

Article

Obesity and Respiratory Diseases: Pathophysiological Mechanisms and Clinical Implications—A Review

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Abstract: Obesity constitutes a significant health global issue; it has been associated with a rise in chronic diseases such as respiratory diseases. Excessive adipose tissue accumulation can affect the lung function in a variety of ways including mechanical, inflammatory, metabolic and hormonal. The greater thoracic and abdominal fat volume may lead to a decrease in lung volume, limitation of diaphragmatic function, hyperwork of breathing and exercise intolerance. Moreover, adipose tissue is an active endocrine and immune organ, and releases adipokines and inflammatory mediators which may affect airway function and respiratory control. These mechanisms have been linked to a number of important respiratory diseases, including obstructive sleep apnea syndrome, asthma, obesity hypoventilation syndrome and respiratory dysfunction in chronic obstructive pulmonary disease. Obesity can also make the diagnosis of respiratory disease difficult, as symptoms like dyspnea, fatigue and exercise intolerance can either be due to the obesity or be caused by other pulmonary disease. This review discusses the key pathophysiological mechanisms linking obesity to respiratory dysfunction and its link to common respiratory diseases. It also covers clinical evaluation, weight-management algorithms, pharmacological management, multi-disciplinary management and novel treatments. Further knowledge of the complex interplay of obesity and respiratory health is needed to aid early diagnosis, personalized treatment, prevention of complications, and clinical outcomes.

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1. Introduction

Obesity is becoming one of the most serious non-communicable diseases in the world, and is a key risk factor for many metabolic, cardiovascular and respiratory diseases. It is defined as an excess of adipose tissue due to a complex interaction of genetic, environmental, behavioral and metabolic factors. While BMI is traditionally used to assess obesity, the distribution and biological activities of adipose tissue are also important determinants of disease. Especially, high levels of visceral and thoracic fat in particular have profound effects on respiratory physiology and overall pulmonary function [1].

There are several interconnected pathways relating obesity to respiratory health. Gaining weight affects the mechanics of the chest-wall and diaphragm, lung volumes, work of breathing, and exercise capacity. Furthermore, adipose tissue is an endocrine and immunological organ which can secrete inflammatory mediators and hormones that can affect airway function and control. These mechanisms have been associated with higher prevalence and severity of several respiratory diseases, such as obesity hypoventilation syndrome, functional limitations due to chronic obstructive pulmonary disease (COPD) and obstructive sleep apnea (OSA) [2] (Figure 1).

A pathophysiological understanding of obesity and respiratory disease is critical to correct diagnosis, management and prevention of complications. The review will provide

an overview of the most important mechanisms involved in the relationship between obesity and respiratory dysfunction, explore the relationship of obesity to important respiratory disease, and discuss the clinical management and future therapeutic prospects.

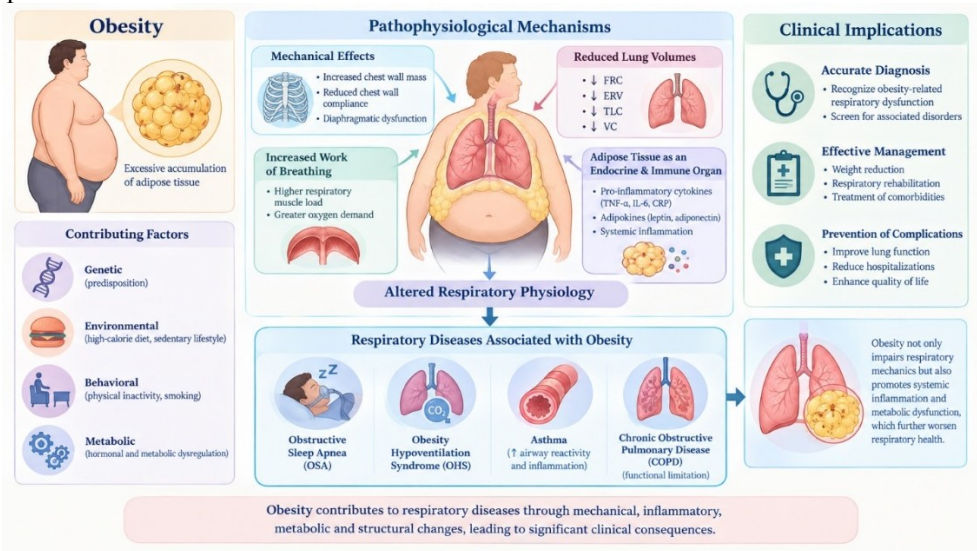


Figure 1. Obesity-Related Respiratory Dysfunction: Mechanisms and Clinical Implications [2].

