

Biomechanical Significance Of Mechanical Neck Pain Associated With Obesity: A Narrative Review

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ABSTRACT

Mechanical neck pain (MNP) is a musculoskeletal disorder commonly exacerbated by obesity. Obesity causes postural changes, such as forward head posture (FHP), which elevates mechanical loading to the cervical spine, accentuating neck pain. This narrative review discusses the biomechanical significance of MNP in the context of obesity. It synthesises existing literature on postural change, muscular dysfunction, and increased spinal burden with systemic inflammation associated with obesity to understand their role in the pathophysiology of MNP. Obesity contributes to postural changes like FHP, elevating mechanical loading on the cervical spine. Central obesity, with visceral fat increase, shifts the body's centre of mass, increasing cervical spine stress and altering muscle activation patterns. These changes result in the underactivation of deep cervical flexors and overactivation of the superficial muscles, which are the sternocleidomastoid and upper trapezius, leading to pain and fatigue. Increased adiposity promotes systemic inflammation, further sensitising pain pathways. Weight control is emphasised as important for MNP prevention, with diet and exercise reducing mechanical load, pain, and inflammation. Correction of posture, such as scapular stabilisation and deep cervical flexor exercises, helps normalise muscle function and posture. Ergonomic workplace adjustments are also necessary for diminishing FHP and preventing cervical spine stress. Therefore, prevention and management of MNP rely on targeted interventions addressing obesity, highlighting need for further research into its biomechanical impact on the cervical spine.

Keywords: Mechanical neck pain, Obesity, Posture change, Adiposity, Fatigue

INTRODUCTION

Neck pain (NP) is primarily characterised by discomfort occurring between the superior nuchal line, an imaginary transverse line at the apex of the first thoracic spinous process, and laterally by sagittal planes adjacent to the neck's lateral boundaries¹. Pain in the neck may be localised and/or referred to the head and one or both upper extremities^{2,3}. Additionally, NP can also be categorised by the duration of pain, such as acute, subacute, and chronic pain, or by the cause of its onset.

“Mechanical neck pain” (MNP) is also referred to as “non-specific neck pain”⁴. Nonspecific neck pain (NP) patients may have an undefined pathoanatomic cause for their disease, and this is taken to mean having mechanical or nonspecific neck pain as a discernible direct pathoanatomic cause⁵. Kanlayanaphotporn et al.⁶ proposed a definition of MNP, i.e., pain predominantly localised to the posterior part of the neck, which may be aggravated by neck movements or prolonged postures. Several studies have employed the definition of MNP with minor modifications. MNP is frequently associated with forward head posture (FHP), characterised

by an excessive anterior displacement of the head relative to the vertical reference line. Excessive anterior positioning of the head denoted the displacement of the head's reference point (external auditory meatus) anterior to the vertical reference line⁷.

While the mechanism of NP may be connected to degenerative processes or pathologies revealed through diagnostic imaging, the specific tissue responsible for a patient's NP is typically unidentified. The global prevalence of mechanical neck pain is estimated to be 86.8%, and one in two people may, at some point in their lives, struggle with it⁸. The primary issue contributing to mechanical neck discomfort is the degradation of neck biomechanics. Optimal neck biomechanics relies on the longus colli and longus capitis muscles, referred to as deep cervical flexor muscle. The activation and endurance deficits of these muscles predispose individuals to mechanical neck pain⁹. Moreover, a segmental degenerative alteration that arises alongside balance and visual system deficiencies due to age might induce mechanically derived neck pain by compromising proper posture biomechanics and muscular endurance¹⁰. Increased body mass index has been reported to adversely impact body biomechanics¹¹. Mechanical neck pain is a common musculoskeletal disorder with considerable socioeconomic and healthcare implications. Although various factors contribute to its development, emerging research indicates that obesity is a significant factor in cervical spine dysfunction. Obesity leads to postural adaptation, enhances spinal loading, and modifies neuromuscular control, all of which contribute to the chronicity and severity of musculoskeletal pain¹². Despite the increasing global incidences of both obesity and MNP, the biomechanical relationship between the two disorders remains inadequately comprehended, as biomechanics and muscle endurance¹¹. Increased BMI has been reported to adversely impact body biomechanics¹³.

