The Lumbar Spine: Evidence-Informed Physical Therapy Patient Management

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ABSTRACT

Physical therapists are uniquely positioned in the health care system to treat individuals with low back pain (LBP). This monograph highlights some of the best available evidence as it relates to the role of physical therapy in the evaluation and management of LBP. The current research suggests that despite the great advances in treatment, the overall prevalence of LBP and LBP-related disability seem to be largely unchanged. This monograph will review the evidence regarding the validity and importance of structural abnormalities as determined by magnetic resonance imaging, and what functional changes are commonly present in patients with chronic LBP (cLBP). Since the last edition of *Current Concepts for Orthopaedic Physical Therapy* was published, updated evidence-based guidelines have been published and will be reviewed in this monograph. Up-to-date information regarding clinical outcome measures like the Optimal Screening for Prediction of Referral and Outcome and how they can be incorporated into physical therapy practice will be reviewed. The evidence behind a variety of treatment interventions will be assessed including various exercise approaches and manual therapy techniques. In addition, the role of education and emerging treatment strategies will be discussed. Following the body of this manuscript the reader will be presented with 4 case scenarios, each will require strong clinical reasoning skills as well as an understanding of the current evidence for the proper treatment and evaluation of the patient. These case scenarios will include patients who have both acute and cLBP with accompanying psychosocial risk factors.

Key Words: education, flags, psychosocial, treatment

LEARNING OBJECTIVES

Upon completion of this monograph, the course participant will be able to:

1. Describe the prevalence of low back pain (LBP), the differences between the point estimates of acute and chronic LBP (cLBP), and how racial and socioeconomic factors can contribute to the conditions.
2. Discuss the relative role of diagnostic imaging and the clinical implications of positive findings.
3. Discuss the different structural and functional changes that can occur with cLBP and the implications of each.
4. Understand the impact of psychosocial risk factors in LBP and identify potential treatment approaches to each.
5. Describe the predictors for the transition of acute to cLBP and how to use the different outcome measures discussed.
6. Use evidence-based clinical guidelines in the treatment of patients with acute and cLBP.
7. Detect yellow and red flags and understand their clinical implications.
8. Integrate into clinical practice the research behind common physical therapy interventions such as: motor control exercises, graded activity/exposure, directional-based exercise approaches, aerobic exercise, spinal manipulation/mobilization, dry needling, and education.
9. Differentiate between pathoanatomical education and pain neuroscience education and the clinical utility of each.
10. Discuss the importance of the therapeutic alliance and strategies to incorporate this alliance into clinical practice.

BACKGROUND

Prevalence and Chronicity

The economic and health burden of low back pain is still on the rise

As it relates to the musculoskeletal system, there is no condition more prevalent or costly as low back pain (LBP).[1,2] The prevalence and disability rate of LBP continues to rise[1,3,4] while the economic burden to the United States economy remains great.[5,6] It is estimated that the United States spends approximately $90 billion annually on the treatment of LBP alone.[7] According to the Global Burden of Disease Study,[1] LBP was a third-tier cause of disability in 1990. Fast-forward 27 years, in the 2017 update, disability caused by LBP increased by 30% to claim the prominent number 1 condition of disability among industrialized nations. In 2009, Freburger et al[4] reported that the percent of the population suffering from chronic LBP (cLBP) rose from 3.9% to 10.2% between 1990 and 2006. Disability rates have also increased; during a similar time period years lived with disability increased by almost 25%.[8] Chronic LBP is also the most common reason for both physician[9] and physical therapy visits.[10] For these reasons, LBP remains an important focus of physical therapy practice.

While the impact of LBP is indisputably high, the exact prevalence of the condition at any given time is difficult to determine. A recent systematic review by Hoy et al[3] estimated the point prevalence of LBP (defined as the amount of people with this condition at any given time) to be 11.9%. However, estimates of LBP can vary widely in the literature and are largely determined by the window of observation used. For example, in 2002, Deyo et al[9] reported that 26.4% of the population experienced LBP in the last 3 months. This finding was consistent with findings by Yang et al[11] who, using a similar 3-month window of observation, reported the prevalence of LBP in 2010 to be 25.7%. However, when one expands the window of observation to 1 year, the incidence of LBP can rise to 65%.[12] Lifetime prevalence of LBP can range between 70% and 84%.[12,13] Another factor that complicates the estimate of the prevalence

of LBP is the difference between acute and cLBP. While the lifetime incidence of LBP is quite high, the prevalence of cLBP is lower. A recent systematic review of 28 studies by Meucci et al[14] found that the prevalence of cLBP ranged anywhere from 2% to 25.4%. Part of the variability of the reported incidence was secondary to the stratification of different age groups. However, across the different studies the prevalence in chronic pain typically increased until about the 7th decade of life, at which time the prevalence stabilized or reduced slightly.