2. Materials and Methods

2.1 Obesity and Respiratory

2.1.1 Global Prevalence and Epidemiology of Obesity

Obesity defined as a chronic and multifactorial disease with excessive accumulation of fat, which can negatively impact physiological function and health. It has become widely prevalent in the entire world and has become a major public health problem. Obesity is caused by a combination of genetic predisposition, dietary habits, lack of exercise, sedentary lifestyles, socioeconomic status, environmental factors and lifestyle changes. BMI is a widely used obesity category, but doesn't adequately capture body-fat distribution or metabolic health. Similarly, visceral and abdominal fat may be more closely linked with systemic complications than total body-weight [3,4].

Obesity has become a widespread public health problem, resulting in a significant burden of metabolic, cardiovascular, musculoskeletal and respiratory diseases. Respiratory complications are especially significant since a high amount of excess adipose tissue may change breathing mechanics and worsen the risk for various respiratory and sleep disorders. Obesity may affect children of all ages and the high proportion of children who are obese is a concern for future respiratory problems [5].

Epidemiology is important from a public health point of view because the rising prevalence of obesity will drive increased needs for prevention, diagnosis and treatment of obesity-related diseases. Thus, obesity is not a metabolic disease but it is also a significant factor to take into account in the field of respiratory health [6].

2.1.2 Impact of Obesity on Respiratory Health

Obesity can significantly impact respiratory health, both mechanically and physiologically, as well as via inflammatory and metabolic pathways. An increased mechanical load is placed on the respiratory system due to excess adipose tissue around the chest and abdomen. Thoracic fat can decrease the compliance of the chest wall, and abdominal fat can cause the diaphragm to be pushed up. This results in an increased work of breathing to keep their ventilation up to normal. These changes can lead to a decrease in functional residual capacity and expiratory reserve volume and can lead to shortness of breath, especially when exercising [7] (figure 2).

Respiratory muscle function can also be impaired by obesity. Respiratory muscle have to exert more force to offset the additional mechanical resistance of excess body weight. The greater oxygen demand and impaired mechanics of the respiratory system during

exercise can lead to early fatigue and decreased exercise tolerance. Besides mechanical effects, fat can also metabolically act, generating hormones and inflammatory mediators which may affect respiratory tissues. The chronic, low-grade inflammation that is seen with obesity can alter airway function and may play a role in respiratory symptoms. Obesity is also linked closely with sleep-related breathing disorders in particular, obstructive sleep apnea (OSA) and in severe cases hypoventilation [8].

So, the pulmonary effects of obesity are not merely the result of decreased lung volumes. They encompass several interacting mechanisms which may affect breathing, exercise capacity, sleep quality and susceptibility to respiratory disease [9].

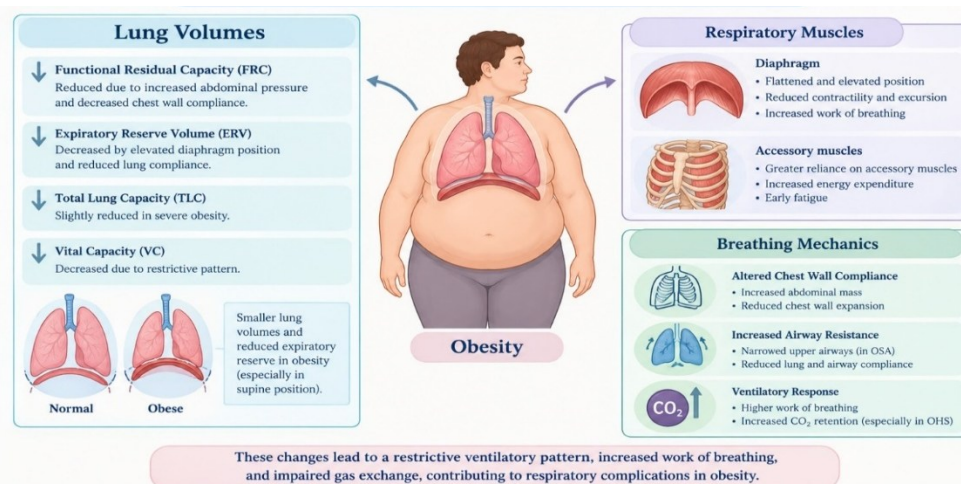


Figure 2. Effects of obesity on lung volumes, respiratory muscles, and breathing mechanics [7].