According to the World Health Organisation, a BMI of 19.9 kg/m² is considered overweight, while a BMI of 24.9 kg/m² is considered obese; the rate of overweight has been increasing rapidly worldwide. The percentage of people with a BMI exceeding normal has increased to 52.1% globally¹⁴. The global prevalence of obesity has more than doubled during the last three decades. The persistent growth in obesity prevalence indicated that existing therapies are inadequate; it is feasible to anticipate a rising number of individuals facing problems linked to excessive body weight. Substantially elevated body weight is regarded as a contributor to heart disease, diabetes, stroke, and certain cancer types¹⁵. Individuals with a BMI above 35 may experience a mortality increase of 40% and 62% in females and males, respectively, relative to those with a BMI within the normal range. Obesity is a global epidemic and poses a potential risk to the prolonged decrease in American death rates. Worldwide, overweight and obesity represent the fifth principal risk factor for mortality, accounting for approximately 2.8 million deaths per year¹⁶.

Despite the growing interest in the correlation between obesity and musculoskeletal illness, substantial gaps persist in comprehending the biomechanical effects of obesity on MNP. Most studies focus on the impact of obesity on the lumbar spine and knee joint, with limited biochemical analyses of the cervical spine. The absence of qualitative data on the impact of excess body weight on cervical spine alignment, posture, and muscle performance complicates the establishment of definitive biomechanical connections between obesity and MNP (Figure 1).

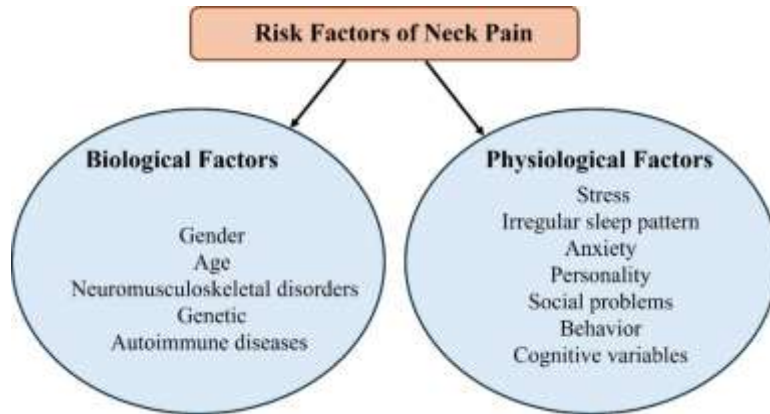


Figure 1 The impact of postural adjustments and load distribution is still insufficiently investigated. Obesity alters the center of mass and undermines postural stability, thereby exacerbating cervical strain and muscle imbalances.

Obesity and pain

There is adequate evidence to suggest the co-existence of pain complaints and obesity. Based on electronic medical record evidence in the Veterans Health Administration, obesity was significantly and consistently associated with chronic pain complaints (odds ratio [OR]=1.89, 95% confidence interval [CI], 1.56–2.30)¹⁷. The same results have been shown in adolescents and children, as well; obesity is highly associated with the prevalence and severity of pain complaints in the young¹⁸⁻²⁰. Similarly, in a multistate study²¹, incrementally, it was shown that the relationship between obesity and pain, i.e., pain complaint, was more prevalent with increasing BMI status. The likelihood of morbidly obese subjects reporting a complaint of pain was four times higher than among non-obese individuals. Incidence of low back pain increases with increasing BMI; less than 3% of subjects in the normal BMI group had low back pain in the last 3 months, compared to 7.7% for obese and 11.6% for morbidly obese subjects reporting low back pain within the US cohort study of 6,796 adults²². A large survey with more than 1 million individuals in the USA²³ demonstrated a linear increase in cases of chronic pain with increasing BMI. Compared to normal-weight individuals, obese individuals reported a 20% higher rate of ongoing pain, and the figures increased to 68% with individuals who had class I obesity, 136% with individuals who had class II obesity, and 254% with morbidly obese individuals. There is sufficient evidence that chronic pain and obesity do coexist. A community study²⁴ was able to document that obesity was linked to diverse pain diagnoses such as low back pain, headaches, fibromyalgia/chronic widespread pain, and abdominal pain (Figure 2).

