# Central Obesity as a Risk Factor for Low Back Pain

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**Abstract**

Central obesity, or abdominal obesity, is a condition marked by excessive fat accumulation around internal organs, leading to a variety of health complications such as cardiovascular disease, type 2 diabetes, and musculoskeletal disorders, including low back pain (LBP). Central obesity is defined by increased waist circumference, with values greater than 80 cm in women and 90 cm in men. The condition is primarily influenced by factors such as physical inactivity, poor eating habits, and aging. The relationship between central obesity and LBP is supported by studies indicating that abdominal fat increases mechanical load on the lumbar spine, contributing to disc degeneration and pain. Additionally, adipose tissue secretes pro-inflammatory cytokines that exacerbate inflammation and pain sensitization in the spine, leading to LBP. Obesity-related complications, such as muscle weakness and sleep disturbances, can further reduce quality of life. Preventive measures for LBP include physical exercise, weight reduction, and posture correction. Effective treatment options range from pharmacological therapies to physical rehabilitation and alternative medicine approaches. Understanding the link between central obesity and LBP is critical in developing preventive strategies and improving the management of this common condition.

**Keywords:** Central obesity, Low Back Pain, waist circumference

# Obesitas Sentral Sebagai Faktor Risiko Pada *Low Back Pain*

**Abstrak**

Obesitas sentral, atau obesitas abdominal, adalah kondisi yang ditandai dengan akumulasi lemak berlebih di sekitar organ dalam yang dapat menyebabkan berbagai komplikasi kesehatan seperti penyakit kardiovaskular, diabetes tipe 2, dan gangguan muskuloskeletal, termasuk *Low Back Pain* (LBP). Obesitas sentral didefinisikan berdasarkan peningkatan ukuran lingkar perut, dengan batas lebih dari 80 cm pada wanita dan 90 cm pada pria. Kondisi ini terutama dipengaruhi oleh berbagai faktor seperti kurangnya aktivitas fisik, pola makan yang buruk, dan penuaan. Hubungan antara obesitas sentral dan LBP didukung oleh penelitian yang menunjukkan bahwa lemak abdominal meningkatkan beban mekanis pada tulang belakang lumbal, yang berkontribusi terhadap degenerasi diskus dan nyeri pada punggung bawah. Selain itu, jaringan adiposa menghasilkan sitokin proinflamasi yang memperburuk peradangan dan meningkatkan sensitivitas nyeri pada tulang belakang, sehingga menyebabkan LBP. Komplikasi akibat obesitas, seperti kelemahan otot dan gangguan tidur, juga dapat menurunkan kualitas hidup. Pencegahan LBP pada individu dengan obesitas sentral dapat dilakukan melalui aktivitas fisik, penurunan berat badan, serta perbaikan postur tubuh. Sementara itu, pilihan pengobatan meliputi terapi farmakologis, rehabilitasi fisik, dan metode alternatif. Hubungan antara obesitas sentral dan LBP sangat penting untuk dipahami dalam upaya pencegahan dan penanganan kondisi ini secara lebih efektif.

**Kata kunci:** *Low Back Pain*, lingkar perut, obesitas sentral

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## Introduction

Central obesity, also known as abdominal obesity, is a condition characterized by excessive fat accumulation in the abdominal area.[1] This accumulation includes subcutaneous fat, located beneath the skin, and visceral fat, which surrounds internal organs.[2] Central obesity has become a major global health issue, reaching pandemic levels worldwide.[3] The global prevalence of central obesity has significantly increased, now standing at 41.5%.[4] Similarly, the 2023 Indonesian Health Survey (SKI) reported a prevalence rate of 36.8% in Indonesia.[5] Central obesity is widely acknowledged as a significant risk factor for degenerative diseases and various musculoskeletal disorders.[3] Beyond its well-established role in cardiovascular disease, hypertension, type 2 diabetes, hip and knee osteoarthritis, and certain cancers, central

obesity also impacts the musculoskeletal system, contributing to conditions such as low back pain (LBP).[6]

Low back pain is one of the most serious and common musculoskeletal disorders globally, affecting all age groups and ranking as the leading cause of disability, significantly impacting workplace productivity and occupational health.[7] According to the World Health Organization (WHO), the global prevalence of LBP reached 1.71 billion cases in 2022,[8] with a projected increase of 36.4% by 2050.[9] In Indonesia, the prevalence of LBP affects 34.4 million people.[10] This condition is particularly common among drivers and is frequently reported as a major occupational health issue in this population.[11] LBP has multiple risk factors which include abdominal obesity. Notably, central obesity has been found to increase the risk of LBP by 30%, with stronger correlations observed among the female population.[12]