2.1.3 Association Between Obesity and Respiratory Diseases

There are a number of respiratory disease traits associated with obesity, but the associations and mechanisms differ among people. The strongest association is with OSA, in which a surplus of fat surrounding the upper airway causes upper airway narrowing, and upper airway collapsibility during sleep. Asthma is also tightly linked with obesity, but the link applies to other clinical phenotypes and might be mediated by mechanical, inflammatory and metabolic mechanisms [10].

Severe obesity may result in decreased ventilation during wakefulness (obesity hypoventilation syndrome). This can happen in the presence or absence of OSA, and can lead to important cardiovascular and respiratory sequelae. COPD is also often present in persons with obesity. These patients may experience greater work of breathing and decreased exercise tolerance due to excess body weight, but the impact of excess weight varies with the severity of the disease and body composition [11].

Obesity and respiratory disease often co-exist and bring diagnostic issues as some symptoms (e.g., dyspnea, fatigue, shortness of breath, decreased exercise capacity) can be related to a number of causes. Respiratory symptoms should not be taken as a direct consequence of obesity. A detailed clinical evaluation is needed to diagnose individual respiratory diseases and to assess the role of obesity in the severity of these diseases. It is important to recognize these relationships and come up with prevention and management methods accordingly [12].

3. Results

3.1. Pathophysiological Mechanisms Linking Obesity to Respiratory Dysfunction

3.1.1 Effects of Obesity on Respiratory Mechanics and Lung Function

Obesity causes substantial alterations in the mechanical characteristics of the respiratory system. Fat deposits in the chest and abdomen lead to an increase in outside pressure against the chest wall and diaphragm. Fat in the abdomen can push up on the diaphragm, making less room for the lungs to expand. Concurrently, thoracic adiposity

could reduce the compliance of the chest wall and increase resistance to movement of the chest wall [13].

A consistent finding in obesity is a decrease in FRC and ERV. These changes tend to be more marked the more obese a person gets and can be more prominent in people who have central obesity. Small airways are prone to collapse during expiration if the volume of the lungs is reduced, which may contribute to ventilation heterogeneity and to breathlessness. The mechanical loading also causes extra work by the respiratory muscles. The same applies during exercise, where the ventilatory load is increased as more body mass moves at a higher rate of speed. This can lead to greater respiratory effort and early exercise limitation in patients with obesity [14] (figure 3).

Importantly, not all people who are obese develop the same pulmonary-function abnormalities. The effects will vary depending on the amount and location of adiposity, age, physical fitness and the presence of underlying lung disease. These mechanical changes offer a basic account of the respiratory changes that are frequently found in obesity [15].

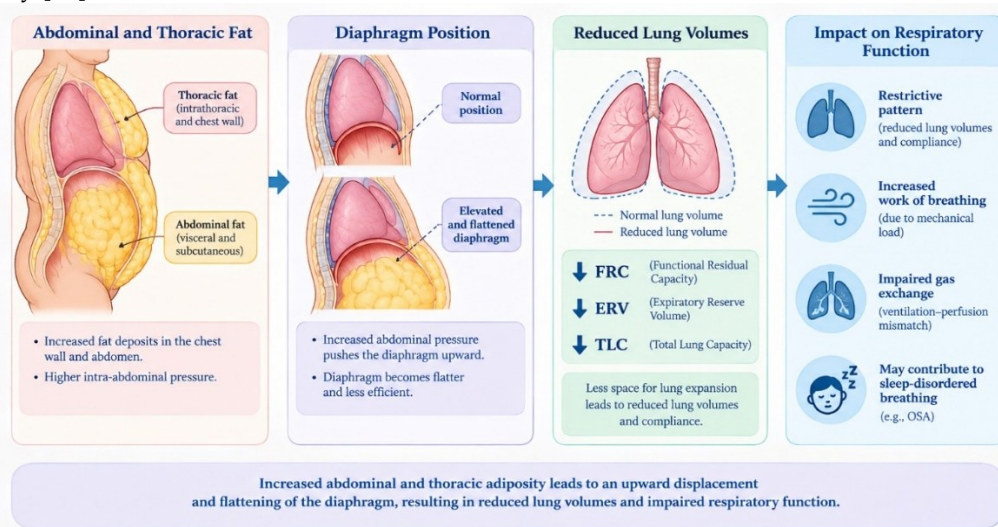


Figure 3. Mechanism showing abdominal and thoracic fat affecting diaphragm position and lung volumes [14].

