International Journal of Multidisciplinary Research and Publications
ISSN (Online): 2581-6187

The Effect of Occupation on the Relationship of Age and Lumbar Herniated Discs: A Literature Review

Moch Rifqi Ilham[1], Muhammad Faris[2]
[1]Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[2]Department of Neurosurgery, University Airlangga, Surabaya, Indonesia
Email address: rifqi-2022@fk.unair.ac.id; mfarisns@fk.unair.ac.id

Abstract *— Lumbar disc herniation (LDH) is one of the leading causes of low back pain, and disability worldwide. Although increasing age remains a major non-modifiable risk factor, accumulating evidence suggests that occupational mechanical loading may accelerate disc degeneration and contribute to earlier disease onset. This review aims to summarise current evidence regarding the influence of occupation on lumbar disc herniation and age, with emphasis on biomechanical mechanism and occupational exposures. Lumbar disc herniation results from a complex interaction between multiple factors including biological aging and cumulative occupational mechanical stress. Future research should adopt standardised occupational classifications and objective workload assessments to better characterise this relationship.*

Keywords *— Occupation, Age, Lumbar Disc Herniation*

I. Introduction

Low back pain is one of the leading causes of disability worldwide, with lumbar disc herniation representing one of the most common causes of lumbar radiculopathy and functional impairment [1]. Lumbar disc herniation occurs when the nucleus pulposus protrudes through a weakened or ruptured annulus fibrosus, resulting in nerve root compression and symptoms including low back pain, sciatica, sensory deficits, and motor weakness. Although lumbar disc herniation has traditionally been regarded as a degenerative disease affecting adults between 30 and 50 years of age, recent epidemiological studies have reported an increasing incidence among younger populations, suggesting that environmental and occupational factors contribute significantly to disease onset [2]. Age remains one of the strongest non-modifiable risk factors for lumbar disc herniation because progressive degeneration decreases disc hydration, proteoglycan content, and biomechanical stability, making the intervertebral disc more susceptible to injury. However, recent mechanobiological and biomechanical studies have demonstrated that repetitive mechanical loading, excessive spinal compression, torsional stress, and cumulative microtrauma can accelerate disc degeneration independently of chronological age [3]. Finite element models further support these findings by showing that repetitive loading substantially increases annular stress and the likelihood of disc herniation [4]. Occupation is considered one of the most important modifiable risk factors affecting lumbar disc health. Occupations involving repetitive heavy lifting, manual material handling, prolonged bending, awkward postures, prolonged sitting, and whole-body vibration expose workers to excessive spinal loading, increasing the risk of early disc degeneration and lumbar disc herniation [1]. Conversely, sedentary occupations may also contribute to lumbar degeneration through prolonged static postures and reduced trunk muscle endurance [5]. These findings suggest that occupational exposure may influence not only the occurrence of lumbar disc herniation but also the age at which the condition develops. Despite extensive research on aging and occupational risk factors independently, limited studies have comprehensively examined how occupation modifies the relationship between age and lumbar disc herniation. Therefore, this literature review aims to summarize current evidence regarding the influence of occupation on age-related lumbar disc herniation, with particular emphasis on biomechanical mechanisms and occupational exposures.

II. Biological Mechanism of Lumbar Disc Herniation

The intervertebral disc consists of three major components: the nucleus pulposus (NP), annulus fibrosus (AF), and cartilaginous endplates. Under normal physiological conditions, these structures maintain spinal flexibility while distributing compressive loads evenly across the vertebral column. The NP is rich in proteoglycans and water, allowing it to resist compressive forces, whereas the AF is composed of concentric collagen fibers that provide tensile strength and contain the NP within the disc [3]. The biological process leading to LDH begins with intervertebral disc degeneration. Aging and repetitive mechanical stress reduce the synthesis of extracellular matrix components, particularly aggrecan and type II collagen, while increasing the activity of matrix-degrading enzymes such as matrix metalloproteinases (MMPs) and a disintegrin and metalloproteinase with thrombospondin motifs (ADAMTS). These catabolic changes decrease water content within the nucleus pulposus, reduce disc height, and weaken the annulus fibrosus, making it more susceptible to fissures and rupture [3]. Mechanical loading further accelerates degeneration by inducing cellular stress and disrupting disc homeostasis. Excessive spinal compression, repetitive flexion, torsional loading, and cumulative microtrauma stimulate nucleus pulposus and annulus fibrosus cells to release inflammatory mediators, including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), interleukin-6 (IL-6), and prostaglandin E2. These cytokines promote extracellular matrix degradation, inhibit matrix synthesis, and induce apoptosis and cellular senescence, thereby accelerating structural deterioration of the intervertebral disc [4]. As