The burden of low back pain is not borne equally in our society

In the above studies, the prevalence of LBP was assessed for the population as a whole. However, there is mounting evidence that the distribution of LBP burden is not equal between race, sex, and socioeconomic status (SES). While the intent of this monograph is not to examine all the interactions between health care and race, sex, and SES, a brief look at some recent studies is warranted. In 2019, Brandão et al[15] found that pain from patients from low-SES was considered less intense and more likely to be the result of psychological influences compared to patients from high-SES. Viewing someone's pain as predominantely psychological as opposed to physiological might have the unfortunate side effect of deeming the pain to be less credible and changing one's course of treatment. Those with lower SES also experience cLBP and subsequent disability at higher rates.[16,17] However, low-SES alone does not account for many of the inequities in health care as it relates to cLBP. In 2020, Anastas et al[18] performed a study where physicians made pain care decisions for 12 computer-simulated patients with chronic back pain. They found that among patients with low-SES, White patients were more likely to receive workplace accommodations compared to Black patients. Additionally, for patients with high SES, Black patients were more likely to be rated as being in distress compared to White patients; indicating greater psychological influence on their symptoms. While studies have shown that White and Black Americans experience cLBP at equal rates[19]—with the potential of White Americans experiencing cLBP at slightly higher rates[13,14]—Black Americans experience more severe and disabling LBP even independent of SES.[19-21] Unfortunately, this bias extends into treatment approaches as well. In 2018, Kohns et al[22] assessed treatment outcomes of 600 adults from 3 different hospital settings. They found that Black Americans were less likely to receive advanced imaging and were less likely to receive opioids. While one might argue that—as will be discussed later on in this monograph—less opioids and imaging might inadvertently be better medical care, the paper highlighted discrepancies in treatment between races. Black Americans were also found less likely to receive physical therapy for cLBP.[19] Disparities exist across sex as well with females routinely experiencing cLBP at higher rates and greater intensities than males.[13,14,23] These studies highlight the need for physical therapists to ensure that equal care and concern is given to all patients regardless of race or sex and that biases, conscious or unconscious, have no role in the treatment of cLBP.

Take Home Messages

- The prevalence and burden of LBP continues to rise.
- Approximately 65% of the population will experience at least 1 episode of LBP in a year.
- The burden of LBP is not borne equally in our society, with minorities and women exhibiting greater pain, greater disability, and poorer treatment.

Pathophysiology

Acute and chronic low back pain are very different

It should come as no surprise that acute and cLBP are very different conditions. While some arguments exist regarding the optimal way to classify pain as "chronic," according to the recent ICD-11 guidelines, chronic pain is "persistent or recurrent pain lasting longer than 3 months."[24] Therefore, throughout this monograph we will consider cLBP as pain lasting greater than 3 months while acute LBP (aLBP) describes a condition with symptoms occurring for less than 3 months.

The natural history of acute low back pain

The prognosis for aLBP is generally favorable, with reports of complete recovery ranging anywhere from 72% to 90% within a year of diagnosis[25-27] (although, it should be mentioned that the 90% figure has come under considerable debate).[28,29] One of the most thorough examinations of the typical course of aLBP was undertaken by Henschke et al in 2008.[25] The authors studied a cohort of 973 patients with recent onset LBP and followed their progress over the course of 1 year. As it related to pain, they found that the probability to be pain free after 6 weeks was 39.9% and by the end of 1 year that probability rose to 72.5%. Interestingly, disability had a better prognosis. By the end of 6 weeks the probability of having no disability was 54.9% and by the end of 1 year that probability rose to 83.3%. This is an important finding as it highlights that a return-to-work criteria for LBP may not need to be a complete resolution of symptoms. Rather, it could be argued that individuals with LBP can participate in the workplace even if their pain has yet to completely resolve.

Another important concept of aLBP is that recurrence rate is high.[28,30,31] Recurrence of aLBP within a year can be as high as 66% to 84%[32] with a similar 3-year recurrence rate.[33] Even within a shortened time span of 3 months, up to 10.5% of patients can have fluctuations in their symptoms.[34] This indicates that physical therapy does not necessarily result in a long term "fix" for LBP for a large portion of patients. Perhaps instead physical therapy should be viewed as a means to reduce current

LBP and a potential vehicle by which a patient can improve their self-efficacy in their own pain management.

The natural history of chronic low back pain

People with cLBP typically present with more comorbidities than do people with aLBP. For example, those with cLBP typically present with symptoms of central sensitization,[35,36] structural alterations in the lumbar musculature,[37] and even changes in the processing of pain in the brain.[38] These changes can make cLBP resistant to treatment,[39-42] leading to the long held belief that those with cLBP generally have a poor prognosis.[32,43] However, in 2009, Costa et al[44] followed a cohort of 406 patients who developed an initial instance of cLBP. They found that 41% of all participants reported a complete recovery at 1 year and furthermore only 11% were not able to return to work. This information should be used to provide encouragement for those newly diagnosed with cLBP. It shows that many people with cLBP do get better with time and that those experiencing cLBP should not lose hope.

For some, however, cLBP continues to be a debilitating condition with which one must live. As is evident from the above examples, these individuals represent the minority of those with back pain. However, they account for an outsized proportion of the health care resources and economic impact. Therefore, the focus of much of this monograph will be on the consequences of cLBP.