3.1.2 Chronic Inflammation and Adipose Tissue-Derived Mediators

Adipose tissue is not just an energy storage organ, but also an active endocrine and immune organ. In obesity, the enlargement and dysfunction of adipocytes may lead to changes in adipokine and inflammatory mediator production. This leads to low grade but ongoing systemic inflammation which may affect various organs of the body such as the respiratory system [16].

Leptin, adiponectin and some pro-inflammatory cytokines are the important mediators derived from adipose tissue. Leptin is generally correlated with adiposity, and plays a crucial role in the regulation of immune system, while adiponectin has anti-inflammatory and metabolic properties and is inversely related to adiposity. This imbalance between these mediators could play a role in the changes in immune responses and inflammatory activity of the airways. The effects of chronic inflammation can have an impact on airway epithelial function, smooth-muscle activity and recruitment of immune cells. These processes are germane to some asthma related to obesity phenotypes, where inflammatory pathways may be different from those in the conventional allergic asthma. The inflammation can also modulate oxidative stress and metabolic abnormalities, thereby interacting with and impacting respiratory tissues (Figure 4) [17].

Therefore, a simple compression of the lungs is not sufficient to explain the respiratory dysfunction of obesity. Adipose tissue is a tissue with active regulation capacity on systemic and pulmonary inflammatory pathway. This biological activity is an important mechanism linking excess adiposity to respiratory disease, and accounts for the benefit of weight loss to respiratory health beyond its mechanical unloading effects [18].

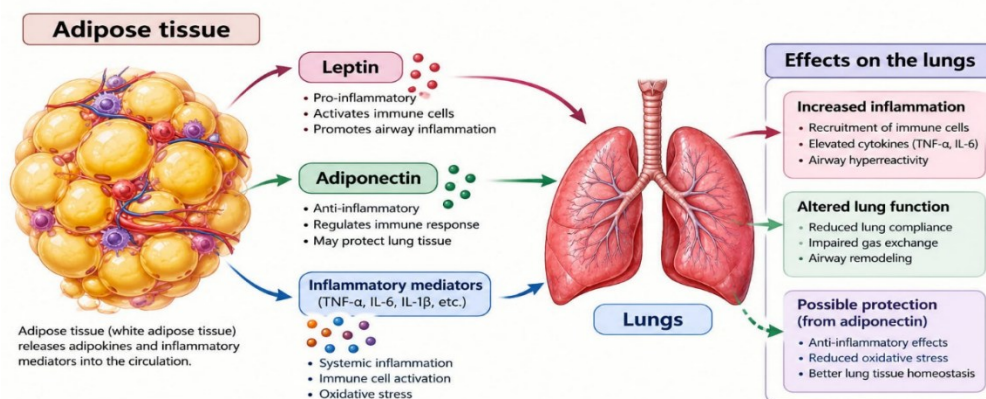


Figure 4. Adipose tissue showing leptin, adiponectin, and inflammatory mediators affecting the lungs [17].

3.1.3 Metabolic and Hormonal Mechanisms Affecting Respiratory Function

The obese have metabolic abnormalities that are not uncommon including insulin resistance, altered glucose regulation, dyslipidemia, and alteration of hormones. These disruptions can be connected to the inflammatory process, airway responsiveness, cell metabolism and respiratory control mechanisms, and therefore impact respiratory function. Insulin resistance is particularly noteworthy because it is associated with metabolic dysfunction in the whole body and perhaps with tissue inflammation of the airways [19].

Adipose tissue also produces hormones that are important for the physiology of respiration. Leptin is involved in the regulation of energy and immune functions, and adiponectin is involved in metabolic function and has anti-inflammatory properties. A change in the levels and activity of these hormones might impact airway inflammation and responsiveness. These metabolic mechanisms could be especially important for obesity-associated asthma and other respiratory diseases with metabolic dysfunction (Figure 5) [20].