Central obesity is a multifactorial disease with complex, poorly understood correlations and is increasingly recognized as a potential risk factor for LBP.[13] Two primary mechanisms explain the link between obesity and LBP. First, excess abdominal fat directly increases the mechanical load on the lumbar spine, causing reduced disc hydration, decreased disc height, disc herniation, or hypertrophy of the lumbar spine ligaments, which leads to disc degeneration. Second, adipose tissue is metabolically active and secretes pro-inflammatory cytokines that can trigger cell phenotype changes, matrix degeneration, and chemokine production.[14]

While central obesity is widely considered a risk factor for LBP, the mechanisms underlying this relationship remain unclear.[15] Some studies suggest that central obesity may not be a strong predictor of LBP risk. The existing literature reveals conflicting findings, emphasizing the need for further research to clarify the relationship between central obesity and LBP.[16] This study aims to explore the potential role of central obesity as a risk factor for low back pain.

**Content**

**Central Obesity**

Fat accumulation in the adipose tissue serves as a storage for body fat, which helps to protect the internal organs in the abdomen and chest. However, excessive body fat can lead to obesity.[17] Central obesity, or abdominal obesity, is an excessive accumulation of fat in the abdominal cavity surrounding the internal organs. It is considered as a risk factor for several diseases, including type 2 diabetes mellitus, gallstone, hypertension, dyslipidemia, atherosclerosis, cardiovascular disease, pulmonary disease, and muskulosceletal disease.[18] Central obesity can be measured using indicators such as waist circumference or abdominal circumferenc, with values greater than 80 cm in women and 90 cm in men indicating central obesity. Waist circumference has been used as a measure of central obesity in various studies and has been associated with an increased likelihood of developing low back pain.[12]

Central obesity is influenced by several factors, including low physical activity, emotional stress, inadequate fruit consumption, poor eating habits, and frequent intake of unhealthy foods.[18] As individuals age, their body weight tends to increase due to the accumulation of fat in muscles, while the number and volume of muscle cells decrease. This process often leads to reduced physical activity, which further exacerbates the risk of central obesity.[19] In addition to these factors, obesity can also be caused by excessive consumption of high-calorie foods, which may be driven by food addiction mechanisms or a combination of brain circuit dysfunctions and neuroendocrine hormone imbalances. These factors contribute to pathological overeating, physical inactivity, and other behaviors that further promote obesity.[20]

**Central Obesity as Risk Factor of Low Back Pain**

Low back pain (LBP) is the pain localized to the region between the lower margins of the 12$^{th}$ rib and the gluteal folds. It can be classified into acute (lasting less than 6 weeks), subacute (lasting between 6 weeks and 3 months), or chronic (lasting more than 3 months).[11] The diagnosis of LBP is established through a

combination of anamnesis, physical examination, and supportive tests. During anamnesis, several key characteristics of LBP should be explored, including the duration (acute, subacute, or chronic), the location and radiation of the pain (axial or radicular), its severity (measured using the Visual Analog Scale or Numeric Rating Scale), the nature of the pain (burning, aching, sharp, tingling, or electric-like), aggravating and relieving factors, social history, previous pain episodes, red flags, and psychological factors.[21] Physical examination involves inspection, palpation, range of motion assessment, strength tests, provocative maneuvers, and neurological evaluation. Supportive tests are indicated for patients with suspected underlying conditions or red flags related to LBP.[22] LBP may originate from various structures, including the spine, muscles, nerves, and surrounding tissues. Contributing factors to the development of LBP can be individual, occupational, and environmental in nature.[23]

Low back pain (LBP) is a multifactorial condition, with genetic, biophysical, psychological, and social factors playing significant roles in its development. In addition, lifestyle factors such as physical inactivity, obesity, and smoking are strongly associated with the incidence of LBP.[24] Approximately 65% of individuals with LBP are overweight or obese, with other lifestyle factors such as inadequate sleep, stress, and smoking also contributing to its onset.[25] Among adults, a sedentary lifestyle is a notable risk factor for LBP, with prolonged sitting and driving times identified as significant contributors. Sedentary behavior has been particularly linked to LBP in office workers. Moreover, excess weight and smoking are also associated with an increased risk of LBP.[26] Studies have shown that central obesity, as defined by waist circumference, is positively correlated with LBP.[27] Obesity is considered a risk factor for degenerative changes in the spine, such as herniated discs, which can lead to LBP.[28] Additionally, individuals with LBP are often found to have metabolic syndrome, which is characterized by abdominal obesity.[29]

**Mechanism Linking Central Obesity and Low Back Pain**

Obesity is associated with both the hyperplasia and hypertrophy of fat tissue, which contributes to the development of low back pain.[28] The relationship between central adiposity and low back pain has been well-documented, with the strongest evidence pointing to the significant association between abdominal obesity and low back pain.[2] Studies have shown that central obesity increases lordosis and doubles the risk of low back pain, especially among drivers.[11] An increase in body weight and obesity is a major risk factor for the development of back pain, as it increases the strain on the weight-bearing spinal elements. Central obesity, characterized by the deposition of fat around the abdominal circumference, leads to an increased lordotic curve and increased axial loading on the lumbar vertebrae, which can cause low back pain.[30]

