

## REVIEW ARTICLE

## Understanding Low Back Pain and its Biochemistry

Sachin Narwadiya<sup>1</sup>, Tanishka Narwadiya<sup>2</sup>

## HOW TO CITE THIS ARTICLE:

Sachin Narwadiya, Tanishka Narwadiya. Understanding Low Back Pain and its Biochemistry. RFP Jour. of Bio. and Biophy. 2025; 10(1): 21-26.

## ABSTRACT

Low back pain (LBP) is a prevalent musculoskeletal disorder and a leading cause of disability globally, affecting individuals across all age groups. This condition arises from a variety of factors including poor posture, sedentary lifestyle, spinal degeneration, and nutritional deficiencies. Effective prevention and management of LBP require a holistic, multidisciplinary approach. This paper explores the significance of ergonomic practices, dietary strategies, weight management, and regular exercise in reducing the incidence and severity of low back pain. Nutritional components such as calcium, vitamin D, magnesium, and anti-inflammatory foods play a critical role in supporting spinal health. Concurrently, exercise programs focusing on core strengthening and flexibility have been shown to enhance lumbar stability and prevent recurrence. Evidence from clinical and epidemiological studies supports the integration of lifestyle modifications into both preventive and therapeutic frameworks. By correlating these interventions with current research, the discussion emphasizes the need for sustainable, non-pharmacological strategies to mitigate the burden of low back pain and improve long-term outcomes.

## KEYWORDS

- Anti-inflammatory Foods • Low Back Pain (LBP) • Back Pain Epidemiology
- Biomechanical Stress • Calcium and Vitamin D • Chronic Pain Management

## AUTHOR'S AFFILIATION:

<sup>1</sup>Scientist D, Department of Biochemistry & Biotechnology, Institute of Advance Study in Science and Technology, Guwahati, Assam, India.

<sup>2</sup>Student, Department of Biochemistry & Biotechnology, Ambedkar College, Nagpur, Maharashtra, India.

## CORRESPONDING AUTHOR:

Sachin Narwadiya, Scientist D, Department of Biochemistry & Biotechnology, Institute of Advance Study in Science and Technology, Guwahati, Assam, India.

E-mail: snarwadiya@gmail.com

➤ Received: 22-04-2025 ➤ Accepted: 06-06-2025



Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (<http://www.creativecommons.org/licenses/by-nc/4.0/>) which permits non-Commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the Red Flower Publication and Open Access pages (<https://www.rfppl.co.in>)

## INTRODUCTION

Low back pain (LBP) is more than just a nuisance it's a global public health concern that affects people across all age groups and lifestyles. Whether it's a dull ache after a long day or a sharp spasm that restricts movement, low back pain is something most people will experience at some point in their lives. But how common is it, what causes it, and what's happening beneath the surface.

Low back pain (LBP) arises from a complex interplay of mechanical, lifestyle, medical, and psychosocial factors. Mechanical causes are the most prevalent, often involving muscle or ligament strain, disc degeneration, herniation, or spinal abnormalities such as spondylolisthesis and spinal stenosis. Lifestyle related contributors, including prolonged sitting, poor posture, obesity, and smoking, can exacerbate strain on the lumbar spine and hinder spinal health. Medical conditions such as arthritis, osteoporosis, infections like spinal tuberculosis, and tumors can also lead to persistent or severe back pain. Moreover, the psychosocial dimension plays a critical role, with stress, anxiety, depression, and poor sleep often amplifying pain perception and contributing to chronicity. This multifactorial nature of LBP underscores the need for a comprehensive, individualized approach to diagnosis, prevention, and treatment.

Low back pain (LBP) arises from a multifactorial set of influences that go beyond mere physical strain. Musculoskeletal and mechanical issues are primary culprits, including muscle or ligament strain from sudden movements or lifting, herniated or bulging discs that irritate spinal nerves, and degenerative disc disease linked to aging. Other structural problems such as facet joint dysfunction and spondylolisthesis further contribute to spinal instability and discomfort. Lifestyle and postural factors also play a crucial role: poor posture, sedentary behavior, obesity, and improper lifting techniques all impose additional stress on the spine and weaken core support. Underlying medical conditions such as spinal stenosis, osteoarthritis, ankylosing spondylitis, and even kidney disorders can manifest as or aggravate back pain through direct or referred mechanisms. Moreover, psychosocial and emotional

factors like stress, anxiety, depression, and occupational dissatisfaction can amplify pain perception, increase muscle tension, and hinder recovery.