In severe obesity, the control of ventilation can also be affected, especially during sleep. The extra strain of the respiratory system, together with changes in regulating metabolism and hormones could compromise the ability to maintain appropriate ventilation. In susceptible individuals, it can result in a chronic reduction in ventilation and CO₂ retention. So the metabolic system and the respiratory system are bi-directional. Impaired respiratory function will result in reduced physical activity, which will result in a further metabolic dysfunction, and impaired metabolic function will result in a worsening of the respiratory function. It is essential to take into account these interactions when developing integrated strategies to tackle obesity and respiratory dysfunction [21].

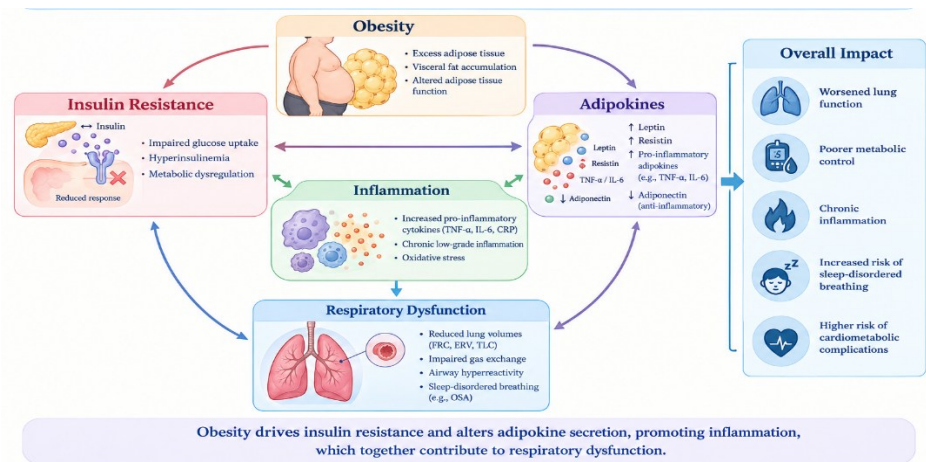


Figure 5. Interaction between obesity, insulin resistance, adipokines, inflammation, and respiratory dysfunction [20].

3.2 Obesity and Major Respiratory Diseases

3.2.1 Obesity and Obstructive Sleep Apnea

Obesity is associated with one of the most clinically significant respiratory diseases, namely, obstructive sleep apnea. It involves recurrent episodes of upper-airway obstruction, either partial or complete, during sleep, causing an interruption of breathing, intermittent oxygen desaturations and repeated sleep arousals. Both anatomical and physiological mechanisms are responsible for obesity's role in this disorder [22].

Gingivoglottopathy (GOG) is a condition where excess adipose tissue around the upper airway and neck results in reduced airway diameter and increased airway collapse during sleep. Gingivoglottopathy (GOG) is a condition where there is an excess of fat around the upper airway and neck which leads to decreased airway diameter and increased airway collapse during sleep. Additionally, obesity-induced lung volume loss could impair the downward pull of upper airway muscles that keeps the airway open. In some people, the changes in respiratory muscle function and ventilatory control may make them even more susceptible. Those who suffer from obstructive sleep apnea often complain of loud snoring, breathing pauses that they are aware of, headaches in the morning, excessive daytime sleepiness and poor concentration. Hypoxemia and sleep fragmentation may also lead to cardiovascular and metabolic complications, which can occur at multiple times throughout episodes of OSA [23] (Figure 6).

Loss of weight can help with OSA by reducing upper-airway adiposity and enhance respiration mechanics. But it is not only obesity that determines disease and the anatomical structure and neural control and ventilatory stability play a role in susceptibility. Thus, proper diagnosis and disease specific management are still crucial. Obesity and OSA have been linked and show how excess adiposity can contribute to respiratory physiology in sleep, and the need to be aware of sleep-related breathing disorders in an obese patient [24].