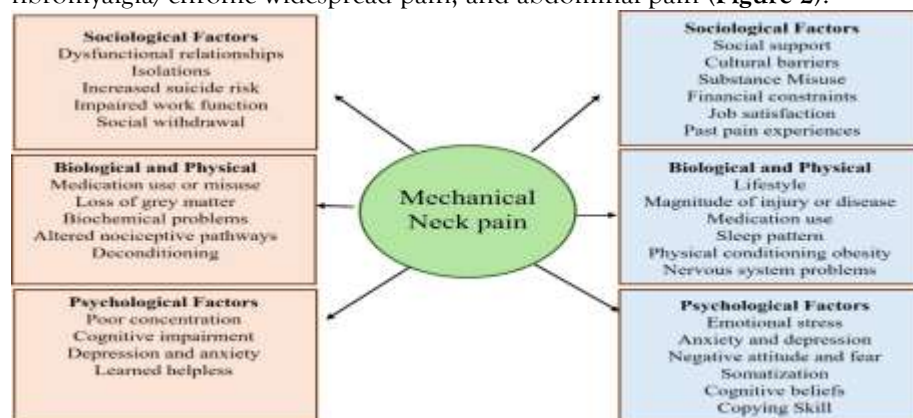


Figure 2

Severe obesity in older adults doubles the risk of chronic pain²⁵. A systematic review²⁶ concludes that obese patients are at higher risk of developing headaches, especially chronic headaches. Likewise, obesity seems to be a risk factor for the development of abdominal pain²⁷, pelvic pain²⁸, and neuropathic pain²⁹⁻³⁰.

On the contrary, obesity is prevalent in individuals with chronic pain. Individuals with reported widespread pain had higher total fat mass and lower total lean mass compared to non-pain reports³¹. Yunus et al.³² demonstrated that more than 60% of the women with fibromyalgia in their study were overweight or more, with 32.2% in the obese category. Another study demonstrated that fibromyalgia patients have higher mean BMI compared to pain-free individuals³³. In another study, up to 58% of women with fibromyalgia were obese³⁴⁻³⁵. Similarly, Neumann et al.³⁶ indicated that among 550 women with fibromyalgia, 28% were overweight, while 45% were obese. Loevinger et al.³⁷ indicated that higher waist circumference, low-density lipoprotein cholesterol, and triglyceride were associated with chronic pain, suggesting that chronic pain is associated with a higher risk of metabolic syndrome. Longitudinal studies indicate that obesity may serve as a risk factor for the onset of chronic pain. A population-based study including more than 30000 participants over a period of 10 years in Norway indicates that obese persons, especially those who are sedentary, face an elevated risk of acquiring chronic arm discomfort (Figure 3)³⁸.

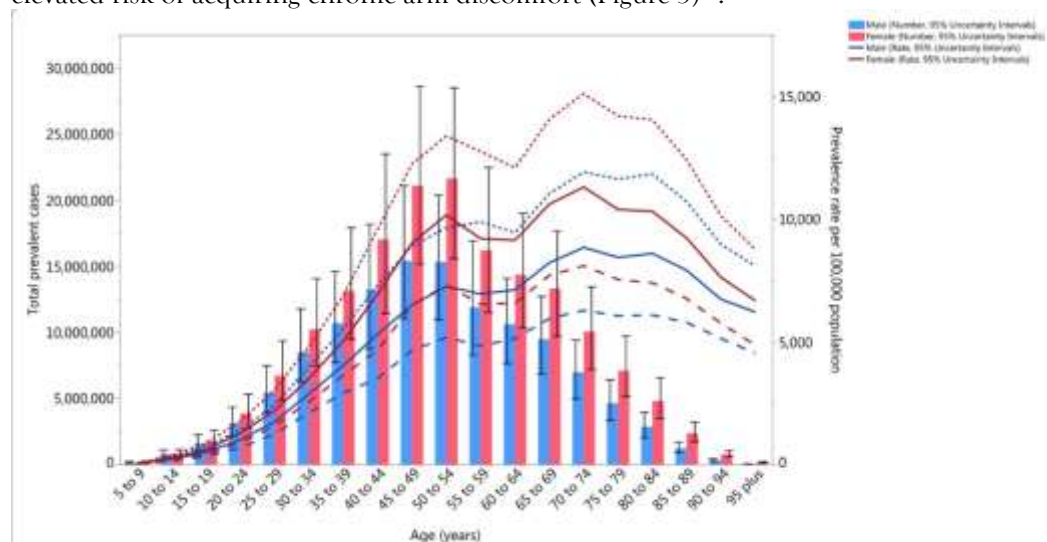


Figure 3

A comprehensive population-based survey involving over 25450 individuals indicated that a higher proportion of obese participants at baseline experienced low back pain 11 years later compared to the nonobese participants.

