Pan-African Journal of Health And Psychological Sciences
ISSN: 3093-4737 | www.pajhps.org
Vol.2, Issue 2 | April-June 2026

Metabolic Syndrome in Lung Disease Patient and Its Impact on Other Organs

Author(s): [1]Firoz Khan, [1]Shafkat Hussain Malik, [1]Taranvirinderdeep Singh, [2]Ruhit Ashraf*, [1]Imtiyaz Hussain [3]Dakush Mahajan.

[1] Assistant Professor, School of Pharmacy, Desh Bhagat University, Mandi Gobindgarh, Punjab, India.
[2]Associate Professor, S. Lal Singh Memorial College of Pharmacy, Desh Bhagat University, Mandi Gobindgarh, Punjab, India.
[3]B. Pharmacy (Student), School of Pharmacy, Desh Bhagat University, Mandi Gobindgarh, Punjab, India.

Corresponding Author:

Ruhit Ashraf
Associate Professor
Email: ruwaidashraf1819@gmail.com

Abstract:

Insulin resistance, obesity, dyslipidemia, and hypertension are among the conditions that make up metabolic syndrome, a complex disorder that significantly affects patients with lung conditions like asthma, interstitial lung disease, and chronic obstructive pulmonary disease. These conditions also increase the risk of cardiovascular disease and type-2 diabetes mellitus. The pathophysiology of metabolic syndrome is examined in this review, with particular attention paid to the roles played by insulin resistance, chronic inflammation, the intricate interactions between lung illnesses and metabolic syndrome, systemic inflammation, oxidative stress, and immune system activation. Chronic renal disease, nonalcoholic fatty liver disease, cardiovascular disease, and cognitive decline are among the multi-organ consequences that are exacerbated by these conditions, which also contribute to pulmonary symptoms. Clinical ramifications are examined, emphasizing the elevated risks of frailty, cognitive decline, and cardiovascular illness, especially in older persons and particular gender groupings. To emphasize the significance of early intervention, management methods are examined, including pharmaceutical treatments, lifestyle modifications, and innovative therapeutic approaches like telmisartan and sacubitril/valsartan. This study also emphasizes the value of a multidisciplinary approach to treatment, combining pharmaceutical treatments (statins, GLP-1 agonists), lifestyle changes, and

How to cite this article: Khan, F. et al. (2026). **Metabolic Syndrome in Lung Disease Patient and Its Impact on Other Organs. Pan-African Journal of Health and Psychological Sciences. Vol 2, Issue 2. April -June 2026. https://doi.org/10.64261/6x43w534.**

ISSN: 3093-4737

new treatments (endolytic, gut-lung axis regulation). In order to improve patient outcomes and lower morbidity and mortality, the report ends with a call for targeted medicines to address the underlying processes of Metabolic Syndrome in high-risk populations. These efforts target pulmonary components.

Keywords:
Metabolic syndrome, lung diseases, systemic inflammation, oxidative stress, Hypertension, Diabetes, Cardiovascular Disease.

1. Introduction

Metabolic syndrome (MetS) is a cluster of conditions, including abdominal obesity, insulin resistance, hypertension, and dyslipidemia, that increase the risk of cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM)[1] MetS is not a single disease but a collection of risk factors that vary in definition across different organizations. Recent studies have highlighted the role of MetS in lung diseases, particularly chronic obstructive pulmonary disease (COPD), asthma, and interstitial lung disease (ILD), where it exacerbates symptoms and increases morbidity and mortality [2].

Pathology of lung disease are driven by infections, pollutants, or temperature changes, activating neutrophilic or eosinophilic inflammation. Severe cases cause impaired gas exchange due to ventilation-perfusion mismatch from airway inflammation, mucus and bronchoconstriction. This cause hypoxia, respiratory acidosis and leads to respiratory failure and death[3, 4] Metabolic syndrome is strongly linked to impaired lung function, core features of metabolic syndrome are central obesity, insulin resistance and systemic inflammation, all that plays a significant role in the lowering of forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) [5, 6]. As the number of metabolic components rises, respiratory function tends to decline, suggesting an association between metabolic syndrome and lung disease [5].

The interplay between MetS and lung diseases is complex, involving systemic inflammation, oxidative stress, and insulin resistance, which collectively contribute to the progression of both pulmonary and extra-pulmonary complications. This review explores the pathophysiology of MetS in lung disease patients, its impact on other organs, and the clinical implications for management and treatment [7].

2. Linkage of Metabolic Syndrome in Lung Disease

Lung dysfunction is caused by metabolic syndrome through systemic mechanisms that include oxidative stress and inflammation. It causes oxidative damage and the release of pro-inflammatory cytokines, which results in endothelial dysfunction and vascular remodeling[8].