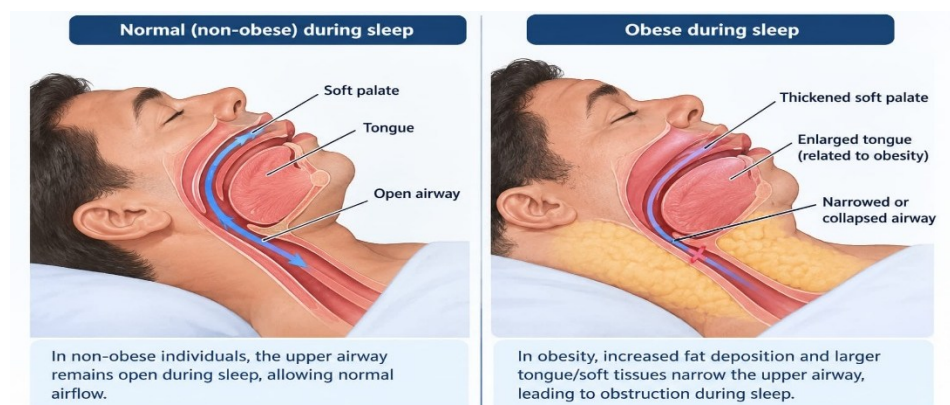


Figure 6. Upper-airway obstruction during sleep in obesity [23].

3.2.2 Obesity and Asthma

There is a complex relationship between obesity and asthma that includes mechanical, inflammatory and metabolic processes. Obesity is a risk factor for asthma and can exacerbate symptoms and control of asthma in people with established asthma. Obesity-associated asthma is, however, a heterogeneous condition, and individuals might have varying physiological and inflammatory features [25].

Because of the reduced lung volumes resulting from obesity, airway mechanics will be affected and airways will be more prone to narrowing. Further, adipose tissue secretes inflammatory mediators which can affect airway epithelial function as well as immune responses and smooth-muscular activity. Airway dysfunction may develop through mechanisms that involve metabolic abnormalities that include insulin resistance. Some people who are obese develop asthma later in life and may have a non-classical inflammatory profile, other people may have allergic asthma, which becomes harder to manage when they gain weight. This separation has clinical significance, since the clinical phenotype can affect the response to therapy (Figure 7) [26].

Obesity can also make it difficult to diagnose asthma as symptoms like dyspnea, wheezing and exercise intolerance could be due to the obesity rather than to active airway disease. Therefore, objective assessment is important when assessing the role of asthma in respiratory symptoms. In people who are overweight, weight loss can help control asthma, increase exercise tolerance, and reduce asthma-related symptoms. However, weight control needs to be done along with a suitable asthma treatment and not as a substitute of the proven medical treatment [27].

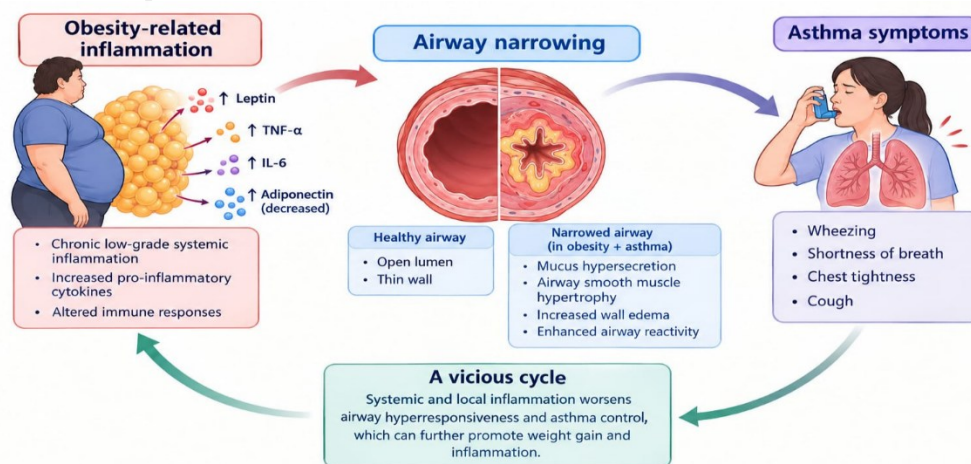


Figure 7. Interaction between obesity-related inflammation, airway narrowing, and asthma symptoms [26].

3.3 Obesity and Chronic Obstructive Pulmonary Disease

COPD often exists along with obesity and its clinical features can be altered by obesity. COPD is the main condition of chronic airflow limitation and respiratory symptoms, and obesity adds to the mechanical and metabolic loads. This combination of conditions is thus clinically complicated. The greater the body mass, the more energy is needed for moving and breathing. When COPD patients have to work harder to breathe, they may have already limited ventilatory reserve, exacerbating dyspnea and making it more difficult to exercise. Abdominal and thoracic adiposity may also affect the respiratory mechanics of the diaphragm, which may make breathing more difficult [28] (Figure 8).