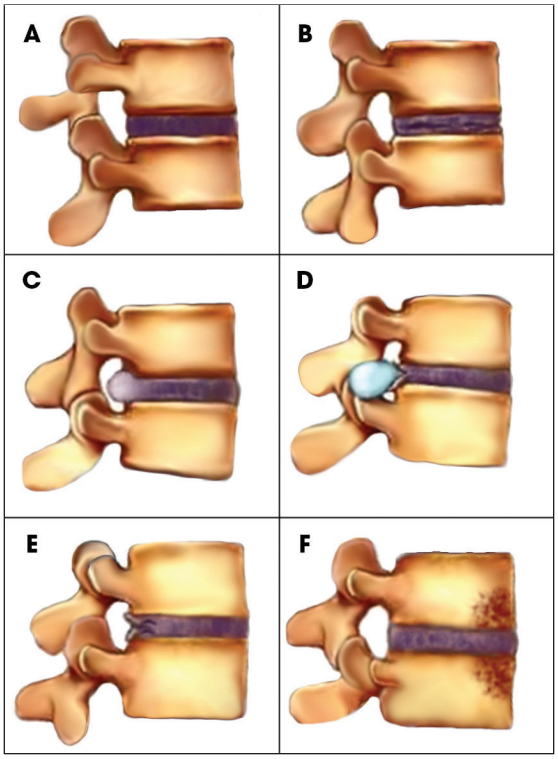
Structural changes associated with low back pain

Structural changes associated with LBP can be thought of in 2 general categories: peripheral and central. Peripheral structures that are thought to be associated with LBP can include the following: intervertebral disc (IVD), facet joints, ligamentous structures (specifically the ligamentum flavum), vertebral bodies (fractures, spondylolysis, etc), and the lumbar paraspinal musculature.[45] It is not the intent of this monograph to review every possible structural change that can result in LBP. However, of the structures listed above, few have been given the attention that the IVD has (**Figure 1**).

The lumbar intervertebral discs are integral to the spine

The IVD serves several very important roles in the lumbar spine. It helps the spine absorb and transmit shock, maintains flexibility, and under higher loads, helps to stabilize the spine.[46] The normal disc is composed of an outer layer of fibrocartilage that surrounds an inner layer comprised of a well hydrated proteoglycan gel. Working together these 2 layers are able to provide resistance in various combinations of compression, shear, and tensile forces.[47] During spinal loading, the IVD is able to distribute the load evenly on the vertebral bodies.[48] Additionally, when the IVD is healthy, it provides a "spacer effect." As spacers, the IVDs provide about 25% of the total height of the lumbar spine. The greater the height, the greater the mobility of the lumbar spine and the greater the space for adequate passage for exiting spinal nerve roots.

Figure 1. Common Disc Pathologies



A, Normal disc. B, Degenerative disc. C, Bulging disc. D, Herniated (Extruded) disc. E, Tear in posterior annulus representing pathology of the High Intensity Zone. F, Modic change (Type 1).

A normal, healthy, IVD is composed of 2 primary layers: the outer annulus fibrosus and the inner nucleus pulposus. The outer annulus is comprised of 15-25 concentric layers—lamellae—that are primarily composed of Type I cartilage.[46,47,49] Each concentric layer is oriented approximately 45° from the transverse plane.[47,50] This alternating orientation of the concentric layers is what helps the anulus fibrosus resist tensile forces in each direction as well as providing resistance to torsional forces. Additionally, between each lamellae, there is a collagen-based bridging network that provides resistance to any shear forces.[51,52] Therefore, with the combination of the alternating concentric layers, the trans-lamellae bridges, and the tough Type I collagen fibers found through the annulus fibrosus, the disc is allowed to provide significant resistance against distraction, shearing, and torsional forces.

The inner portion of the disc, or the nucleus pulposus, is a gelatinous structure that accounts for approximately half of the volume of the IVD.[53,54] Approximately 70% to 90% of the nucleus pulposus is comprised of water, creating great hydrostatic pressure.[47,50] In fact, intradiscal pressures within the lumbar

spine have been found to range from 100 kPa (laying supine) all the way up to 2300 kPa (lifting 20 kg, bent over with round back).[55] Under compressive loads the increasing hydrostatic pressure will generate tension in the surrounding anulus fibrosus, increasing the stability of the spine.[56] Located within the nucleus pulposus are glycosaminoglycans bound to a proteoglycan molecule that act to thicken the surrounding water.[47,57] The thickening of the water within the nucleus pulposus acts to increase the load-bearing capacities of the lumbar spine.[47]

The vertebral endplate borders the IVD on the superior and inferior vertebral body. Typically, the endplates are less than 1 mm thick but it varies from region to region.[58,59] They create a semi-permeable barrier between the vertebral body and the IVD that allows for the diffusion of some nutrients into the largely avascular inner nucleus.[47] While the portion of the end plate that borders the IVD is generally comprised of strong fibrocartilage, where the end plate borders the vertebral body it is primarily comprised of hyaline cartilage.[47] This creates a potential area of weakness that, when the IVD is exposed to trauma or simply over time, might lead to the nuclear material of the IVD protruding into the vertebral marrow.[60] This is thought to contribute to the development of disc degeneration.[58]

Disc degeneration occurs naturally and does not always result in pain

Disc degeneration has commonly been associated with LBP.[61-63] It has been shown that as an individual ages the water content within the nucleus decreases, causing the annulus fibrosus to resist compression.[48] This change can lead to decreases in both disc flexibility and height.[63] With the loss of disc height, the potential for foraminal narrowing increases as well as increased facet arthritis[63] and spondylosis. However, the impact of the mere presence of a degenerated disc is debated.