Beyond the visible and structural aspects, LBP is deeply rooted in biochemical changes that govern pain onset and persistence. Inflammatory mediators such as cytokines (IL-1 $\beta$ , IL-6, TNF- $\alpha$ ), prostaglandins (PGE2), and bradykinin are released during injury or disc degeneration, heightening pain sensitivity and promoting immune activity. Simultaneously, oxidative stress driven by the accumulation of reactive oxygen species (ROS) damages cellular structures and disc matrix components, impeding regeneration and perpetuating degeneration. In chronic pain states, neurochemical changes in the nervous system become prominent. Elevated levels of neurotransmitters like substance P, glutamate, and brain derived neurotrophic factor (BDNF) intensify central sensitization, while dysregulation of endogenous opioids reduces the body's natural pain control. Within the disc itself, a biochemical shift occurs: water-retaining proteoglycans like aggrecan diminish, Type II collagen is replaced by stiffer Type I, and matrix metalloproteinases (MMPs) degrade extracellular components, accelerating disc collapse and stiffness.

Emerging research on biochemical biomarkers such as CRP, IL-6, NGF, COMP, IL-8, and MMPs offers new avenues for precise diagnosis and personalized treatment of LBP. These markers not only indicate inflammation and disc degeneration but also guide therapeutic strategies. Treatment implications now extend beyond traditional pain relief. Anti-inflammatory agents like NSAIDs and biologics target cytokines; antioxidants combat ROS; and innovative approaches such as gene therapy and regenerative medicine aim to restore disc integrity. Neuromodulators, meanwhile, can influence neurotransmitter activity to reshape pain perception. Equally important is the mind-body biochemical connection: chronic stress disrupts healing via elevated cortisol, while imbalances in serotonin and dopamine lower pain thresholds and affect mood, reinforcing the chronic pain cycle.

In conclusion, low back pain is not merely a product of strained muscles or degenerative discs it is a dynamic, multilayered condition

involving mechanical dysfunction, lifestyle habits, biochemical cascades, and emotional well-being. A comprehensive understanding of these factors paves the way for more holistic

and personalized approaches to prevention and treatment, ultimately aiming to reduce disability and improve quality of life for millions affected worldwide.

**Table 1:** Significant Blood Parameters in Low Back Pain Evaluation

Blood Test	What It Indicates	Possible Related Conditions
Erythrocyte Sedimentation Rate (ESR)	Inflammation	Infections (e.g., discitis), malignancy, inflammatory spondyloarthropathy
C-Reactive Protein (CRP)	Acute-phase inflammation	Infections, inflammatory arthritis, systemic illness
White Blood Cell Count (WBC)	Immune response or infection	Osteomyelitis, epidural abscess, systemic infection
HLA-B27 antigen	Genetic marker for spondyloarthropathies	Ankylosing spondylitis, reactive arthritis
Alkaline Phosphatase (ALP)	Bone turnover or metastases	Bone metastases, Paget's disease
Calcium / Phosphorus / Vitamin D	Bone metabolism	Osteoporosis, osteomalacia
Rheumatoid Factor (RF) / Anti-CCP / ANA	Autoimmune activity	Rheumatoid arthritis, lupus
Prostate-Specific Antigen (PSA)	Prostate cancer marker	Possible metastatic cause of LBP
Creatinine / BUN	Kidney function	Referred pain from kidney disorders

