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REVIEW ARTICLE

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A Narrative Review of the Impacts of Obesity on Pulmonary Function and Muscle Strength

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Abstract

Obesity has reached alarming proportions worldwide, requiring close attention and comprehensive knowledge about its health consequences. Obesity today affects people of all ages and socioeconomic levels. This narrative review is devoted to the multifaceted impacts of obesity on pulmonary function and muscle strength. Obesity, characterized by excessive fat accumulation, induces complex physiological changes, transforming adipose tissue into a metabolically dynamic organ. The multifarious nature of obesity causes complicated physiological alterations that affect the whole body. Besides its well-known impacts on metabolic health, obesity, particularly abdominal obesity, challenges the respiratory system mechanically. This review navigates through the mechanical challenges that obesity poses to pulmonary function, elucidating how excess adipose tissue in the abdominal region compromises lung expansion and increases the workload on respiratory muscles. Simultaneously, the review explores the dynamic interplay between obesity and muscle strength. Obesity and muscle strength are linked by metabolic dysfunction, muscle composition changes, and lifestyle variables. Clinical implications of obesity extend beyond metabolic consequences, emphasizing impaired pulmonary function and diminished muscle strength as crucial determinants of clinical outcomes. A multidisciplinary approach involving collaboration among healthcare professionals is advocated, addressing the physiological and psychological factors contributing to obesity.**(International Journal of Biomedicine. 2024;14(2):217-226.)**

Keywords: adipose tissue • obesity • pulmonary function • muscle strength

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Abbreviations

AT, adipose tissue; **BMI**, body mass index; **CBT**, cognitive behavioral therapy; **COPD**, chronic obstructive pulmonary disease; **ERV**, expiratory reserve volume; **FEV1**, forced expiratory volume in 1 second**; FVC**, forced vital capacity; **FRC**, functional residual capacity; **MIP**, maximal inspiratory pressure; **MEP**, maximal expiratory pressure; **OSA**, obstructive sleep apnea; **OHS**, obesity-hypoventilation syndrome; **WHO,** World Health Organization.

Introduction

Obesity is a metabolic state generated by the expansion of adipose tissue resulting from a complex interaction of hereditary, environmental, and behavioral variables.(1) The primary cause of obesity is an imbalance between energy intake and expenditure, disrupting energy homeostasis and resulting in initial fat accumulation in adipose tissue and subsequent accumulation in other tissues.

Obesity has reached worrisome proportions globally due to lifestyle and nutritional changes, requiring careful attention and a comprehensive knowledge of its health effects. Around 800 million people worldwide are living with obesity, according to statistics from 2016. WHO (2) estimates that by 2025, approximately 167 million more people will become less healthy because they are overweight or obese. This frequency crosses socioeconomic borders, impacting people in industrialized and underdeveloped countries.

The obesogenic environment, with the easy availability of high-calorie meals and a sedentary lifestyle, leads to rising obesity rates. Obesity contributes to the development of type 2 diabetes, cardiovascular disease, liver dysfunction, and cancer, which collectively account for over 70% of premature deaths worldwide.[3-7] This highlights the critical need for comprehensive initiatives that address lifestyle choices and social and environmental variables affecting obesity.

Body mass index (BMI) is one of the ways to measure obesity in the population.[8-10] Obesity is linked not only to an increased BMI and body weight but also affects multiple physiological systems, causing complex alterations beyond adipose tissue.[11] Obesity involves a complex interaction of metabolic, hormonal, and inflammatory variables that disrupt the body beyond outward indicators of obesity and cause a variety of health issues.[12] Global obesity is a major public health issue. Obesity today affects people of all ages and socioeconomic levels. The WHO considers obesity a major risk factor for a variety of chronic illnesses, including cardiovascular disease, metabolic disorders, and cancer.[13] This change in viewpoint emphasizes the need for a comprehensive strategy to treat obesity's complex health concerns beyond its evident physical effects.[8,11]

This review aims to assess the complex interactions in obesity and, in particular, the impact of obesity on pulmonary function and muscle strength.