There is now substantial evidence that shows as one ages, the incidence of disc degeneration (DD) increases.[64-66] In fact, it was found that individuals over the age of 50 had an 88% likelihood of having a degenerated disc in their lumbar spine.[65] However, this naturally occurring phenomenon does not always correlate with pain. In 2015, Brinjikji et al[67] performed a systematic review of the literature that assessed the prevalence of asymptomatic spinal degeneration across the lifespan. They included 33 articles in their systematic review that had a total of 3110 asymptomatic individuals. They found that the incidence of lumbar DD was approximately 37% in individuals in their 20s, and that percentage increased to 96% by the time an individual reached their 80s. In fact, that systematic review showed that by the time an individual is in their 30s, they are *more likely to have a degenerated disc than not*. A later study that highlighted the ubiquitous nature of DD was performed in 2019 by Romeo et al.[68] In that study they assessed the incidence of spinal abnormalities in a population of 350 asymptomatic young adults (between the ages of 18 and 22 years). They found that 30% of participants had signs of a disc desiccation and 13% presented with at least 1 disc narrowing. Clearly, the implications of a positive finding of DD should be taken with a grain of salt.

It should be noted, however, that evidence does exist that, despite the wide spread prevalence of degenerative disc disease (DDD), symptomatic individuals are more likely to have DD than asymptomatic individuals.[65,69] In the same year they published their systematic review of the prevalence of DDD in an asymptomatic population, Brinjikji et al[69] performed a meta-analysis comparing the prevalence of DDD in symptomatic and asymptomatic individuals. They included 14 studies with a total sample size of 3097 individuals, all were 50 years of age or less. They reported that individuals with LBP are 2.24 times more likely to have DD compared to asymptomatic individuals. However, it should be noted that due to the high prevalence of naturally occurring degeneration in the spine, the authors did not include individuals older than 50 years of age. This led them to the conclusion that "the association between these entities and LBP [may be] less significant in older age groups." To further support the notion that DDD is more prevalent in a symptomatic population, a more recent study has shown that DD, especially in the presence of additional degenerative findings in the lower lumbar spine, is more likely to be correlated with LBP. This brings up a very pertinent question: how can one differentiate between symptomatic and asymptomatic DD?

How do we differentiate an asymptomatic degenerative disc from a symptomatic one?

Several studies have focused on answering the question of how to differentiate a symptomatic versus an asymptomatic degenerative disc. The focus of this work has largely revolved around the presence of a high intensity zone (HIZ) within the posterior aspect of the annulus fibrosus. First discussed in 1992 by Aprill and Bogduk,[70] the HIZ is defined as the "high signal contained within the annulus of a disc, separated from the nucleus pulposus, on lumbar spine MRI." According to their initial research they deemed the sign as diagnostic of internal disc disruption. Since then, significant work has been done to examine the pathogenesis and clinical significance of the HIZ. In 2006, Peng et al[61] performed biopsies on 21 discs that were found to have both an HIZ in the posterior region of the annulus and reproduced pain during discography. After performing a histological study of the disc and with supportive data from their concurrent study,[62] they described a potential mechanism for how an HIZ might become pathologic. They hypothesized that a tear in the structurally weak[71] posterior annulus occurs from some unspecified trauma. Following the tear, the normal structure of the posterior annulus is replaced with granulation tissue. However, due to the poor vascularization of the IVD the healing is defective.[62] Therefore, following the injury there is an "abundance of inflammatory mediators, specifically prostaglandin E_2, IL-6, and IL-8 that can sensitize the nociceptors in the disc."[61] With movements of flexion and extension, the increase

in the intradiscal pressure might be enough to elicit a nociceptive signal from the disc.

While the work from Peng et al[61] provides a plausible biological mechanism for HIZ being related to symptomatic DDD, epidemiologic data supporting the notion that the HIZ is associated with pain is inconclusive. Wang et al[72] reviewed the lumbar magnetic resonance imaging (MRI) of 637 participants. Of the participants, roughly half had symptoms of LBP while the other half were asymptomatic. While they found that the rate of HIZ was statistically higher in those with LBP, the difference was not large. Those with LBP had an incidence rate of 36% while those without LBP had an incidence rate of 27%. Other studies found the prevalence of asymptomatic HIZ to be as high as 56%[73] although 20% to 32% is more common.[74-76] These studies[73-76] also found a correlation between increased age and weight with increased prevalence of HIZ, suggesting that the development of HIZ could be part of the natural aging process. In a different study, Campos et al[77] assessed the prevalence of HIZ in adults who underwent an abdominal and pelvic MRI as a screening tool. While they found that HIZ was significantly more prevalent in asymptomatic discs with degeneration, the overall prevalence was at 11.06%. This was actually a higher prevalence rate than the symptomatic prevalence rate (10.4%) that Brinjikji et al[69] reported in their meta-analysis in 2015.