**Table 2:** Spinal Cord and Nerve Associations with Low Back Pain

Condition	Spinal Cord or Nerve Involvement	Common Symptoms
Lumbar Disc Herniation	Compression of nerve roots (e.g., sciatic nerve)	Radiating leg pain (sciatica), numbness, tingling, weakness
Spinal Stenosis	Narrowing of spinal canal compresses spinal cord or caudaequina	Pain with walking, relief when sitting or bending forward, leg weakness
CaudaEquina Syndrome	Compression of the caudaequina at the lower end of the spinal cord	Saddle anesthesia, bowel/bladder dysfunction, leg paralysis
Spinal Tumors or Metastases	Can compress spinal cord or nerves	Progressive pain, night pain, weakness, sensory loss
Spinal Infections (e.g., abscess, discitis)	Inflammatory compression or invasion of spinal tissues	Severe pain, fever, neurological signs
Spondylolisthesis	Slipping of a vertebra that can pinch spinal nerves	Local back pain, nerve impingement symptoms
Trauma (e.g., fracture, dislocation)	Direct injury to the spinal cord or nerve roots	Immediate pain, possible paralysis or sensory loss
Multiple Sclerosis / Transverse Myelitis	Inflammatory or demyelinating diseases of the spinal cord	Back pain, weakness, sensory changes, bowel/bladder issues

Low back pain (LBP) and the spinal cord are closely related because the spine especially the lumbar region houses and protects the spinal cord and its nerve roots, which are critical to sensation and motor control in the lower body. While most LBP cases are mechanical and involve muscles, discs, or joints, certain types of LBP are directly associated with spinal cord or nerve root involvement, leading to more

serious neurological symptoms.

### When Are Blood Tests Especially Important?

Blood work is typically indicated when LBP is accompanied by:

Fever or chills

Unexplained weight loss

Night pain unrelieved by rest

### Neurological deficits

Age >50 with new-onset pain

History of cancer or immunosuppression

Failure to improve with conservative therapy

### Key Neurological Signs Suggesting Spinal Cord Involvement

Numbness or tingling in the legs or feet

Weakness or paralysis in the lower limbs

Changes in reflexes (hyperreflexia or areflexia)

Loss of bowel or bladder control

Gait disturbance or unsteadiness

Saddle anesthesia (loss of sensation in the buttocks/perineum)

### Diagnostic Tools for Spinal Involvement

**MRI Spine** – Best imaging for cord, disc, and soft tissue pathology

**CT Scan** – Detailed view of bony structures

**Electromyography (EMG)/Nerve conduction studies** – Assess nerve root function

**Neurological examination** – To evaluate sensory, motor, and reflex changes

### Preventive Measures for Low Back Pain

Preventing low back pain involves a combination of maintaining good posture, strengthening core muscles, and adopting healthy lifestyle habits. Regular physical activity, particularly exercises that target the abdominal and back muscles, helps to support the spine and reduce strain. Proper lifting techniques such as bending at the knees and keeping the object close to the body can prevent mechanical stress on the lower back. Ergonomic modifications in the workplace, including supportive chairs and adjustable desks, can minimize the risk of pain related to prolonged sitting. Maintaining a healthy weight reduces the load on the spine, while avoiding smoking improves blood flow to spinal structures and supports healing. Additionally, staying mindful of body mechanics during daily activities, ensuring adequate rest and sleep, and managing stress can further contribute to spinal health. These preventive measures, when practiced consistently, can significantly lower the risk of developing chronic or acute low back pain.

### Diet for Prevention of Low Back Pain

A well-balanced diet plays a vital role in preventing low back pain by supporting bone health, reducing inflammation, and maintaining a healthy body weight. Calcium and vitamin D are essential nutrients for strong bones and vertebrae; foods like dairy products, leafy greens, fortified cereals, eggs, and fatty fish help meet these needs.<sup>13</sup> Magnesium, found in nuts, seeds, whole grains, and legumes, supports muscle and nerve function,<sup>14</sup> while vitamin K (from green vegetables like spinach and broccoli) aids in bone metabolism.<sup>3</sup> Anti-inflammatory foods such as berries, turmeric, ginger, green tea, and omega-3-rich fish like salmon can reduce chronic inflammation that may contribute to back discomfort.<sup>15</sup>

Avoiding processed foods, excessive sugar, and refined carbohydrates is equally important, as these can promote inflammation and weight gain.<sup>16</sup> Hydration is also key; drinking plenty of water helps maintain spinal disc integrity and prevent stiffness.<sup>17</sup> Overall, a nutrient dense, anti-inflammatory diet not only supports spinal health but also helps manage body weight, reducing stress on the lower back.