Some Physiological Changes in Obesity

Adipose tissue is an endocrine organ that not only stores lipids but also secretes various biologically active substances, such as cytokines, adipokines, chemokines, and hormonal factors, that regulate metabolic processes in the organism and affect inflammation and endocrine functions.[14,15] Adipokines are involved in many functions and processes, including modulation of energy and appetite, lipid and glucose metabolism, insulin sensitivity, endothelial cell function, inflammation, blood pressure, the development of metabolic syndrome, and atherosclerosis.[16,17] Visfatin, a recently discovered adipokine, has been positively correlated with the accumulation of adipose tissue. Visfatin has pro-oxidant and pro-inflammatory activity and is elevated in obese individuals. Chemerin, highly expressed in white adipose, is involved in inflammation, angiogenesis, adipogenesis, energy metabolism, and oxidative stress.[16] Chemerin is best characterized as a chemoattractant for dendritic cells and macrophages. Most studies report increased chemerin levels with increased body weight.

Obesity is associated with the activation of pro-inflammatory adipokines and the development of chronic low-grade inflammation.[18,19] One of the best-known pro-inflammatory adipokines is leptin. Leptin, a hormone synthesized mainly in adipocytes, provides central weight control and appetite regulation. Obesity-induced hyperleptinemia stimulates the production of pro-inflammatory cytokines such as TNF-α, IL-6, IL-2, IL-1β, and IFN-γ by monocytes and T-helper 1 and also inhibits the production of the anti-inflammatory cytokine IL-4.[20,21] Studies have also shown that leptin increases serum levels of C-reactive protein. The abnormal expression of TNF-α in adipose tissue plays a critical role in peripheral insulin resistance in obesity. It has been demonstrated as a marker of insulin resistance.[22]

Inflammation contributes to the development of leptin resistance. Leptin resistance reduces leptin's ability to send satiety signals to the brain, causing overeating and weight gain.[23] In contrast to leptin, adiponectin, which is secreted by differentiated adipocytes, has anti-inflammatory and anti-atherogenic effects. In obesity, adiponectin, a key adipokine for insulin sensitivity and glucose control, paradoxically decreases,[24,25] causing metabolic events such as impaired glucose absorption and a higher risk of type 2 diabetes.[24] The complex hormonal and inflammatory environment of obesity's adipose tissue shows its dynamic character.[24,25]

Various biologically active substances secreted by adipose tissue, including adipokines, cytokines, chemokines, excess lipids, and toxic lipid metabolites, promote insulin resistance, an impaired biologic response of target tissues to insulin stimulation. Abdominal obesity and insulin resistance, along with hypertension and dyslipidemia, are key components of metabolic syndrome,[26] which contributes to endothelial dysfunction, platelet hyperactivity, oxidative stress, and low-grade inflammation, resulting in the development of cardiovascular disease.

Dyslipidemia (elevated levels of triglycerides and low-density lipoprotein cholesterol), low-grade inflammation, and metabolic dysregulation associated with obesity contribute to the development of atherosclerosis and cardiovascular complications.[27,28]

In obesity, the total blood volume increases due to adipose tissue excess, leading to increased stroke volume and cardiac output.[29] Increasing cardiac output in obese patients is intended to meet the metabolic demands of excess adipose tissue. The left ventricle (LV) dilates to accommodate the increased venous return and becomes hypertrophied. The left atrium also dilates due to increased circulating blood volume and elevated LV diastolic filling pressure. A dilated left atrium and increased LV filling pressure increase the risk of heart failure and atrial fibrillation. Changes in myocardial structure due to infiltration of adipose tissue predispose to conduction abnormalities and ventricular arrhythmias,[30,31] worsening heart failure. The long-term cardiovascular effects of metabolic dysfunction in obesity necessitate a comprehensive strategy.