Adverse effects of obesity vary with degree of COPD and body composition. Advanced COPD sufferers may lose a substantial amount of weight and experience skeletal-muscle wasting while others may be overweight or obese. Thus, BMI cannot be used as a sole measure of the functional status of these patients. Assessment of muscle mass, performance in daily activities, nutrition and exercise capacity is also relevant [29].

Obesity could also play a role in systemic inflammation and metabolic disturbance, which can modulate inflammation contributing to COPD. Individualized management is

therefore needed and can consist of smoking cessation, pulmonary rehabilitation, pharmacotherapy, exercise, and nutrition or weight-management. The co-existence of obesity and COPD is of importance as treating airflow limitation alone may not be sufficient to treat dyspnea and exercise limitation. Optimizing respiratory and functional outcomes will need a comprehensive approach [30].

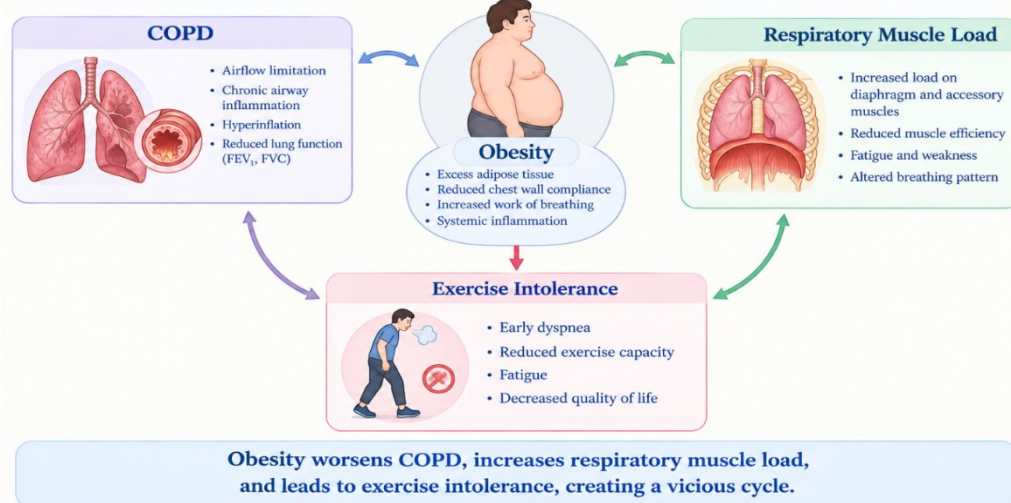


Figure 8. Interaction between obesity, COPD, respiratory muscle load, and exercise intolerance [28].

4. Discussion

4.1 Clinical Implications and Management of Obesity-Related Respiratory Disorders

4.1.1 Clinical Assessment and Diagnosis

When evaluating a patient's respiratory function who is obese, it is important to have a systematic approach as similar symptoms can be caused by different conditions. Obesity can cause dyspnea, fatigue, decreased exercise tolerance, snoring, and poor sleep, or these symptoms can be signs of asthma, COPD, obstructive sleep apnea, obesity hypoventilation syndrome, and cardiovascular disease. The clinical evaluation should start with a thorough history that asks about the onset and nature of respiratory symptoms, smoking, exercise, sleep disturbance, snoring, witnessed apneas, daytime sleepiness, medications and any previous respiratory diagnosis. Assessment of body habitus, respiratory rate, oxygen saturation, blood pressure and cardiopulmonary disease should be performed during physical examination [31].

PFTs can be used to help detect ventilatory abnormalities, such as airflow obstruction and altered lung volume. If sleep disordered breathing is suspected, then proper sleep assessment should be considered. Evaluation of carbon dioxide level is especially important in patients with severe obesity and suspected hypoventilation. BMI is a helpful tool for informing about body size, and is not a complete measure of the respiratory risk associated with obesity. Other clinical data such as Waist circumference, Body-fat distribution, Muscle mass and Functional capacity can give more information. The diagnosis is critical since each type of respiratory illness needs a specific treatment. A systematic assessment will avoid the misdiagnosis of symptoms to obesity and allow early detection of treatable respiratory complications [32].

4.1.2 Weight Management and Lifestyle Interventions

Obesity-related respiratory diseases require weight management as a crucial part of their treatment. Mechanically, excess adipose tissue causes respiratory dysfunction by loading the lungs, decreasing lung volumes, causing systemic inflammation, and causing metabolic abnormalities. This means that a long-term decrease in weight can help with breathing in some patients and decrease disease burden [33].