### DISCUSSION

Low back pain remains one of the leading global causes of disability, significantly impacting quality of life and productivity across populations. According to the **Global Burden of Disease Study (2021)**, low back pain is the top cause of years lived with disability worldwide, particularly in working-age adults.<sup>19</sup> Prevention strategies are increasingly supported by evidence-based research. Studies emphasize that maintaining spinal alignment through ergonomics and posture awareness is critical in both occupational and daily settings.<sup>20</sup> Diet also plays a foundational role in spine health. Research by Holick (2007) and Calder (2006) supports the role of **vitamin D and omega-3 fatty acids** in reducing inflammation and promoting musculoskeletal resilience.<sup>21,22</sup> Weight control through diet helps reduce biomechanical stress on the lumbar spine, as documented in lifestyle intervention studies<sup>23</sup>.

Physical exercise remains a cornerstone of low back pain prevention and management. Clinical trials show that **core stabilization**



**exercises**, aerobic activity, and flexibility training significantly reduce the incidence and recurrence of LBP episodes<sup>24</sup>. A 2016 Cochrane review concluded that exercise is more effective than passive interventions in preventing future back pain and disability.<sup>25</sup> Thus, a multidisciplinary approach integrating diet, posture, weight control, and regular exercise reflects the current scientific consensus on preventing and managing low back pain, supporting a shift from pharmacological to holistic care models for long-term relief and spine health.

## CONCLUSION

Low back pain is a common and often debilitating condition that affects individuals of all ages, stemming from causes that range from poor posture and muscle strain to spinal disorders and systemic diseases. Prevention of low back pain involves a multifaceted approach that includes maintaining proper posture, using ergonomically supportive furniture, and avoiding excessive strain on the spine. A nutrient-rich, anti-inflammatory diet plays a crucial role in maintaining spinal health by supporting bone density through calcium, vitamin D, magnesium, and vitamin K, and reducing inflammation through omega-3 fatty acids and antioxidant-rich foods. Controlling body weight is equally important, as excess weight can increase pressure on the lumbar spine. Regular physical activity, particularly core-strengthening and flexibility exercises such as yoga, swimming, and walking, enhances spinal support and prevents stiffness. Together, these lifestyle modifications not only alleviate existing discomfort but also build resilience against future episodes of low back pain, promoting long-term musculoskeletal health and improved quality of life.

## REFERENCES

1. Apkarian, A.V., Hashmi, J.A., & Baliki, M. N. (2011). Pain and the brain: Specificity and plasticity of the brain in clinical chronic pain. *Pain*, 152 (Suppl), S49-S64. <https://doi.org/10.1016/j.pain.2010.11.010>
2. Balagué, F., Mannion, A.F., Pellisé, F., & Cedraschi, C. (2012). Non-specific low back pain. *The Lancet*, 379(9814), 482-491. [https://doi.org/10.1016/S0140-6736\(11\)60610-7](https://doi.org/10.1016/S0140-6736(11)60610-7)
3. Deyo, R.A., Mirza, S.K., Turner, J.A., & Martin, B.I. (2009). Overtreating chronic back pain: Time to back off? *Journal of the American Board of Family Medicine*, 22(1), 62-68. <https://doi.org/10.3122/jabfm.2009.01.080102>
4. Hoy, D., Bain, C., Williams, G., March, L., Brooks, P., Blyth, F., ...& Buchbinder, R. (2012). A systematic review of the global prevalence of low back pain. *Arthritis & Rheumatism*, 64(6), 2028-2037. <https://doi.org/10.1002/art.34347>
5. Kang, J.D., Georgescu, H.I., McIntyre-Larkin, L., Stefanovic-Racic, M., & Evans, C.H. (1996). Increased expression of matrix metalloproteinase-3 (stromelysin) in herniated human intervertebral disc tissue. *Journal of Spinal Disorders*, 9(4), 269-275. <https://doi.org/10.1097/00002517-199608000-00002>
6. Krock, E., Rosenzweig, D.H., & Haglund, L. (2015). The inflammatory milieu of the degenerating intervertebral disc. *Frontiers in Immunology*, 6, 120. <https://doi.org/10.3389/fimmu.2015.00120>
7. Pincus, T., Burton, A.K., Vogel, S., & Field, A. P. (2002). A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine*, 27(5), E109-E120. <https://doi.org/10.1097/00007632-200203010-00017>
8. Risbud, M.V., & Shapiro, I.M. (2014). Role of cytokines in intervertebral disc degeneration: Pain and disc content. *Nature Reviews Rheumatology*, 10(1), 44-56. <https://doi.org/10.1038/nrrheum.2013.160>
9. Vos, T., Lim, S.S., Abbafati, C., Abbas, K. M., Abbasi, M., Abbasifard, M., ...& Murray, C. J.L. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: A systematic analysis. *The Lancet*, 396(10258), 1204-1222. [https://doi.org/10.1016/S0140-6736\(20\)30925-9](https://doi.org/10.1016/S0140-6736(20)30925-9)
10. Wang, K., Wu, D., Zhang, H., Li, W., & Zhang, X. (2016). Cartilage oligomeric matrix protein as a biomarker of intervertebral disc degeneration. *Osteoarthritis and Cartilage*, 24(3), 598-603. <https://doi.org/10.1016/j.joca.2015.10.009>
11. World Health Organization. (n.d.). *Musculoskeletal conditions*. <https://www.who.int/news-room/fact-sheets/detail/musculoskeletal-conditions>
12. Yoshizawa, H., Deie, M., & Yamamuro, T. (1998). Experimental disc degeneration induced by a nitric oxide donor in rabbits: Correlation between nitric oxide and matrix degradation. *Journal of Orthopaedic Research*, 16(3), 289-297. <https://doi.org/10.1002/jor.1100160303>