Obesity and Pulmonary Function

Besides its well-known impacts on metabolic health, obesity, particularly abdominal obesity, challenges the respiratory system mechanically. Excess body fat, or obesity, affects pulmonary function via several routes.[32] Obesity limits lung expansion mechanically. Excess chest and abdominal fat restrict the diaphragm's fall during inhalation, reducing lung expansion. Obesity alters the breathing pattern, resulting in a substantial reduction in both the expiratory reserve volume and the resting volume of the lung, known as the functional residual capacity (FRC).[33] The reduction in FRC is proportional to the

severity of obesity – overweight, mildly obese and severely obese subjects without asthma demonstrate reductions in FRC of up to 10%, 22% and 33%, respectively.[34]

The lowering in FVC and FEV1 impedes gas exchange. Weight also strains respiratory muscles, making them work harder to overcome resistance.[35] This increased effort may cause tiredness and respiratory muscle weakness. Obesity is linked to chronic inflammation beyond mechanical consequences.[25,36] Inflammatory mediators released by adipose tissue in obesity may affect airway anatomy and function.[37] This inflammatory milieu may cause respiratory disorders, including asthma and chronic obstructive pulmonary disease (COPD). Leptin resistance and other hormonal variables significantly influence the complex interaction between obesity and pulmonary function. Adipose tissue produces leptin, which regulates hunger and energy. Obesity may cause leptin resistance.[38] Leptin resistance may upregulate the generation of reactive oxygen species, increasing oxidative stress and promoting inflammation in airways and lung parenchyma.[39]

Obstructive sleep apnea (OSA), strongly associated with obesity, especially central obesity, complicates matters. Obesity, especially around the neck and throat, narrows or collapses the upper airway, obstructing sleep. Obstructive sleep apnea causes disrupted sleep patterns, periodic hypoxia and hypercapnia, straining the respiratory system. The cyclical nature of OSA, with periods of slowed or stopped breathing followed by sudden awakenings, makes it harder for obese people to maintain adequate respiratory function. Disrupted sleep habits may increase weight gain, affecting hormone balance and hunger, causing daily weariness and impairment of cognitive performance.[40]

Obesity-related neuromuscular, mechanical, and metabolic factors may cause obesity hypoventilation syndrome (OHS), [41,42] also known as Pickwickian syndrome. Obesity hypoventilation syndrome is defined as the presence of awake alveolar hypoventilation characterized by daytime hypercapnia, which is thought to be a consequence of diminished ventilatory drive and capacity related to obesity. Obesity hypoventilation syndrome results from the mechanical load on the respiratory pump, leading to low tidal volumes and blunting of the chemoreflex to CO_2, leading to inappropriate central respiratory effort in individuals with obesity. The hypercapnia of OHS may be augmented by leptin resistance. Individuals with OHS have a greater degree of central obesity reflected by larger neck circumferences and higher waist-to-hip ratios than those with eucapnic obesity or OSA, which explains the lower lung volumes seen in such individuals.[43] Individuals with OHS are exposed to prolonged periods of daytime and nocturnal hypoxia and are consequently at higher risk for pulmonary hypertension and cor pulmonale.[44-47] Obesity hypoventilation syndrome is more common in women than men and postmenopausal women with OSA have a higher prevalence of OHS due to hormonal influences.

Due to lower lung function, obese people may also be more susceptible to respiratory infections. Obesity's impaired mucus clearance and immune response promote respiratory diseases, including pneumonia and bronchitis. Airway hyperresponsiveness, a hallmark of asthma, is linked to obesity. Due to airway sensitivity, obese people may acquire or worsen asthma. Asthma is one of the best-characterized diseases related to obesity. A meta-analysis involving over 300,000 adults found obesity and asthma were related, and the risk of asthma increased with increasing BMI.[48] Excess weight weakens the respiratory muscles, reducing airflow, especially when the respiratory load is high.