Conversely, to support the use of HIZ as a diagnostic parameter to determine if DD contributes to LBP, Fang et al[78] performed a meta-analysis combining the data of more than 11 studies. They specifically looked at the relationship between pain reproduction based on provocative discography and the HIZ. Their sample, by nature of their inquiry, only contained individuals who had LBP. They reported that the odds are 8.64 times greater that a disc with an HIZ will result in a positive provocative discography compared to a disc without an HIZ. This led them to the conclusion that the presence of a HIZ and pain are linked. Furthermore, Waldenburg et al[79] examined the differences in IVD characteristics as measured with quantitative MRI and found that there were small but significant differences in the tissue composition between asymptomatic and symptomatic IVD. Specifically, they found decreased heterogeneity between the nucleus pulposus and the annulus fibrosis in the posterior portion of discs in symptomatic participants. However, when they excluded the IVDs which demonstrated HIZs, difference could no longer be found between symptomatic and asymptomatic IVDs. This led them to the conclusion that the quantitative differences they found in symptomatic and asymptomatic discs were the result of the presence of the HIZs.

The shortcoming of both of the above studies is that they did not address the significant presence of asymptomatic discs that have a HIZ. In the meta-analysis performed by Fang et al,[78] the general conclusion is that provocative discs as measured by discography have significantly increased odds of also having a present HIZ. This does not explain the high proportion of asymptomatic discs having a HIZ. Waldenburg[79] demonstrated that differences in the quantitative measures in IVDs between symptomatic and asymptomatic patients could be attributed to the HIZs, but did not explain why asymptomatic patients have HIZs in the first place. Ultimately, the prevalence of HIZ is too great in the asymptomatic population for meaningful clinical use[75] and the results of MRI alone is not enough to definitively conclude that a disc is symptomatic or asymptomatic.[80] More work is needed to fully elucidate the clinical relevance of the HIZ.

A disc by any other name…

Another potential source of pathoanatomical pain in the lumbar spine could be the presence of a bulging disc.[81] However, clinicians and medical professionals use different terms for the same observed pathology, leading to confusion.[82,83] Therefore, this monograph will attempt to use the terminology proposed by the combined task forces of the North American Spine Society, the American Society of Spine Radiology, and the American Society of Neuroradiology in their publication "Lumbar Disc Nomenclature: Version 2.0."[84] With that in mind, the following definitions will be used. A bulging disc, which is not considered a type of herniated disc, is the presence of disc tissue extending beyond the edges of the ring apophyses throughout the circumference of the disc (**Figure 1C**). A herniated disc **(Figure 1D)** can be subdivided into 2 categories: protrusion or extrusion. A protruded disc is present "if the greatest distance between the edges of the disc material presenting outside the disc space is less than the distance between the edges of the base of that disc material extending outside the disc space." A disc extrusion is defined when "any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base of the disc material beyond the disc space." A sequestration can exist if the disc material beyond the disc space is no longer connected to the remaining disc and is considered a subcategory of a disc extrusion.

What role does a herniated (or bulging) disc play in low back pain?

There is strong evidence that both bulging and herniated discs occur in asymptomatic individuals. Since the early 1990s the presence of asymptomatic bulging and herniated discs has been described.[85,86] Looking back at the systematic review by Brinjikji et al,[67] a bulging disc was found in 30% of individuals in their 20s and up to 84% of individuals in their 80s. Disc protrusions were found in 29% of individuals in their 20s and by the time an individual reaches their ninth decade of life that prevalence increases to 43%. Kim et al[87] reviewed the MRI of 102 participants and found that the incidence for an asymptomatic bulging, protruded, or extruded disc was 61.3%, 46.3%, and 31.7%, respectively. Romeo et al[68] demonstrated the prevalence of asymptomatic bulging and protruding discs in young adults to be as high as 49% and 26%, respectively. What is interesting is that in 2 more recent publications,[68,87] using

a 3T MRI as opposed to the lower strength MRI of previous studies, a higher prevalence of asymptomatic discs was found. This raises the question that as technology improves and the accessibility to better equipment is greater, is the chance of finding false-positive imaging even higher?

Herniated discs get better with time

One piece of very encouraging news for patients who have been diagnosed with a herniated or bulging disc is that, with time, the probability of a spontaneous resolution of the herniated disc is quite high. Chiu et al[88] performed a systematic review of the literature assessing the probability of spontaneous regression of herniated and bulging discs in the lumbar spine. The results of their study are astounding. After combining the data from 9 separate studies, they found that the rate of spontaneous regression was 96% for a sequestered disc, 70% for an extruded disc, 41% for a protruded disc, and 13% for a bulging disc. While most spontaneous regression occurred within the first year—with the initiation of regression being observed as early as 2 to 3 months—it was found to take some individuals up to 40 months to experience full regression. What is interesting is that as the severity of the condition increased, the likelihood of spontaneous resolution also increased. However, it should be noted that the disc regression did not correlate well to clinical outcome. Since then, this finding has been supported by additional research.[89,90] This should come as great relief for patients and as a powerful tool for the physical therapist. When patients come into the clinic having been diagnosed with a bulging/herniated disc, the astute physical therapist can explain to them that not only is their disc likely to recover on its own, but the worse it is on imaging, the more likely the resolution.