Biomechanical link between obesity and MNP

Neck pain is the leading cause of disability worldwide, with a prevalence ranging from 16% to 75% among various populations⁸. Obesity is a strong risk factor for this type of MNP, and studies have reported an increased prevalence of chronic neck pain in overweight and obese persons. The association between obesity and MNP is most likely mediated by mechanical, postural, and inflammatory processes³⁹. Body mass index (BMI) has been widely used to assess obesity-related health risks but does not consider individual fat distribution, which is a major determinant of the cervical spine³⁹. Central obesity, characterised by increased visceral fat, displaces the centre of mass of the body forward, loading the cervical spine with increased stress. Obesity is also related to higher systemic inflammation with elevated levels of pro-inflammatory cytokines, interleukin-6, and tumour necrosis factor-alpha, with heightened sensitivity to pain and extension of musculoskeletal pain³⁸. Co-morbid conditions of obesity including diabetes, metabolic syndrome, and inactivity also result in muscle deconditioning and joint instability. A sedentary lifestyle in obese persons further compromises the deep cervical muscles, leading to poor postural control and an increased risk of

chronic pain⁴⁰. Psychological disorders, including depression and stress, which are more prevalent in obese people, can also lead to heightened pain sensitivity and a greater risk of chronic neck pain⁴¹.

Excess weight of the body produces significant postural changes, particularly in the thoracic and cervical spine. Forward head posture (FHP) is another of the most common obesity compensation postures. FHP, or anteriorly displaced head positioning relative to the trunk, presents as a bearing load on the cervical spine⁴². Studies have found that with every inch of anterior displacement of the head, the effective weight of the head increases by approximately 10 pounds and produces more tension within the cervical joints and muscles⁴³. Redistribution of body weight in obesity also produces compensatory changes of the thoracic and lumbar spine. Thoracic kyphosis and lumbar lordosis are common findings in obese individuals as the body tries to resist the forward displacement of mass³⁹. The spinal deformities change the pattern of forces along the vertebral column.

Proprioceptive dysfunction is another consequence of postural change in obesity. Abnormal excess fat deposition interferes with sensory feedback, and faulty head-neck coordination and increased postural instability follow. Proprioceptive dysfunction can also be a cause of chronic pain by increasing muscular tension and decreasing neuromuscular efficiency in head and neck stabilisation. Obesity elicits extreme cervical muscle activation pattern changes, primarily due to increased mechanical loads and postural instability⁴². Electromyographic (EMG) testing identified that MNP subjects have increased superficial neck muscle activation, such as sternocleidomastoid (SCM) and upper trapezius, and reduced deep cervical stabiliser activity, such as the longus colli and longus capitis, respectively³⁹.

This muscle compensatory recruitment mechanism is a result of increased cervical loading. When obesity-induced respiratory dysfunction and postural loading compromise the stabilising deep muscles, superficial muscles take on increased load in stabilising the head and become fatigued and painful⁴³. As body weight increases, cervical muscles labor more in stabilising the head, which is more energy-intensive and results in increased muscle fatigue and pain⁴⁰. It has been established that obese patients have increased activity in the cervical region muscles and decreased relaxation, typical of an adaptive response to increased mechanical stress⁴³. Obesity-induced respiratory dysfunction also indirectly influences neck muscle function. Obese patients typically have deranged breathing patterns like shallow breathing and increased usage of accessory breathing muscles like SCM and scalene muscles. This increased respiratory workload puts an extra burden on the cervical musculature, leading to extra pain and dysfunction⁴².

Excess weight significantly affects spinal loading, accelerating degenerative change in the cervical spine. Obese patients have a higher risk of cervical spondylosis, intervertebral disc degeneration, and facet joint osteoarthritis owing to chronic exposure to abnormal mechanical stress (Table I). Intervertebral discs, being shock absorbers, are prone to compression forces owing to obesity. Literature has proven that increased BMI is associated with reduced disc height, increased disc bulging, and increased incidence of herniated discs³⁸. Increased loading causes dehydration of the nucleus pulposus, which renders the disc less effective in evenly distributing evenly distributed forces, which increases shear stress on adjacent vertebrae.