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Beyond metabolic tissues, these processes impact lung structures and aid in the development of conditions like asthma, COPD, and ILD. With an emphasis on its effects on lung health[9], the figure demonstrates how metabolic syndrome serves as a primary cause of multi-organ failure (Figure 1) and in the context of obesity, dyslipidemia, and diabetes mellitus, this (Figure 2) illustrates the intricate relationship among metabolic abnormalities, chronic inflammation, and hormonal imbalances. Increased leptin and decreased adiponectin are signs of adipokine imbalance, which intensifies immunological activation and inflammatory signaling in the lungs. These elements intensify oxidative stress and fibrotic alterations in lung tissue when combined with hyperglycemia and lipid disorders[10]. The graphic highlights the role that hormonal and metabolic abnormalities play in lung dysfunction in diseases associated with obesity.

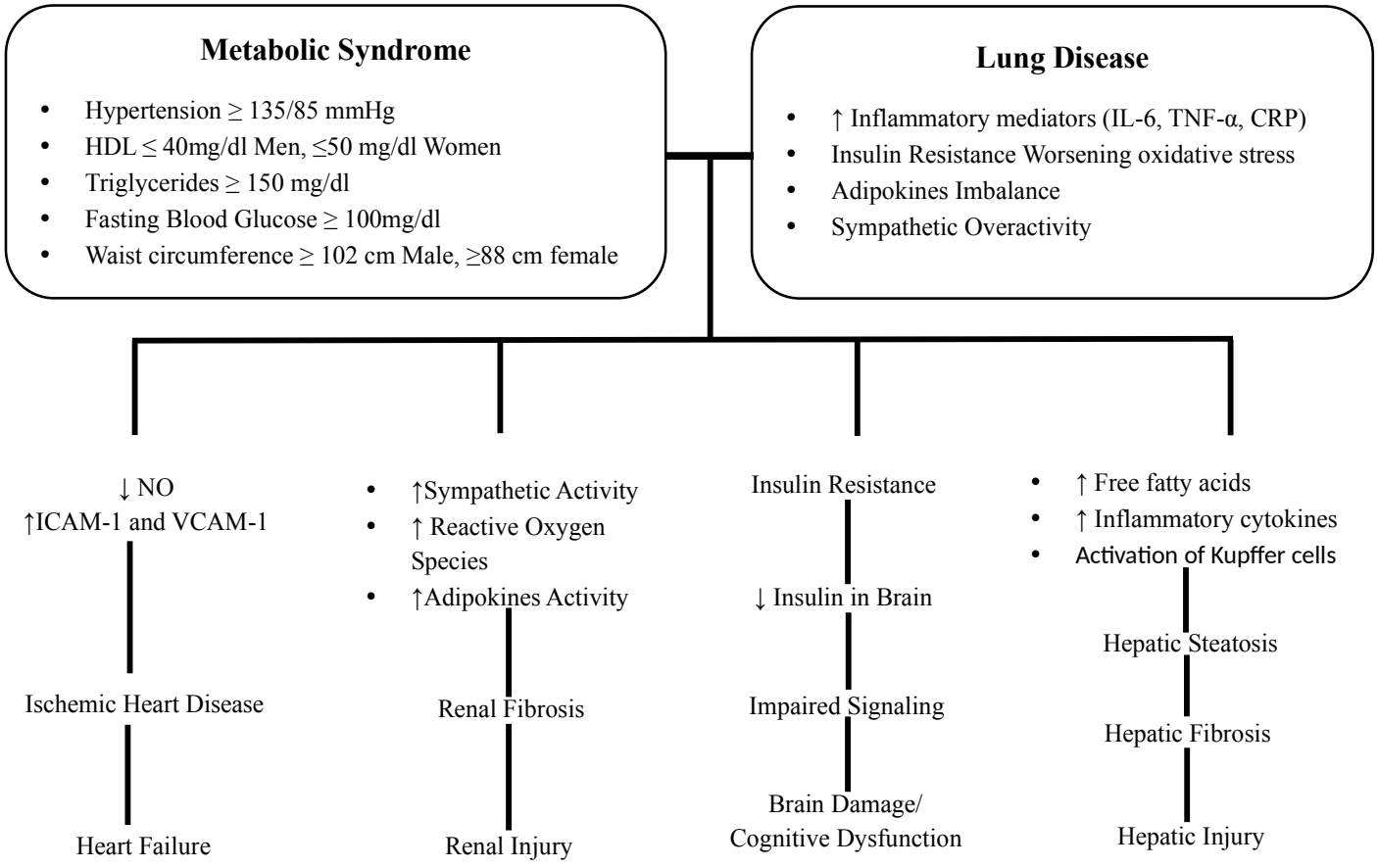

Figure 1: Impact of Metabolic Syndrome on Other Organs Associated Lung Disease.