The potential role of endplate changes in low back pain

In recent years changes in the vertebral endplates and subchondral bone have been further evaluated as a potential source of LBP. Commonly referred to as Modic changes, these clusters of findings represent changes in the vertebral endplate and vertebral bone marrow lesions.[91,92] Modic changes type 1 are hypothesized to represent an inflammatory reaction in the bone marrow (**Figure 1F)**, Modic changes type 2 are associated with fatty infiltration of the bone marrow, while Modic changes type 3 are associated with sclerotic change of the vertebral bone marrow.[92] Modic changes, specifically type 1, have been associated with the presence of LBP;[69] however, the association is not clear. A meta-analysis of 31 studies was performed by Herlin et al[92] where they attempted to elucidate the relationship between the presence of Modic changes and the presence of pain and/or activity limitation, as well as if this relationship can be modified by other factors. First, they found that the association between Modic changes (all types) and LBP was tenuous. Only approximately half of the studies they evaluated demonstrated a statistically significant association between the Modic changes and LBP. When they stratified their data based on Modic change type, they found no significant differences in the strength of the associations. Other findings of interest from the study included the fact that the size of the Modic change did not correlate with pain intensity, there was no difference in the pain intensity between patients with and without Modic changes, and finally that there was no support for an association between the Modic changes and activity limitation.[92] Interestingly, another systematic review in the same year, which assessed the relationship between Modic changes and LBP, also found that only half of their studies demonstrated a statistically significant association.[93]

Other systematic reviews have attempted to determine the role of Modic changes in the relationship to LBP, concluding that the presence of a type 1 Modic change has a non-significant association with postoperative clinical outcomes;[94,95] Modic changes are not associated with occupational loading;[96] and the presence of Modic changes cannot guide treatment.[97] Furthermore, single studies have also called into question the clinical utility of Modic changes.[98,99] Much like HIZ, the prevalence of Modic changes in the population at large can be high and the prevalence increases as one ages. Wu et al[100] found that approximately 45% of individuals in their population-based study had Modic changes (although it should be noted that they did not include how many of their participants had LBP) and that the prevalence increased with age. In fact, after adjusting for age, sex, and body mass index, there were no statistically significant associations between Modic changes and other lifestyle factors. This helps support the argument that Modic changes can and do occur naturally with age.

Structural changes in the central nervous system can occur with low back pain

In recent years, more and more researchers are looking at changes within the brain to help explain some of the symptoms associated with LBP. These efforts have largely fallen into 2 categories: changes in functional activity and structural morphology. Functional activity relates to how different parts of the brain activate or communicate with one another. Structural morphology, which will be the focus of this section, largely relates to changes in the gray matter volume (GMV).

Two recent systematic reviews have examined the effects of LBP on the structural morphology of the brain. In 2015, Kregel et al[101] examined the reported structural organization of grey matter (GM) in 10 studies. While there was inconsistent reporting in multiple brain regions, 3 regions stood out. Reduced GMV was reported for the dorsal lateral prefrontal cortex (DLPFC), temporal lobes, and insula. There were mixed results for the somatosensory cortex (S1) with studies showing both increased and decreased GMV. Reduction in the insula makes intuitive sense as it plays an integral role in both sensory and emotional component of pain processing.[102] However, the reduction in the gray matter of the DLPFC is of particular

interest because it has been shown to limit the magnitude of perceived pain.[103] Disruption of this region might signify that individuals with cLBP are less able to actively control their pain perception and not able to modulate nociception.[103]

A more recent systematic review confirmed several of these findings. In 2018, Ng et al[104] examined the literature and reviewed the results of 55 imaging studies. They found further evidence of decreased GMV in the DLPFC, insula, and temporal lobe, with mixed results for the S1 region. Furthermore, they found evidence of decreased GMV within the cuneus, thalamus, medial prefrontal cortex (mPFC), the posterior cingulate cortex (PCC), as well as the precentral region of the brain. Without examining the specific role of each region and to simply summarize the authors' remarks, they concluded that many of the regions demonstrating altered GMV were associated with emotion and cognition rather than nociception.[104] Since the publication of this systematic review, another study has been published with similar findings of decreased GMV in the DLPFC.[105]

While the results of these 2 systematic reviews indicate structural change following cLBP, it should be noted that their results are not universal. Mansour et al[106] performed a region-of-interest analysis to more systematically examine the regions that have been previously found to demonstrate differences in volume. After enrolling a total of 130 participants who had either subacute or cLBP, or no pain at all, they demonstrated no differences in GMV. However, they did not include the DLPFC in their assessment. An additional study found that after controlling for age, depression/anxiety, pain medication, and pain phenotype, any reductions in GMV disappeared.[107]

Assessing morphological changes in GM is a relatively recent endeavor with the first study published in 2004.[108] While the full extent of the changes that occur during cLBP requires further exploration, the bulk of the literature at this moment suggests that there are changes in the GMV for the DLPFC, insula, temporal lobe, and the S1 region. The fact that changes occur in these 4 regions is important as the structural changes brought on by pain are not necessarily permanent.