Facet joint dysfunction is the second major problem in obese MNP patients. Biomechanical changes result in a change in spinal positioning, which places facet joints under higher axial loading, resulting in cartilage degeneration and inflammation. Facet joint arthropathy is the resulting pathology causing chronic pain and reduced range of motion in the cervical spine. Besides, metabolic derangement due to obesity may also contribute to enhanced spinal degeneration⁴⁴. Pro-inflammatory mediators secreted by adipose tissue induce degeneration of cartilage and inhibit tissue repair³⁸. Inflammation also supports the pathogenesis of osteoarthritis and other degenerative spinal lesions. Lastly, obesity is associated with enhanced spinal loading, and this is associated with reduced vertebral blood supply and reduced delivery of nutrients to the intervertebral discs. These vascular remodeling changes result in enhanced disc aging and reduced healing potential, and thus obese patients are at increased risk of chronic pain syndromes and reduced spinal resilience⁴⁵.

Role of Postural adaptation and load distribution

Obesity is a significant public health issue with far-reaching consequences for musculoskeletal health, particularly affecting spinal alignment and posture (Figure 4)⁴⁶.

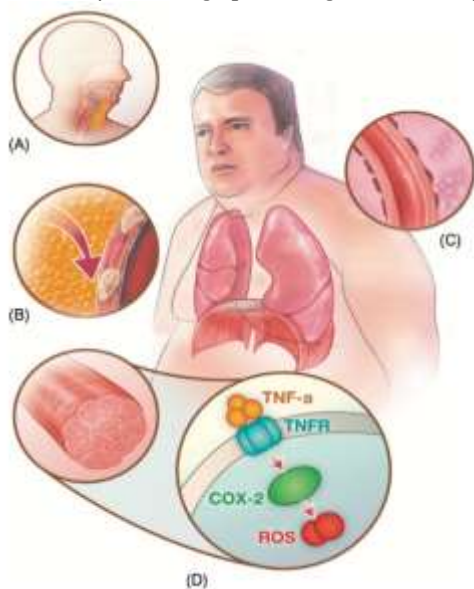


Figure 4

With the escalating rise in the prevalence of obesity globally, understanding the impact of excess weight on spinal mechanics, with an emphasis on the cervical spine, has become more critical. Among the significant issues is how postural adaptations due to obesity lead to cervical spine misalignment and to the progression of neck pain, balance impairment, and coordination impairment⁴⁷. This narrative review discusses the role of obesity in affecting postural adjustments, the distribution of weight, the resultant stress on the cervical spine, and the more general effects on balance and coordination.

The cervical spine is situated at the top of the spine and is under more stress when the head is bent forward in order to balance with the centre of gravity⁴⁷. FHP puts extra strain on the muscles of the neck, especially the extensor muscles, as they require more work to hold the head⁴⁸. Research shows that obese people are more prone to FHP, which will lead to chronic neck pain and misalignment^{46,49}. The cervical spine, which is not designed to carry much weight, becomes stressed more as the forces are not distributed evenly, and thus there is mechanical stress and possible spinal misalignments.

Obesity impacts the alignment of the spine, not only in the neck region but also in the middle and lower regions of the spine. Research has established that obese individuals have changes in the upper back curve (thoracic kyphosis) and lower back curve (lumbar lordosis), which also influence the alignment of the neck. When the upper back is more curved and the lower back is less curved, it alters the function of the spine and causes it to experience abnormal stress. These changes in posture can be seen in the gait and movement patterns of the obese population. The thoracic kyphosis and changed lumbar alignment can cause the flexion of the cervical spine to compensate and rise to balance and not overload the lower back. A cervical vertebrae formed misalignment due to the head moving forward and downward, resulting in increased tension and pain in the neck and shoulders⁵⁰. The changed lumbar spine alignment influences coordination between the upper and lower body, which leads to poor posture and further enhances the risk of musculoskeletal disorders, such as neck pain.