Most lifestyle interventions will involve diet, exercise, behavioral support and long-term monitoring. Emphasis should be placed on sustainable approaches to energy intake and adequate nutrition rather than short-term restrictive approaches. Medical conditions,

cultural preferences, socioeconomic factors and ability to sustain long-term adherence should all be taken into account when creating an individual plan. Physical activity is especially significant because it helps to increase cardiovascular fitness, muscle strength and functional capacity. But severe obesity or respiratory disease may mean that patients have low exercise tolerance. Exercise programs should therefore be tailored to each participant and build-up slowly. Pulmonary rehabilitation concepts could be beneficial to patients with severe respiratory restrictions [34].

Losing weight can have advantages for a number of respiratory diseases. It can enhance respiratory mechanics and exercise capacity and can also decrease the severity of sleep-related breathing disorders. Weight loss also can help control asthma symptoms in certain patients who also have obesity. It is important to note that the use of weight management should not be a substitute for the disease-specific respiratory therapy. Rather, it must be part of a holistic treatment plan. Ongoing assessment of body weight, respiratory symptoms, physical function and disease control can also be used to assess whether the intervention is having significant clinical effect [35].

4.1.3 Pharmacological and Multidisciplinary Management

Both specific obesity and specific respiratory management may be required for the treatment of obesity-related respiratory disorders. The best course of action is dependent on the type of respiratory disorder, its severity, presence of other medical conditions, and patient factors. Anti-inflammatory and bronchodilator medication (inhaled) may be necessary in asthma patients, while COPD patients may need bronchodilator therapy, pulmonary rehabilitation, and other supportive therapies. People who suffer from OSA might be able to benefit from positive airway pressure therapy in order to avoid upper-airway collapse during sleep. If a patient is obese with hypoventilation syndrome, they might need to receive a ventilatory support device as well as being closely monitored [36].

The use of medications to treat obesity is becoming an increasingly important therapeutic option in selected patients. Anti-obesity drugs can lead to a sustained weight loss and can diminish mechanical and metabolic factors in respiratory illness. However, therapy has to be individualized and monitored for efficacy, tolerability and adherence to treatment. Metabolic surgery and/or bariatric surgery may be indicated when clinically appropriate for severe obesity. Weight loss can have a significant effect on the mechanics of respirations and could help lessen the effects of obesity on respiratory disease [37].

A multidisciplinary management is especially beneficial as obesity frequently co-occurs with metabolic and cardiovascular disease. Multidisciplinary care is possible between respiratory physicians, sleep specialists, obesity specialists, dietitians, physiotherapists and primary-care physicians. This holistic treatment takes care of breathing function, weight, food consumption, physical activity, sleep and other comorbidities at the same time [38].

5. Conclusion

Obesity is an important chronic disease that has significant impact on respiratory disease, via multiple mechanically, inflammatory, metabolic and hormonal mechanisms. When excess fat is present, it can cause a decrease in lung volumes, an increase in work of breathing, loss of efficiency of respiratory muscles and a decrease in physical performance. Simultaneously, fat tissue is responsible for chronic low-grade inflammation and metabolic disturbances that could also contribute to the dysfunction of these airways and the regulation of breathing.

The clinical association of obesity and respiratory disease is especially marked in the following areas: obstructive sleep apnea, asthma, obesity hypoventilation syndrome, and the functional effects of COPD. Careful clinical evaluation is important to differentiate between the various mechanisms that can contribute to respiratory symptoms, and more targeted interventions for managing respiratory complications may be possible with obesity. In conclusion, acknowledging obesity as a significant risk factor for respiratory health will help in early detection, holistic management, and achieving better long-term results. Therefore, it is necessary to adjust restated physiological changes in relation to specific respiratory diseases in the integration of respiratory and obesity management.

The best way to manage is to use an integrated approach to treat excess body weight and the underlying respiratory disease. Depending on the clinical characteristics of individuals, lifestyle changes, physical activity, medical and pharmacological therapy, respiratory therapy, or multidisciplinary treatment may all help to achieve better results.

Personalized medicine and novel methods specifically aimed at obesity could offer more targeted remedies to the respiratory complications of obesity in the future. In conclusion, addressing obesity as a significant factor in respiratory health can help identify it early, manage it effectively, and ultimately enhance long-term patient outcomes. Respiratory and obesity management should therefore continue to be a part of contemporary clinical practice.

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