Thus, respiratory dysfunctions observed in patients with obesity are characterized by impaired breathing mechanics, decreased respiratory system compliance, increased small airway resistance, and alterations in both breathing patterns and respiratory drive. [49-52]

Pulmonary Function Assessment

The multimodal evaluation of pulmonary function in obese people provides useful insights into the complex relationship between obesity and respiratory health. FVC and FEV1 readings in pulmonary function tests frequently suggest a restrictive pattern, indicating decreased lung capacity. Body fat distribution patterns have a stronger relationship with lung function than weight or BMI.[53,54] A large population-based study of 121,965 people found that abdominal obesity predicted FEV1 and FVC independent of BMI.[53] Changing pulmonary function due to obesity is not limited to volume changes. Respiratory mechanics are also impacted, with greater breathing effort and decreased respiratory muscle efficiency. This is essential because it shows that obesity and respiratory health are dynamic beyond volume measures.[35]

Some studies showed that measuring respiratory system impedance may be a more sensitive measure of lung dysfunction related to obesity than spirometry.[55-57] Whole-body plethysmography, impulse oscillometry, or the forced oscillation technique can also be used to assess the mechanical properties of the airways in obese individuals. Respiratory muscle strength can be assessed by measuring maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP). In obese individuals, both MIP and MEP may be reduced.

Obesity also affects gas exchange. Ventilation-perfusion mismatch and poor diffusion capacity are common, stressing the necessity for a complete respiratory examination. Comprehensive examinations are important for identifying obesity's increased risk of respiratory disorders, such as hypoventilation syndromes and OSA.[58]

Obesity-related changes in pulmonary function vary by person. Body fat distribution, obesity duration, and comorbidities affect pulmonary function measures. Thus, obesity-related pulmonary function testing must be nuanced and customized for correct diagnosis, risk classification, and focused therapy.[59] A complete study of pulmonary function in obese persons gives a full understanding of the complicated link between excess body weight and respiratory health, enabling informed clinical decision-making and individualized therapy methods.

Obesity and Muscle Strength

Obesity and muscle strength are interconnected health factors. Body mass index, which incorporates weight and height, measures obesity as excessive body fat. In contrast, muscle strength encompasses muscle size, composition, and neuromuscular efficiency, determining force production.[60]

Obesity affects muscle mass, which is vital to body composition and health. Sarcopenia and excessive body fat commonly occur with obesity. The rise in body fat and decrease in lean muscle mass are combined issues. Strength depends on muscle mass. In obesity, extra body fat may hide muscle atrophy, making it harder to diagnose and treat. The role of adipose tissue in maintaining muscle mass and function has been well studied. Adipose tissue is an active endocrine organ that produces signaling chemicals that may cause inflammation. This inflammatory milieu may further catabolize muscle tissue, disrupting muscle protein production and breakdown.[61] Chronic, low-grade inflammation, often found in obesity, reduces muscle strength, impairs physical function, and can cause sarcopenia. Insulin resistance in obesity can affect the ability of muscle cells to use glucose for energy, affecting strength and function. Obesity-related inflammation can affect neuromuscular connections, leading to decreased coordinated contractility.[61,62]

The relationship between fat and muscle strength grows increasingly complex with age. Muscle mass and muscle strength naturally drop with age. Marcus et al. reported that intramuscular adipose tissue was inversely related to physical performance in older adults.[63] Moreover, aging results in a shift towards a higher proportion of type I muscle fibers, muscle fiber atrophy, especially in type II fibers,[64] and changes in muscle structure (i.e., pennation angle, and fascicle length).[65]

Sarcopenia, age-related muscle loss and function, is aggravated by hormonal changes, reduced physical activity, and nutrition metabolism changes. Obesity rises with age. Obesity and age together threaten muscular health. Sarcopenic obesity, when muscle loss and body fat increase with age, exacerbates this.[61] Chronic inflammation accelerates muscle deterioration in obese older adults. Aging and fat cause inflammation, which might make muscle tissue more vulnerable. Chronic inflammation increases muscle atrophy and age-related illnesses, including arthritis and cardiovascular disease, reducing mobility and functioning. The reduction in muscle mass and muscle strength caused by aging and obesity affects older persons' functional independence and quality of life. Muscle weakness may cause falls, fragility, and difficulty with regular tasks.

Obesity affects the musculoskeletal system beyond muscle strength. The skeletal system, including joints, carries the body's weight, and excessive load may damage joint function and health, especially knees and hips.[66] This increased strain may accelerate joint cartilage wear and tear, contributing to degenerative disorders and osteoarthritis.[67,68] The production of several pro-inflammatory cytokines during obesity contributes to joint tissue destruction.