The changes in weight and standing posture make it more difficult to remain balanced. Postural changes due to changes in weight are one of the major issues that lead to changes in balance and coordination. When the centre of gravity of a person shifts forward, it becomes difficult for obese persons to remain balanced when walking or standing. The change reduces the surface area under the body, and thus they have to employ other

means to remain balanced⁴⁷. When the trunk and head lean forward even further, it takes more energy to stabilise the body. This makes it harder to move and creates more fatigue. The neck and back muscles have to work harder, which destabilises the spine and makes it more likely to fall. Because of this, obese people tend to have worse balance control. This not only makes them more likely to fall but also puts more pressure on the neck⁵¹. Additionally, defective balance and coordination tend to go hand in hand with decreased proprioception, the perception the body has of where it is in space. This unawareness can cause further falls and bone and muscle injury. Indeed, research indicates that obese individuals tend to fall more than non-obese individuals. This is usually associated with alterations in posture and coordination issues⁵².

Obesity-caused postural change also results in cervical spine misalignment, increasing the risk of musculoskeletal injury. The stress on the neck muscles and the altered force transmission to the cervical vertebrae can result in chronic diseases such as neck pain, cervical spondylosis, and, in severe cases, nerve compression or disc herniation (Table II). The added stress on the cervical spine due to FHP and poor spinal posture results in muscular fatigue and joint degeneration, predisposing the person to injury. The compromised postural stability and coordination also increase the risk of falls and cervical spine injury^{53,54}.

Clinical and Preventive Implications and Weight Management

Pain relief after weight loss can be seen aside from dynamic strength, joint alignment, and degree of cartilage degeneration in obese knee OA patients^{55,57}. Hence, weight loss is a very important aspect of the treatment of pain. Development of weight management interventions in obese patients with arthralgia is problematic. However, on average, at least 1500 kcal/wk of energy expenditure is recommended for weight loss⁵⁸. Sole emphasis on the achievement of energy expenditure targets can be overwhelming to this population in the setting of pain flares, and psychosocial factors can happen on a weekly basis. One potential method for starting a program is to stabilise the musculature and solidify painful joints before initiating weekly sessions of weight-bearing aerobic exercise. Self-efficacy is maximised with exercise and reduces depressive symptoms⁵⁹ and joint pain⁶⁰. Modifying these factors can prepare an individual for regular aerobic exercise, increasing energy expenditure. Regular resistance exercise is linked to reduced analgesic use for joint pain compared to no participation in strengthening activity⁶¹. Strengthening exercises have other effects, such as improved mobility, functional improvement, proprioceptive awareness, and postural control⁶². Combining posture training and multisensorial training (training protocols enhancing the perception of posture position from feet to the occiput) with resistance exercise can improve mobility changes in obese subjects with chronic musculoskeletal pain (e.g., hyperkyphotic spine).

A substantial reduction in depressive symptoms (by 43%) might transpire following a fall in BMI from 51.9 to 31.8 kg/m² during one year. Supervised exercise and food modification programs are beneficial for achieving sustained weight loss, alleviating musculoskeletal discomfort, and enhancing functional mobility⁶⁰. Individual or integrated exercise and dietary therapies are efficacious in enhancing ambulation duration, gait metrics, and stair ascent time. Seventy-nine dietary adjustments often involve a calorie reduction of roughly 500 kcal per day. A correlation has been shown between physical exercise and pain sensations. Obese women face a 20% increased risk of persistent low back, neck, and shoulder discomfort compared to nonobese women, engaging in over one hour of exercise weekly. One study assessed the effects of two low-energy diets (415 or 810 kcal/day for 2 months, followed by 1200 kcal/day for another 2 months) on weight loss and knee osteoarthritis symptoms. Both groups achieved similar weight reductions (12–13% of body weight), with an 11–12% decrease in pain. The trial evaluated the effectiveness of diet, exercise, and a combined diet and exercise intervention on body weight, pain symptoms, and physical function in obese individuals with knee osteoarthritis¹⁸. Self-reported knee joint pain diminished by 30% in the combination intervention, while pain reduction ranged from 6% to 17% in the other groups. The combined therapy group improved walking distance and stair climb time by up to 23%. A mean weight reduction of 9.2 kg decreased the ambulatory compressive pressures at the knee by 2.6%. Additional research indicates that weight loss in osteoarthritis diminishes systemic inflammation (interleukin 6 and C-reactive protein) and collagen breakdown while enhancing cartilage formation, suggesting a possible protective impact against further deterioration.