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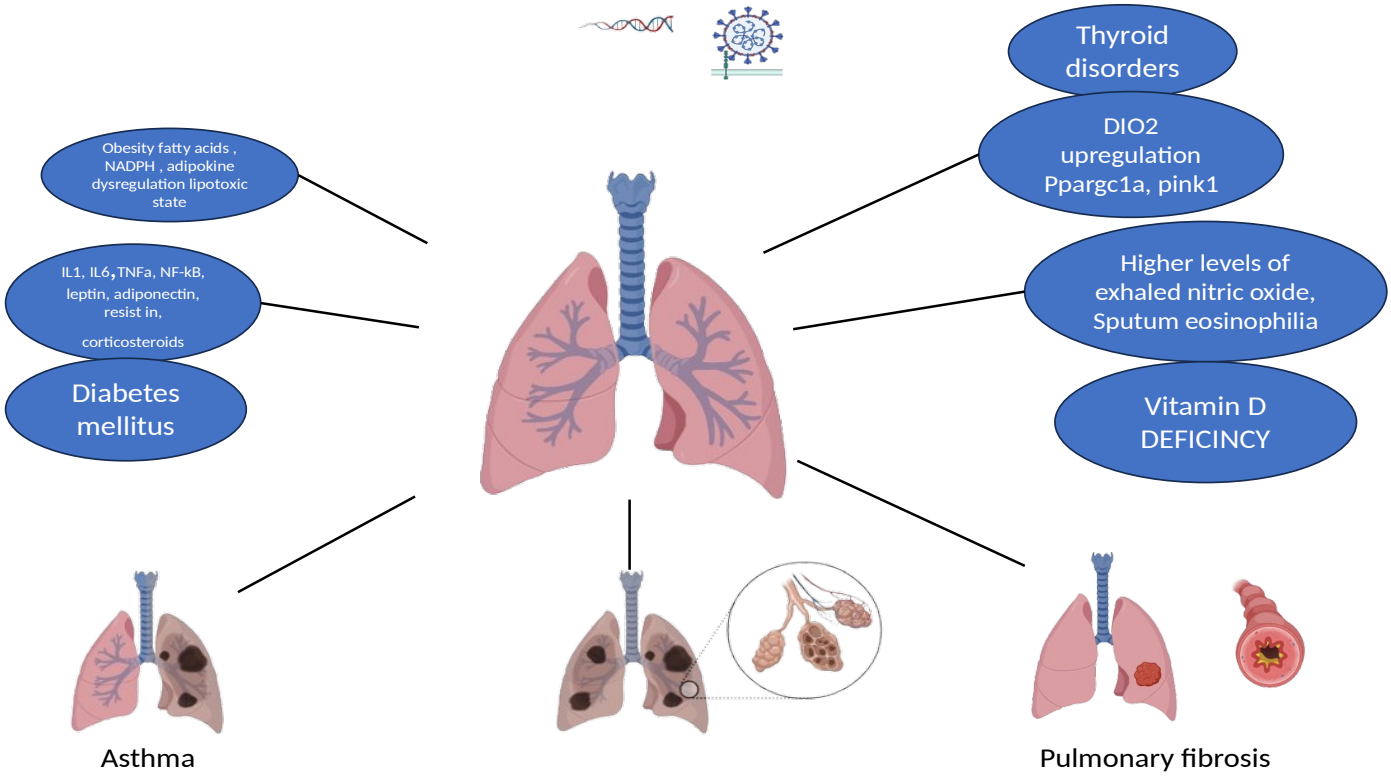

Figure 2: Metabolic-Inflammatory Pathways Linking Obesity, Dyslipidemia, and Diabetes to Lung Disease Pathogenesis

2.1. Systemic Inflammation and Oxidative Stress

MetS is characterized by chronic low-grade inflammation and oxidative stress, which play a significant role in the progression of lung diseases such as COPD and asthma [11]. In COPD, systemic inflammation driven by adipokines and pro-inflammatory cytokines (e.g., IL-6, TNF-α) exacerbates airway obstruction and accelerates lung function decline. Similarly, in asthma, obesity-related inflammation contributes to airway hyperresponsiveness and poor disease control[12].