Maintaining older physical strength is essential for mobility, independence, and well-being. To address the difficulties of aging and obesity, a personalized strategy is necessary. Interventions should include lifestyle changes, physical exercise, and nutritional choices to address the relationship between obesity and muscle strength. A balanced diet is essential for weight control. Regular aerobic and resistance training are needed to maintain muscle mass and muscle strength. Resistance exercise reduces age-related muscle loss and boosts strength.[69,70] Weight reduction should be gradual and sustained to minimize muscle loss.[71,72]

Muscle Strength Assessment

Examining muscle strength in obesity reveals a complex picture beyond weight control. Excess weight severely impacts musculoskeletal health, affecting muscle function and performance. Understanding the complex relationship between obesity and musculoskeletal function requires comprehensive muscle strength evaluation in obese people. Research consistently links obesity to decreased muscle strength in both proximal and distal muscle groups. Increased mechanical strain on weight-bearing joints, changed muscle composition, and extra adipose tissue-related inflammation contribute to this strength loss. These variables reduce muscular force production and endurance, affecting everyday functioning and quality of life.[61,62]

Isometric and isokinetic tests measure obesity-related muscular strength. Detailed strength analysis across joint motions reveals damaged muscle areas using these data. The assessment of arms, shoulders, and lower extremity strength is particularly relevant.

In two British cohort studies, grip strength was positively associated with BMI, while it was negatively associated with central obesity measured using waist circumference.[73,74] Some studies reported a relationship between muscle/grip strength with blood lipid profile.[75,76,77]

Systematically testing muscle strength helps uncover weakening patterns and comprehend functional consequences for everyday life and mobility.[59] Sedentary behavior worsens muscular weakness, fat and musculoskeletal health are linked. A complete health plan must include measures to preserve and improve muscle strength in obese people.

Interaction between Pulmonary Function and Muscle Strength

The relationship between pulmonary function and muscle strength is complicated, especially in obesity. Multiple factors, including mechanical, inflammatory, metabolic, and lifestyle variables, may affect this relationship in obesity. [35,66] As obesity increases, the function of the respiratory muscles may be impaired due to stress on the diaphragm. Respiratory muscle dysfunction may be partially explained by increased resistance caused by the presence of excess fatty tissue on the chest and abdomen, which leads to mechanical damage to these muscles.[78,79]

This increased breathing effort may cause dyspnea and weariness, especially during oxygen-intensive tasks.

Ventilatory muscle weakness reduces respiratory functions, vital capacity, and lung function. Inspiratory and expiratory flows may be impeded, affecting lung gas exchange. Reduced carbon dioxide clearance may cause respiratory issues and low oxygen saturation.

In addition to mechanical restrictions, obesity causes persistent low-grade inflammation that affects the respiratory system. This inflammation may damage airways and lung tissues, worsening asthma and chronic obstructive pulmonary disease. The inflammatory milieu in obesity may make the respiratory system more susceptible to malfunction, affecting pulmonary function.[37,80]

Obesity-related metabolic changes impair pulmonary function. The body's metabolic and respiratory systems are interconnected, highlighting obesity's systemic effects. Obesity contributes to difficulty breathing and decreased respiratory efficiency due to mechanical, immunological, and metabolic effects. Obesity-related muscle strength and respiratory alterations impair exercise tolerance. Obesity weakens respiratory muscles and muscle strength, making prolonged physical activity difficult. A positive feedback loop exists between exercise ability and musculoskeletal health.[81] Weak muscles and joint tension hinder physical activity, deconditioning muscles and reducing exercise capacity. Evaluating metabolic variables and their systemic effects on obesity is vital for comprehending the connection between the pulmonary and musculoskeletal systems.[82] Insulin resistance and persistent low-grade inflammation impair the respiratory and musculoskeletal systems. Insulin resistance, as a characteristic of obesity, is crucial to metabolic variables and musculoskeletal health. With insulin resistance, skeletal muscles absorb glucose less, and energy production by muscle cells decreases, leading to muscle weakness and fatigue. Muscle dysfunction may influence musculoskeletal health, mobility, and exercise performance.[83]