Moch Rifqi Ilham and Muhammad Faris, "The Effect of Occupation on the Relationship of Age and Lumbar Herniated Discs: A Literature Review," *International Journal of Multidisciplinary Research and Publications (IJMRAP)*, Volume 9, Issue 3, pp. 35-37, August 2026.

degeneration progresses, annular fissures allow nucleus pulposus material to migrate outward. Herniated disc tissue not only compresses adjacent nerve roots mechanically but also initiates a biochemical inflammatory response. Exposure of the nucleus pulposus to the epidural space activates macrophages and other immune cells, leading to the release of additional pro-inflammatory cytokines and chemokines. This inflammatory cascade sensitizes nociceptors, increases vascular permeability, and produces nerve root edema, contributing significantly to radicular pain and neurological symptoms [1].

Recent mechanobiological studies have demonstrated that biological degeneration and biomechanical loading are closely interconnected. Abnormal mechanical stress alters intracellular signaling pathways, including nuclear factor-kappa B (NF-κB) and mitogen-activated protein kinase (MAPK), which regulate inflammatory responses and matrix metabolism. Persistent activation of these pathways promotes chronic inflammation and progressive disc degeneration while reducing the regenerative capacity of disc cells. These findings have led to increasing interest in regenerative therapies, including stem cell transplantation, growth factor delivery, and tissue-engineering approaches aimed at restoring extracellular matrix integrity and slowing disease progression [3]. Overall, lumbar disc herniation results from a complex interaction between degenerative biological processes, inflammatory responses, and mechanical stress.

III. Occupational Exposure

Occupational exposure occurs through actions such as heavy lifting, frequent bending, twisting, vibration, prolonged sitting and repetitive movements. Manual labor occupations, including construction workers, warehouse personnel, and agricultural and factory workers, often involve repetitive biomechanical loading of the lumbar spine which stress may accelerate intervertebral disc degeneration, increasing the likelihood of herniation and potentially contributing to an earlier clinical presentation. Heavy lifting and extreme lumbar flexion or extension are particularly important risk factors, while the combination of lifting with trunk rotation may impose greater mechanical stress on the intervertebral discs [7]. Cumulative occupational lumbar load has also demonstrated a dose–response relationship with lumbar disc herniation. However, prolonged sitting and repetitive movements should be interpreted as potential contributing exposures rather than definitive independent causes, because evidence for these factors alone remains mixed. A German multi centre case-control study similarly identified a positive dose–response relationship between cumulative occupational lumbar load and LDH. Among men in the highest cumulative-load category, the adjusted odds ratio was 3.43.43.4 (95% CI 2.2–5.0), compared with the lowest exposure category; among women, the highest category had an adjusted OR of 2.32.32.3 (95% CI 1.5–3.6) [6]. The role of forward bending and load handling was also supported by an earlier case-control study, which reported a statistically significant association between extreme forward bending and symptomatic LDH, as well as between cumulative weight lifting/carrying and LDH accompanied by osteochondrosis or spondylosis. A systematic review of occupational lumbar disorders further describes lifting, carrying, bent-trunk work, and lifting while twisting as important sources of spinal loading [6].

IV. Gaps and Future Directions

Despite the available literature of the relationship between occupational factors to lumber disc herniation and age, there is no standardised system for classifying occupational exposure across studies, with occupations often categorised broadly or described inconsistently, limiting meaningful comparisons and analysis. Furthermore, occupational exposure is frequently assessed using self-reported job titles without detailed measure of cumulative workload, including the intensity, frequency, and duration of activities, especially activities such as repetitive movements, prolonged sitting or standing, and awkward postures. These self assessments may not accurately reflect the true mechanical demands placed on the lumbar spine throughout an individual's working life. Additionally, although age and occupation are a well recognised risk factor for lumbar disc herniation, relatively few studies have explored their interaction, measuring these factors independently instead of evaluating whether specific occupational exposures accelerate disc degeneration or result in an earlier age of onset. Addressing these methodological limitations through standardised occupational classification systems, objective workload measurements and multicenter longitudinal studies would provide a more comprehensive understanding. Other than age of onset, occupation may also affect treatment due to socioeconomic burden and choice, through the cost of certain treatments, and the time required to return back to work, which can be an alternative perspective to be further investigated. A better understanding of these interactions will support more effective workplace disease prevention strategies, and individualised management of lumbar disc herniation.