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Tables

Table I. Summary of Biomechanical Changes in Obesity-Related Neck Pain

Biomechanical factor	Effect on obesity patients	Impact on NP	References
Forward head posture	Increased cervical extension	Higher strain on neck muscles and joints	Hoy <i>et al.</i> (2010) ⁴¹ and Damasceno <i>et al.</i> (2018) ⁶³
Increased spinal loading	Excessive pressure on intervertebral discs	Risk of early degeneration and pain	Cheng <i>et al.</i> (2016) ⁴³
Reduce range of motion (ROM)	Stiffness and limited mobility	Exacerbates pain and increased injury	Genebra <i>et al.</i> (2017) ⁸ and

			Hoy et al. (2010) ⁴¹
Increased inflammatory markers	Elevated proinflammatory cytokines	Pain sensitization and tissue degeneration	Stannus et al. (2010) ⁶⁴
Overactive upper trapezius	Compensatory mechanism for weak flexors	Tension-type headaches and chronic pain	Dennison et al. (2016) ⁴
Impaired proprioception	Reduced proprioceptive awareness	Poor postural control and injury risk	Vincent et al. (2012) ⁶⁵
Reduced physical activity	Sedentary behavior and muscle deconditioning	Weakness and stiffness in the cervical region	Safiri et al. (2021) ³⁸
Altered Thoracic spine alignment	Increased thoracic kyphosis	Altered cervical alignment and increased strain	Kado et al. (2004) ⁶⁶
Increased fat deposition in the cervical region	Fat accumulation around the neck	Additional mechanical stress on the cervical structure	Cheung et al. (2013) ⁴⁵
Weak deep cervical flexors	Reduced stability of the cervical spine	Muscle fatigue and stiffness	Falla et al. (2004) ³⁹ and Jull et al. (2013) ⁴⁰

Table II. Soft tissue and inflammatory contributions to Neck pain

Factor	Mechanism	Contribution to neck pain	References
Impaired blood flow	Obesity and inflammation reduce microvascular circulation	Inadequate oxygen and nutrition supply hinders muscle regeneration and exacerbates stiffness.	Fejer et al. (2006) ⁶⁷ ; Burri et al. (2015) ⁶⁸
Proinflammatory cytokines	Adipose tissue releases TNF- α , IL-6, and C-reactive protein	Chronic low-grade inflammation increases pain perception by sensitizing pain pathways	Sturgeon et al. (2016) ⁶⁹
Muscle dysfunction	Altered muscle activation pattern and delayed recovery from fatigue	Compensatory motions and chronic fatigue elevate the stress on cervical muscles	Falla et al. (2017) ⁷⁰
Central sensitization	Chronic inflammation and pain lead to CNS hyper-responsiveness	Induces pain signals, leading to chronic pain conditions	Nijs et al. (2021) ⁷¹
Fat infiltration in muscle	Fatty deposition in cervical muscle	Reduce contractile efficiency, leading to muscle weakness, stiffness, and strain	Jones (2020) and Sluka & Clauw (2016) ^{72,73}
Sedentary lifestyle	Physical inactivity leads to muscle deconditioning	Decreases cervical muscular strength, heightening vulnerability to pain and injury	Geneen et al. (2017) ⁷⁴
Biomechanical stress	Elevated stress on the cervical spine resulting from obesity and suboptimal posture	Forward head position and degenerative alterations elevate stress on muscles and joints	Damasceno et al. (2018) ⁶³ Fejer et al. (2006) ⁶⁷
Immune system dysregulation	Adipose tissue macrophages release inflammatory cytokines	Sustain systemic inflammation, prolonged pain, and tissue injury	Jones (2020) ⁷²
Degenerative change	Chronic inflammation accelerates disc and facet joint degeneration	Facilitates structural alteration in the cervical spine, resulting in discomfort and rigidity	D'hooge et al. (2012) ⁷⁵ and Cohen & Mao (2014) ⁷⁶
Hormonal imbalance	Dysregulation of pro-inflammatory leptin and anti-inflammatory adiponectin	Promotes systemic inflammation and impairs muscle repair	Sturgeon et al. (2016) ⁶⁹
Psychological factors	Depression, anxiety, and stress associated with chronic pain	Reduces pain tolerance and escalates pain perception	Bair et al. (2003) ⁷⁷

Figure legends

Figure 1 Factors associated with Mechanical neck pain

Figure 2 Obesity and mechanical neck pain factors

Figure 3 Global incidence of the age-standardized annual incidence rate of neck pain per 100000 population³⁸

Figure 4 Obesity and Musculoskeletal Pain⁴⁶