Oxidative stress, a hallmark of MetS, further damages lung tissue by promoting the release of reactive oxygen species (ROS), which impair endothelial function and exacerbate pulmonary fibrosis [13]. This oxidative damage is particularly pronounced in patients with ILD, where it contributes to the progression of fibrotic changes and poor prognosis [14]

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2.2. Insulin Resistance

Insulin resistance (IR) is a key feature of MetS. IR leads to impaired glucose metabolism and hyperinsulinemia, which contribute to endothelial dysfunction and vascular remodeling in the lungs. In COPD patients, IR is associated with increased systemic inflammation and a higher risk of exacerbations [11]. The harmful cascade that is triggered by the fundamental elements of metabolic syndrome, such as central obesity, insulin resistance, dyslipidemia, and hypertension (Figure 3). These elements work together to promote the advancement of both systemic and pulmonary diseases. Chronic oxidative stress is caused by dysfunctional adipose tissue, which is a significant source of reactive oxygen species and pro-inflammatory adipokines. Through the activation of fibroblasts and the deposition of extracellular matrix, this biochemical milieu encourages fibrotic remodeling in several organs, including the lungs[11].

2.3. Obesity

Obesity, a major component of MetS, also mechanically restricts lung function by reducing chest wall compliance and increasing airway resistance. This is particularly evident in obese asthma patients, who experience more severe symptoms and poorer quality of life compared to non-obese asthmatics.[15]

2.4. Dyslipidemia

Cholesterol is important for the structure and function of lung cell membranes, with LDL and HDL supplying cholesterol to lung cells via designated receptors[16]. HDL maintain antioxidant delivery and surfactant production by alveolar type II cells, which is crucial for healthy lung function. However, raised cholesterol disrupts surfactant function and composition, highlighting the harmful effect of cholesterol imbalance on lung health[17].

2.5. Hypertension

High blood pressure changes the structure of the lungs and impair pulmonary circulation by increasing peripheral vascular resistance and decreasing blood flow and oxygen supply. This cause impaired function of small airways and damage to tissues, contributing to reduced lung capacity and endurance. The resulting ventilation abnormalities link hypertension directly to the deterioration of pulmonary function [18].

3. Impact of Metabolic Syndrome on Other Organs Associated Lung Disease

Insulin resistance, obesity, imbalanced cholesterol, and high blood pressure are the main components of metabolic syndrome, and how they work together to negatively impact lung health[5]. There is a relationship between metabolic syndrome and lung dysfunction, which is

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exacerbated by mechanisms such as oxidative stress, pro-inflammatory signaling, and vascular remodeling (Figure 4).

The body finds it difficult to control blood sugar when insulin resistance sets established, which results in elevated insulin levels. This weakens and stiffens the lungs' blood vessels over time, which makes it more difficult for air to pass through[19].

Being obese makes it difficult to take deep breathes since it physically presses against the lungs, like a heavy book on your chest[20]. The lungs lose out on protective antioxidants and have difficulty producing surfactant, a slippery material that maintains the flexibility of air sacs, when cholesterol levels are out of balance, particularly when "good" HDL levels are low. The lungs feel less flexible and rigid without it[21]. Contract blood arteries caused by high blood pressure decrease oxygen supply and blood flow. Your lungs' small airways become starved as a result, weakening and decreasing their effectiveness[22].

When combined, these difficulties can lead to severe lung disorders. Damage is further exacerbated by the accumulation of hard, scarred lung tissue and the proliferation of dangerous chemicals (reactive oxygen species). Metabolic syndrome exacerbates inflammation, which is akin to your body's warning system going into overdrive. Chronic illnesses like COPD and asthma are caused by fat-cell hormones (adipokines) and proteins like TNF-α and IL-6 that keep airways constricted and inflamed[10].

The main lesson is a malfunctioning metabolism affects not just your heart and waist, but also your lungs through a combination of oxidative stress, inflammation, poor blood flow, and physical strain. It is like a domino effect, when one issue causes another.

3.1. Cardiovascular System

MetS significantly increases the risk of CVD in lung disease patients. The combination of hypertension, dyslipidemia, and insulin resistance promotes atherosclerosis, leading to play a crucial role in the onset of left ventricular diastolic dysfunction, ultimately causing ischemic heart disease and heart failure [23] .In COPD patients,

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ISSN: 3093-4737

Figure 3. Pathophysiological mechanisms linking metabolic syndrome components to lung function impairment.

