur Spine J.* 2012;21(2):220-227.
22. Nadeau M, Rosas-Arellano MP, Gurr KR, et al. The reliability of differentiating neurogenic claudication from vascular claudication based on symptomatic presentation. *Can J Surg.* 2013;56(6):372-377.
23. Kuijpers T, van Middelkoop M, Rubinstein SM, et al. A systematic review on the effectiveness of pharmacological interventions for chronic non-specific low-back pain. *Eur Spine J.* 2011;20(1):40-50.
24. White AP, Arnold PM, Norvell DC, Ecker E, Fehlings MG. Pharmacologic management of chronic low back pain: synthesis of the evidence. *Spine.* 2011;36(21 suppl):S131-S143.
25. Bannwarth B, Kostine M, Shipley E. Nonspecific low back pain: assessment of available medications. *Joint Bone Spine.* 2012;79(2):134-136.
26. Balagué F, Piguet V, Dudler J. Steroids for LBP – from rationale to inconvenient truth. *Swiss Med Wkly.* 2012;142:w13566.
27. Standaert CJ, Friedly J, Erwin MW, et al. Comparative effectiveness of exercise, acupuncture, and spinal manipulation for low back pain. *Spine.* 2011;36(21 suppl):S120-S130.
28. Taylor P, Pezzullo L, Grant SJ, Bensoussan A. Cost-effectiveness of acupuncture for chronic nonspecific low back pain. *Pain Pract.* 2014;14(7):599-606.
29. Jones LE, Roberts LC, Little PS, Mullee MA, Cleland JA, Cooper C. Shared decision-making in back pain consultations: an illusion or reality? *Eur Spine J.* 2014;23(suppl 1):S13-S19.
30. Wertli MM, Held U, Campello M, Schecter Weiner S. Obesity is associated with more disability at presentation and after treatment in low back pain but not in neck pain: findings from the OIOC registry. *BMC Musculoskelet Disord.* 2016;17:1-14.

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