13. Holick, M.F. (2007). Vitamin D deficiency. *New England Journal of Medicine*, 357(3), 266–281. <https://doi.org/10.1056/NEJMr070553>
14. deBaaij, J.H.F., Hoenderop, J.G.J., Bindels, R.J.M. (2015). Magnesium in man: Implications for health and disease. *Physiological Reviews*, 95(1), 1–46. <https://doi.org/10.1152/physrev.00012.2014>
15. Cockayne, S., Adamson, J., Lanham-New, S., *et al.* (2006). Vitamin K and the prevention of fractures. *Archives of Internal Medicine*, 166(12), 1256–1261. <https://doi.org/10.1001/archinte.166.12.1256>
16. Calder, P.C. (2006). n-3 Polyunsaturated fatty acids, inflammation, and inflammatory diseases. *The American Journal of Clinical Nutrition*, 83(6), S1505–S1519. <https://doi.org/10.1093/ajcn/83.6.1505S>
17. Cordain, L., Eaton, S.B., Sebastian, A., *et al.* (2005). Origins and evolution of the Western diet: health implications for the 21st century. *The American Journal of Clinical Nutrition*, 81(2), 341–354. <https://doi.org/10.1093/ajcn.81.2.341>
18. McGill, S.M. (2007). *Low Back Disorders: Evidence-Based Prevention and Rehabilitation*. Human Kinetics.
19. Wu, A., March, L., Zheng, X., *et al.* (2021). Global low back pain prevalence and burden: Estimates from the Global Burden of Disease Study 2019. *Lancet Rheumatology*, 3(6), e416–e429. [https://doi.org/10.1016/S2665-9913\(21\)00059-4](https://doi.org/10.1016/S2665-9913(21)00059-4)
20. vanTulder, M., *et al.* (2006). Multidisciplinary biopsychosocial rehabilitation for chronic low back pain. *Cochrane Database Syst Rev*, (1), CD000963.
21. Holick, M.F. (2007). Vitamin D deficiency. *New England Journal of Medicine*, 357(3), 266–281. <https://doi.org/10.1056/NEJMr070553>
22. Calder, P.C. (2006). n-3 Polyunsaturated fatty acids, inflammation, and inflammatory diseases. *The American Journal of Clinical Nutrition*, 83(6), S1505–S1519.
23. Shiri, R., *et al.* (2010). The association between obesity and low back pain: a meta-analysis. *American Journal of Epidemiology*, 171(2), 135–154.
24. Panjabi, M.M. (1992). The stabilizing system of the spine: Part I. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders*, 5(4), 383–389.
25. Hayden, J.A., Ellis, J., Ogilvie, R., Malmivaara, A., van Tulder, M.W. (2021). **Exercise therapy for chronic low back pain**. *Cochrane Database Syst Rev*, 9, CD009790.