Research has also demonstrated that the accumulation of fat in the abdominal area places additional load on the spine, particularly in the lower back region. This, in turn, increases the pressure on the intervertebral discs, leading to pain.[31] Obesity is linked to low back pain due to the increased weight pressing down on the intervertebral discs.[32] Furthermore, obese individuals may experience greater mechanical load on their joints due to weaker lumbar muscle strength and reduced lumbar motion, which increases their risk of developing low back pain. People with abdominal obesity accumulate fat in the waist area, which weakens the strength of their waist muscles and significantly reduces the range of motion in their lumbar spine, thus increasing the risk of chronic low back pain.[33]

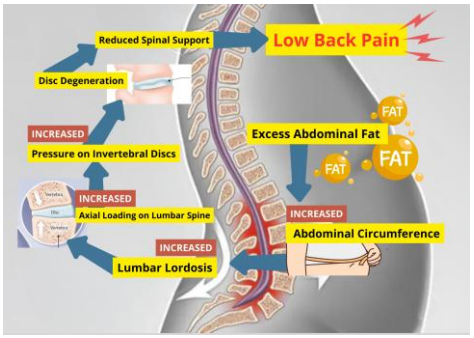


**Figure 1.** Mechanisms Linking Central Obesity to Low Back Pain: Biomechanical Pathways

Figure 1 illustrates the relationship between central obesity and low back pain, highlighting the biomechanical pathways involved. Excess abdominal fat leads to an increased abdominal circumference, which contributes to lumbar lordosis due to the augmented axial loading on the lumbar spine. This additional load increases the pressure on intervertebral discs, accelerating disc degeneration and reducing spinal support. These combined factors ultimately result in low back pain, emphasizing the significant impact of abdominal obesity on spinal health and lumbar biomechanics.

Adiposity plays a crucial role in musculoskeletal pain, not only through mechanical load but also via its role as an endocrine organ. Adipose tissue releases pro-inflammatory cytokines and adipokines that can heighten inflammatory changes, leading to tissue destruction and increased pain and disability.[2] In individuals with abdominal obesity, levels of pro-inflammatory adipocytokines such as tumor necrosis factor-α, interleukin 1β, and leptin are significantly elevated. Some of these inflammatory cytokines are involved in the initiation and persistence of pain by directly activating nociceptive neurons. Additionally, adipose tissue plays a role in inflammation, which lowers the threshold of nerve excitation and enhances the nociceptive response to stimuli. This leads to both peripheral and central pain sensitization. Studies have found that individuals with higher abdominal obesity had higher Visual Analog Scale (VAS) scores, elevated levels of interleukin 6, and tumor necrosis factor-α, indicating a significant inflammatory component in the experience of low back pain.[32] Adipokines, such as leptin and resistin, contribute to the inflammatory reaction in the joints and promote tissue degradation, including in conditions like LBP, where they stimulate the production of damaging mediators like MMPs, further exacerbating pain and disability. Additionally, the role of adiponectin in promoting bone erosion and progression of joint damage aligns with the inflammatory processes seen in individuals with central obesity, intensifying the experience of LBP.[34]

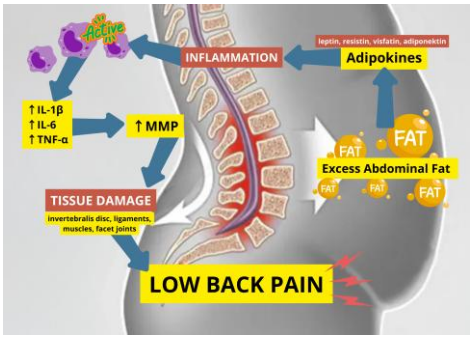


**Figure 2.** Mechanisms Linking Central Obesity to Low Back Pain: Inflammatory Pathways

Figure 2 illustrates the biochemical mechanisms linking central obesity to low back pain through inflammatory pathways. Adipose tissue, particularly in individuals with abdominal obesity, secretes adipokines such as leptin, resistin, and visfatin, along with pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6. These mediators drive systemic inflammation, increasing matrix metalloproteinase (MMP) activity, which degrades intervertebral discs, ligaments, muscles, and facet joints. This inflammatory cascade leads to tissue damage, lowers the pain threshold, and amplifies nociceptive responses, ultimately contributing to low back pain. The diagram emphasizes the interplay of mechanical and biochemical factors in the development of low back pain associated with central obesity.