Changes in gray matter volume can reverse with treatment

As previously mentioned, one of the key regions that has been shown to have decreased GMV is the DLPFC.[101,104] Seminowicz et al[109] performed a longitudinal study of 18 patients with cLPB and 14 healthy controls. They performed an MRI both before and 6 months after treatment (either surgery or injection). At baseline measure, they found that the cortical thickness of the DLPFC for those with cLBP was thinner than for the controls. However, for those with cLBP who responded positively to the intervention (n=11), cortical thickness increased, whereas for those who did not respond to treatment (n=3), the thickness remained the same. The authors observed the initial thinning of the cortex and subsequent thickening in responders even after controlling for depression.

The previous systematic reviews also demonstrated that the cortical thickness of S1 was altered in patients with cLBP.[101,104] Kim et al[110] recruited 102 participants with cLBP and 50 age-matched controls. Those with cLBP were randomized into 4 groups: traditional acupuncture, sham-acupuncture with and without somatosensory afference, and no treatment. The authors took MRI scans at baseline and again after treatment (duration between the assessments was a mean ± standard deviation of 7.0 ± 2.7 weeks). At baseline, they found that those with cLBP demonstrated decreased 2-point discrimination threshold in the low back but not when testing a finger, indicating site specific decreases in sensation. Furthermore, this decrease in tactile acuity was associated with an increase in S1 region GMV (interestingly, Li et al[105] in their study in 2018 found a similar increase in S1 region GMV). Following treatment, those in the true acupuncture group who responded to treatment demonstrated improved 2-point discrimination thresholds and reduced S1 region GMV. Interestingly, those in the sham acupuncture groups and in the no treatment group demonstrated no significant change. Therefore, they concluded that the change in S1 region GMV was associated with a decrease in 2-point discrimination, and that both improved after treatment along with a reduction in symptoms.

Wrapping it up: What are the structural causes of low back pain?

As is evident by now, it is difficult to determine what the anatomical cause of LBP is. Multiple studies looking at changes in GMV, DD, bulging discs, end-plate changes, stenosis, etc have found conflicting results as to which degenerative change is pathologic and which is benign. Part of the problem with trying to pinpoint a pathoanatomic cause for cLBP is that there are so many possible causes for the pain. The HIZ or the DD might be only one of many findings and as such a single source cannot be identified. It might be that the patient's lumbar spine MRI is normal, but there have been structural changes within the GM of the DLPFC that is causing the pain. This would, for example, diminish the association found between a DD and LBP. Additionally, while all patients with LBP have pain, that pain might be influenced by multiple factors other than nociception.[92] This would further diminish the correlation between any one degenerative change and pain.

The heterogeneous nature of LBP makes it difficult for the clinician because one of the primary reasons why patients seek health care is to obtain information on their condition.[111] Patients desire a clear explanation about the source of their symptoms,[111] and in trying to give an explanation as to what is causing the patient's symptoms, the clinician may be understandably reluctant to say, "the literature is unclear." The patient, also understandably, might interpret the clinician to mean, "I don't know what is wrong with you" or potentially think, "my therapist doesn't know what is going on." A lack of

perceived expertise on the part of the health care provider can have detrimental effects on the patient.[112] One solution to this dilemma might be to discuss some of the commonly promulgated myths about LBP.

In 2020, O'Sullivan et al[113] compiled a list of 10 myths that are commonly believed but not supported by evidence (**Table 1**). Some examples of myths taken from the article include the following: LBP is usually a serious medical condition; persistent LBP is always related to tissue damage; imaging scans are always needed to detect the cause of LBP; pain related to exercise and movement is always a warning that harm is being done to the spine and a signal to stop or modify activity; and treatments such as strong medications, injections, and surgery are effective, and necessary, to treat LBP. As an alternative, a therapist can provide the patient with the facts regarding their condition (as written by O'Sullivan et al)[113]: LBP is not a serious life-threatening medical condition; most episodes of LBP improve and LBP does not get worse as we age; imaging scans do not determine prognosis of the current episode of LBP, the likelihood of future LBP disability, and do not improve LBP; and effective care for LBP is relatively cheap and safe, including engaging in physical activity and exercise, social activities, healthy sleep habits and body weight, and remaining employed. By reviewing these "myths" and "facts" about LBP, perhaps the therapist can diffuse some of the patient's justified concerns about not knowing exactly what is happening in their low back.

Table 1. Myths versus Facts Regarding Low Back Pain as Defined by O'Sullivan et al[113]