V. Conclusion

Lumbar disc herniation is a multifactorial disorder resulting from the interaction between biological aging, intervertebral disc degeneration, inflammation and cumulative mechanical stress, while advancing age remains an important non modifiable risk factor, growing evidence indicates that occupational exposures, including heavy lifting, repetitive bending, prolonged sitting, awkward postures, and whole-body vibration, can accelerate degenerative changes within the intervertebral disc through biological mechanism. Despite increasing recognition of occupational risk factors, current evidence remains limited by heterogenous occupational classifications and inconsistent and unavailable measurement of cumulative mechanical workload.

Acknowledgment

The authors would like to extend their gratitude to Universitas Airlangga for the resources that supported the completion of this literature review, and to all individuals who has helped the successful completion of this work.

References

[1] M. Pojskic, E. Bisson, J. Oertel, T. Takami, C. Zygourakis, and F. Costa,

"Lumbar disc herniation: Epidemiology, clinical and radiologic diagnosis WFNS spine committee recommendations," *World Neurosurgery X*, vol. 22, p. 100279, 2024

[2] C. A. Hincapié, D. Kroismayr, L. Hofstetter, A. Kurmann, C. Cancelliere, Y. Raja Rampersaud, E. Boyle, G. A. Tomlinson, A. R. Jadad, J. Hartvigsen, P. Côté, and J. D. Cassidy, "Incidence of and risk factors for lumbar disc herniation with radiculopathy in adults: A systematic review," *European Spine Journal*, vol. 34, no. 1, pp. 263–294, 2024

[3] G. Vadalà, F. Russo, I.-H. Han, A. Jain, and J. Tavakoli, "The Biomechanical Landscape of Lumbar Disc Herniation: Mechanobiological Insights into Injury and Regeneration," *Neurospine*, vol. 23, pp. 159–175, 2026.

[4] S. Rossman, E. Meyer, and S. Rundell, "Development of a finite element lumbar spine model to predict intervertebral disc herniation risk factors," *Comput. Methods Biomech. Biomed. Eng.*, vol. 25, no. 1, pp. 1–13, 2022.

[5] A. Alcántara Guardado, A. Baker, A. Weightman, J. A. Hoyland, and G. Cooper, "Lumbar Intervertebral Disc Herniation: Annular Closure Devices and Key Design Requirements," *Bioengineering*, vol. 9, no. 2, Art. no. 47, 2022.

[6] A. Seidler, U. Bolm-Audorff, T. Siol, N. Henkel, C. Fuchs, H. Schug, C. Leheta, H. Marziniak, M. Schmitt, and G. Elsner, "Occupational risk factors for symptomatic lumbar disc herniation: A case-control study," Occup. Environ. Med., vol. 60, no. 11, pp. 821–830, Nov. 2003, doi: 10.1136/oem.60.11.821.

[7] I. A. Díaz and M. V. R. Mallorquí, "La resolución del concurso en las empresas canarias," *Journal title appears to be incorrect*, vol. 17, no. 41, pp. 85–110, 2014. doi: 10.3390/ijerph17134832.

[8] M. Zhou, A. A. Theologis, and G. D. O'Connell, "Understanding the etiopathogenesis of lumbar intervertebral disc herniation: From clinical evidence to basic scientific research", *JOR SPINE*, vol. 7, no. 1, Oct. 2023.

[9] T. D. Slater, H.-J. Wilke, G. Gurusamy, S. Rajasekaran, and N. Newell, "Lumbar disc herniation modelling: A review of ex-vivo mechanical models and a comparison with clinical data", *European Spine Journal*, vol. 34, no. 10, Jun. 2025.

[10] N. Withanage, S. Perera, H. Peiris, S. Prathapan, and L. Athiththan, "Regression model for socio-demographic, behavioural and occupational risk factors in lumbar disc herniation (ldh) and lumbar disc degeneration (ldhd)-in comparison to apparently healthy subjects.", *International Journal of Human and Health Sciences (IJHHS)*, vol. 5, no. 4, Jul. 2021.