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# CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND OBESITY: CLINICAL AND SOCIAL ASPECTS

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**Abstract.**

The relationship between chronic obstructive pulmonary disease (COPD) and obesity represents a complex clinical challenge. Obesity exacerbates the course of COPD by reducing quality of life and increasing the risk of cardiovascular complications, while paradoxically demonstrating improved survival among overweight patients, known as the "obesity paradox." The aim of this review is to analyze current evidence on the impact of obesity on the progression and prognosis of COPD. The scientific novelty of this work lies in the systematization of conflicting data and the identification of key directions for future research.

Keywords: COPD, obesity, obesity paradox, lung function, prognosis

**Introduction.**Chronic obstructive pulmonary disease (COPD) remains one of the leading causes of morbidity and mortality worldwide. In addition to traditional risk factors such as smoking and air pollution, increasing attention is being paid to metabolic disorders, particularly obesity [1]. Recent studies show that nearly 50% of COPD patients have a body mass index (BMI) above the normal range [2]. Against the backdrop of the global obesity epidemic, understanding its influence on COPD progression is of growing importance.

The coexistence of chronic obstructive pulmonary disease (COPD) and obesity presents a unique pathophysiological challenge due to the complex interaction of respiratory, metabolic, and inflammatory mechanisms. Obesity is not merely a mechanical burden on respiration; it significantly alters systemic inflammation, immune responses, and metabolic regulation, which in turn aggravate the course of COPD (1).

One of the key pathogenetic mechanisms is chronic low-grade systemic inflammation. Adipose tissue, especially visceral fat, acts as an endocrine organ and secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and leptin. These mediators enhance systemic and pulmonary inflammation, contributing to airway remodeling and alveolar destruction characteristic of COPD (2).

Moreover, adipokines such as leptin and adiponectin play opposing roles. Leptin, typically elevated in obese individuals, has pro-inflammatory effects and may exacerbate airway inflammation. In contrast, adiponectin, which has anti-inflammatory properties, is often decreased in obesity, further aggravating the inflammatory status (3).

Oxidative stress is another important link. In obese patients with COPD, oxidative stress is amplified due to both systemic metabolic dysregulation and chronic pulmonary inflammation. This leads to damage of airway epithelium and decreased antioxidant defense mechanisms (4).

Additionally, obesity leads to ventilatory dysfunction. Excess fat in the thoracic and abdominal regions reduces lung volumes (e.g., functional residual capacity and expiratory reserve volume), increases airway resistance, and impairs gas exchange. This mechanical limitation, combined with hyperinflation and decreased diaphragmatic mobility, worsens dyspnea and exercise tolerance in COPD patients (5).

Insulin resistance, commonly seen in obese individuals, may also influence COPD progression by affecting mitochondrial function, promoting further oxidative stress, and altering the energy metabolism of respiratory muscles (6).

The obesity paradox in COPD. Despite the expected negative effects, multiple studies have identified the “obesity paradox” — improved survival among COPD patients with higher BMI [5]. The mechanisms underlying this phenomenon are not fully understood. It is hypothesized that fat reserves and the anti-inflammatory properties of adipose tissue may play protective roles during terminal stages of disease [6].

Risks and comorbid conditions. Obesity in COPD patients is associated with an increased risk of cardiovascular disease, type 2 diabetes, and COPD exacerbations [7]. Overweight status worsens exercise tolerance, decreases quality of life, and increases healthcare utilization [8].

Specifics of therapy for COPD patients with obesity. Rehabilitation programs for COPD patients should consider the presence of obesity. Physical exercise training and nutritional support help to improve exercise tolerance and optimize body weight [9]. Pharmacokinetic considerations must also be taken into account when prescribing medications.

Finally, gut-lung axis dysregulation, driven by obesity-related changes in microbiota, has been suggested as a contributing factor. Altered gut permeability and microbiome composition may lead to systemic endotoxemia and immune dysregulation, further fueling pulmonary inflammation (7).

In conclusion, the comorbid state of COPD and obesity represents a synergistic interaction of inflammatory, metabolic, and mechanical factors. Understanding these mechanisms is essential for developing personalized therapeutic strategies aimed at improving outcomes in this growing patient population (8).

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