Two primary mechanisms explain the link between obesity and low back pain. First, excess abdominal fat directly increases the mechanical load on the lumbar spine, which leads to reduced disc hydration, decreased disc height, disc herniation, or hypertrophy of the lumbar spine ligaments, ultimately contributing to disc degeneration. Second, adipose tissue is metabolically active and secretes pro-inflammatory cytokines that can trigger cell phenotype changes, matrix degeneration, and chemokine production, further exacerbating the development of low back pain.[12]

**Complication**

Complications arising from untreated or poorly managed low back pain (LBP) can be diverse. One common complication is muscle weakness or atrophy, which significantly impacts an individual's ability to perform daily

activities. Additionally, disturbances in kidney or urinary tract function may occur, particularly due to issues with organs around the lower back, such as the urinary system or associated nerves. Prolonged LBP can also lead to sleep disturbances, further exacerbating both physical and mental health. This, in turn, negatively affects quality of life by reducing mobility and impairing social interactions. In some cases, individuals may develop a dependence on pain-relieving medications, increasing the risk of long-term side effects. Therefore, appropriate management and effective treatment are critical to prevent such complications.[35]

**Preventive Measures**

Preventing low back pain (LBP) requires sufficient physical exercise and knowledge about the condition.[36] There is low to moderate evidence suggesting that exercise therapy is more effective than conservative treatment in reducing pain severity in LBP.[37] Aerobic exercises, Pilates, core strengthening, stabilization exercises, and flexibility exercises have all been found to be effective in reducing pain, with no significant differences observed between these types of training.[12] Furthermore, reducing sedentary behavior and encouraging moderate levels of physical activity are generally believed to be beneficial for individuals with low back pain.[24] Core stabilization exercises (CSE) have been used as both a treatment and preventive option for LBP, as they help maintain neutral spinal posture and support coordinated muscle contractions.[38] Given that LBP is multifactorial, it is unlikely that a single type of exercise would be the best treatment. However, a combination of aerobic, Pilates, core strengthening, stabilization, and stretching exercises has been shown to significantly reduce pain severity in LBP patients.[39]

In addition to exercise, other treatment approaches are also relevant. Pharmacological therapies, physical and rehabilitation treatments, psychiatric treatments, complementary and alternative medicine, and minimally invasive percutaneous approaches are important courses of care. Spinal manipulation, for example, is widely used to relieve pressure on the joints, reduce inflammation, and improve nerve function.[40] Manual acupuncture, a key treatment in Traditional Chinese Medicine, involves the insertion of needles into the skin without injections and is commonly used for pain relief and various disorders. For individuals who are overweight or obese, weight reduction is often prescribed as a therapeutic strategy to alleviate pain and disability. Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used as first-line analgesics in LBP treatment. Poor posture and prolonged sitting are well-known risk factors for LBP. When sitting, the average individual's thighs and torso form a nearly 90-degree angle, causing the pelvis to tilt backward. In contrast, standing causes the pelvis to tilt forward. Therefore, posture correction is essential in minimizing the risk of developing LBP.[41]

**Summary**

Central obesity, characterized by excessive fat accumulation in the abdominal area, is a growing global health concern linked to various diseases, including musculoskeletal disorders such as low back pain (LBP). The prevalence of central obesity has significantly increased worldwide, with substantial rates reported in Indonesia. LBP is a leading cause of disability and has been associated with central obesity through both biomechanical and inflammatory mechanisms. Excess abdominal fat increases spinal loading, accelerates disc degeneration, and contributes to systemic inflammation through pro-inflammatory cytokines, exacerbating pain and disability.

The relationship between central obesity and LBP is influenced by multiple factors, including sedentary lifestyles, poor dietary habits, and metabolic disorders. Studies have shown a positive correlation between waist circumference and LBP, emphasizing the impact of obesity on spinal health. If left untreated, LBP can lead to complications such as reduced mobility, chronic pain, and dependence on pain medications, affecting overall quality of life.

Preventive strategies include regular physical activity, weight management, and posture

correction, along with therapeutic approaches such as physiotherapy, pharmacological treatments, and alternative medicine. Given the complex interplay between central obesity and LBP, further research is essential to better understand the underlying mechanisms and develop targeted interventions to mitigate their impact.

**Conclusion**

Central obesity plays a significant role in the development of low back pain (LBP) through both mechanical and metabolic mechanisms. The excessive accumulation of abdominal fat places additional strain on the lumbar spine, increasing the risk of disc degeneration and LBP. Additionally, pro-inflammatory cytokines released from adipose tissue contribute to increased pain sensitivity and inflammation in the spine, further exacerbating LBP symptoms. Given the rising prevalence of central obesity and its association with LBP, addressing obesity through preventive strategies, including physical activity, weight management, and posture correction, is crucial in reducing the burden of LBP on individuals and the healthcare system. Future research should aim to clarify the complex relationship between central obesity and LBP, with a focus on better understanding the underlying mechanisms and identifying effective interventions.

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