Myth	Fact
LBP is usually a serious medical condition	LBP is not a serious life-threatening medical condition
LBP will become persistent and deteriorate in later life	Most episodes of LBP improve and LBP does not get worse as we age
Persistent LBP is always related to tissue damage	A negative mindset, fear-avoidance behavior, negative recovery expectations, and poor pain coping behaviors are more strongly associated with persistent pain than is tissue damage
Scans are always needed to detect the cause of LBP	Scans do not determine prognosis of the current episode of LBP, the likelihood of future LBP disability, and do not improve LBP clinical outcomes
Pain related to exercise and movement is always a warning that harm is being done to the spine and a signal to stop or modify activity	Graduated exercise and movement in all directions is safe and healthy for the spine
LBP is caused by poor posture when sitting, standing, and lifting	Spine posture during sitting, standing, and lifting does not predict LBP or its persistence
LBP is caused by weak 'core' muscles and having a strong core protects against future LBP	A weak core does not cause LBP, and some people with LBP tend to overtense their 'core' muscles. While it is good to keep the trunk muscles strong, it is also helpful to relax them when they are not needed
Repeated spinal loading results in 'wear and tear' and tissue damage	Spine movement and loading is safe and builds structural resilience when it is graded
Pain flare-ups are a sign of tissue damage and require rest	Pain flare-ups are more related to changes in activity, stress, and mood rather than structural damage
Treatments such as strong medications, injections, and surgery are effective, and necessary, to treat LBP	Effective care for LBP is relatively cheap and safe. This includes education that is patient-centered and fosters a positive mindset, and coaching people to optimize their physical and mental health (such as engaging in physical activity and exercise, social activities, healthy sleep habits and bodyweight, and remaining in employment)

Abbreviation: LBP, low back pain

As previously discussed, it is very difficult to pinpoint an exact pathoanatomical cause for LBP. In fact, Deyo and Weinstein[114] found that 85% of all cLBP has no known anatomical cause. Consistent with these findings, Hoy et al[31] reported that for 95% of all individuals with cLBP there was no known anatomical cause. However, not all changes that occur with LBP result in structural abnormalities.

Non-structural changes occur in individuals with low back pain

What is now clear is that LBP cannot consistently be attributed to an anatomical cause. For this reason, researchers have looked beyond the anatomy to look for an explanation.

People with low back pain move differently. Considerable effort has been made to determine motor control changes associated with LBP. As described by Sung et al,[115] motor control changes can be divided into 4 separate categories: altered muscle timing, changes in muscle quality, altered proprioception, and altered stiffness. The phenomenon of altered muscle timing largely revolves around the premise of altered recruitment of the abdominal obliques and transverse abdominis muscles prior to engagement of distal extremities.[116-118] The general premise is that the transverse abdominis and internal oblique will activate in an anticipatory manner to stabilize the trunk prior to distal perturbations.[117] This activation, specifically of the transverse abdominis, is thought to help support the spine by increasing intra-abdominal pressure without additional compression at the disc. However, recent data from Morris et al[119] suggested that activation of the transverse abdominis is not a bilateral event to stiffen the spine, it rather reflected a muscle synergy associated with efficient transfer of momentum from ground to hand. Since then, additional research has supported the notion that the feed-forward mechanism might not play the role that it was once thought to play.[120,121]

Other motor control changes are thought to occur through the alterations in the muscle quality. Specifically, multiple studies have investigated the role of fatty infiltrates in the lumbar multifidus as a potential source of nociception. Fatty infiltrates have consistently been found in individuals with cLBP at higher rates than in asymptomatic controls.[122-124] Furthermore, upon biopsy of the multifidus muscle, evidence of muscle degeneration, inflammation, and decreased vascularity was common in those with cLBP compared to normative data.[125] Because the multifidus is thought to assist with providing segmental stability to the vertebra and this promotes stiffness in the spine during movement,[126] it is readily apparent how decreases in the structural integrity of the multifidus might contribute to LBP. Furthermore, this alteration in the quality of the multifidus muscle could potentially explain the decreased proprioception noted in individuals with LBP. People with LBP tend to demonstrate decreased positional sense compared to asymptomatic controls[127] while also having greater repositioning error.[128,129] As the multifidus is thought to play a strong role in the proprioception of the spine,[130] any deficits of the multifidus could help explain poor positional sense and repositioning error. However, it is unclear the extent to which this happens.[131]

In an attempt to promote a unifying theory on how an individual in pain moves differently, Hodges and Tucker[132] penned a review article that outlined their new theory on how an individual with pain adapts. The authors described 5 basic elements (**Table 2**). The first element is that "pain leads to redistribution of activity within and between muscles." They explain how people with pain do not always exhibit a uniform increase or decrease in muscular excitation, but rather a redistribution of activity aimed to protect the painful region. Second, "adaptation to pain changes mechanical behavior." Specific to the low back they indicate how individuals exhibit increases in trunk stiffness and move to a more en bloc movement pattern. Third, "adaptation to pain leads to protection from pain or injury, or threatened pain or injury." This element highlights how the ability to either excite or inhibit muscular activation protects the injured or irritated tissue. The fourth element states that "adaptation to pain involves changes at multiple levels of the motor system." The theory postulates that changes can occur at the spinal level (eg, the spinal cord can be changed by direct input of nociceptive afferents), while cortical changes can result in changes in motor planning and reorganization of somatotopical

Table 2. The 5 Elements of Hodges and Tucker's[132] Theory of Motor Adaptation to Pain

Element 1	Pain leads to redistribution of activity within and between muscles
Element 2	Adaptation to pain changes mechanical behavior
Element 3	Adaptation to pain leads to protection from pain or injury, or threatened pain or injury
Element 4	Adaptation to pain involves changes at multiple levels of the motor system
Element 5	Adaptation to pain has short-term benefit, but with potential long-term consequences