Understanding these pulmonary problems and functional muscle damage in the setting of obesity is essential for creating tailored therapies to improve respiratory health and avoid respiratory consequences in obese people.[35]

Non-Pharmacological Therapeutic Modalities for Obesity

Comprehensive obesity treatment emphasizes non-pharmacological therapies. Obesity control relies on lifestyle changes, especially nutrition. A balanced, calorie-controlled diet rich in nutritious foods, fruits, vegetables, and lean meats promotes healthy eating habits. Structured exercise regimens, which include aerobic and resistance training, help lose weight, maintain muscle mass, and improve metabolic health.[84] Thus, lifestyle modification, which generally consists of a combination of nutrition, physical activity, and behavioral modification, is an oft-used strategy to help patients achieve weight loss and maintenance.[85,86]

Several studies have investigated the effects of weight loss on airway reactivity.[56, 87, 88] Aaron et al.[87] found a trend towards reduced airway hyperresponsiveness with weight loss following an intense diet-induced weight reduction program in obese asthmatics and controls. Several studies have shown that ERV increases after weight loss, adopting a calorie-restricted diet, or bariatric surgery. Hakala et al. [89] found a considerable increase in the ERV of patients whose BMI decreased from 45 to 39 kg/m^2 after adopting a calorie-restricted diet. Weight loss also causes changes in other parameters, including functional residual capacity, total lung capacity, and gas exchange, resulting in increased blood oxygenation.[89] Babb et al. showed that even modest reductions in weight, i.e., a decrease in BMI from 35 to 33 kg/m^2, induce an increase in end-expiratory lung volume during submaximal exercise.[90]

Addressing obesity's complex psychosocial implications requires behavioral therapies. Cognitive behavioral therapy (CBT) for obesity is a treatment modality that combines the traditional procedures of standard behavioral therapy for obesity with a set of specific cognitive strategies and procedures. It is aimed at not only losing weight but also preventing weight regain, thereby avoiding the dissatisfactory long-term results of earlier behavioral treatments.[91,92] These behavioral techniques are enhanced by goal setting, self-monitoring, and stress- and emotion-management strategies.[93,94]

Regarding the appropriate threshold, previous behavioral weight-loss studies often reported 5% weight loss as a clinically significant threshold.[95-98] In a study by Dalle Grave,[99] 67 adult patients with obesity (BMI $\geq$ 30 kg/m^2) were recruited from consecutive referrals to an Italian National Health Service obesity clinic. Each patient was offered 22 group cognitive behavioral therapy sessions (14 in the 6-month weight-loss phase and 8 in the subsequent 12-month weight-maintenance phase). Weight loss of 11.5% after 6 months and 9.9% after 18 months of CBT was associated with a significant reduction in cardiovascular risk factors, anxiety, depression, eating disorder psychopathology, and an improvement in obesity-related quality of life.

Technological advances have extended non-pharmacological obesity treatments. Self-monitoring, goal setting, and progress tracking are possible via mobile apps, wearables, and internet platforms. This tech-driven strategy improves responsibility, gives real-time feedback, and empowers diet and exercise choices.[100]

Non-pharmacological therapies need long-term maintenance. Long-term success goes beyond weight reduction. Relapse prevention, behavioral support, and frequent evaluation and revisions of the intervention plan are essential for enduring improvements and decreasing obesity-related comorbidities.[101,102]

Conclusion

This narrative review illuminates the complex links between obesity, pulmonary function, and muscle strength. The multifarious nature of obesity causes complicated physiological alterations that affect the whole body. Excess belly fat tissue hinders lung expansion and strains respiratory muscles. Obesity and muscle strength are linked by metabolic dysfunction, muscle composition changes, and lifestyle variables. Obesity has far-reaching health effects, including

reduced respiratory functioning and muscle strength. Obesity's loss in pulmonary function and muscle strength highlights the necessity for multimodal intervention and therapy. This strategy should include doctors, nutritionists, physical therapists, and psychologists working together to address the behavioral and psychological causes of obesity.

Competing Interests

The authors declare that they have no competing interests.

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