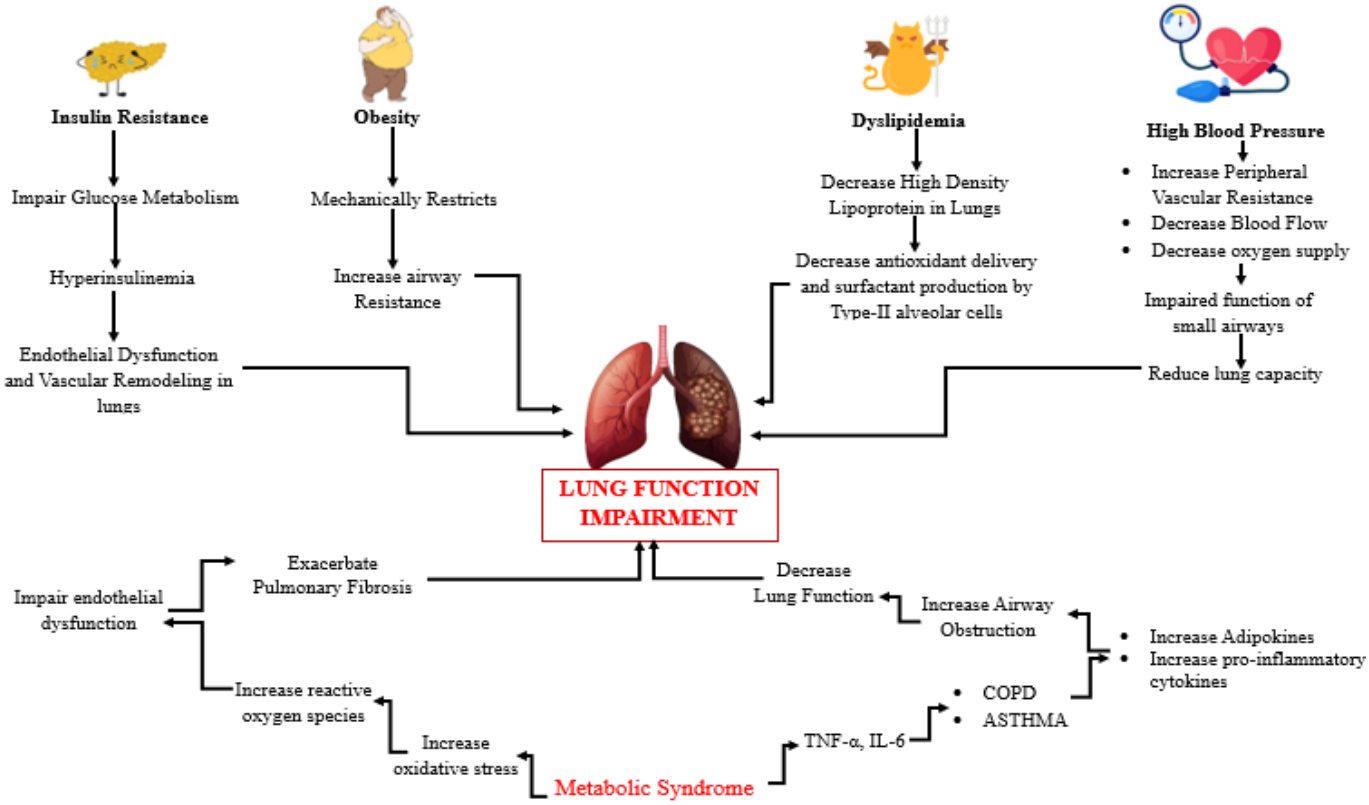

Figure 3: Shows Linkage Between Metabolic Syndrome Factors and Lung Function Impairment the presence of MetS is associated with a higher incidence of cardiovascular events, including myocardial infarction and stroke.

3.2. Liver

Non-alcoholic fatty liver disease (NAFLD) is a common comorbidity in patients with MetS and lung diseases. The systemic inflammation and insulin resistance associated with MetS promote hepatic steatosis and fibrosis, increasing the risk of liver-related morbidity and mortality. In COPD patients, NAFLD exacerbates systemic inflammation and contributes to the progression of both liver and lung disease[24].

3.3. Kidneys

Chronic kidney disease (CKD) is another common complication of MetS, particularly in patients with COPD and ILD. The activation of the mineralocorticoid receptor in MetS promotes renal

ISSN: 3093-4737

inflammation and fibrosis, leading to progressive kidney dysfunction. This renal impairment further exacerbates systemic inflammation and contributes to the progression of lung disease[25].

3.4. Brain

MetS is associated with an increased risk of neurodegenerative diseases, including Alzheimer's disease and Parkinson's disease, due to chronic inflammation and oxidative stress[26]. In lung disease patients, the systemic inflammation associated with MetS may accelerate cognitive decline and increase the risk of dementia[27].

4.1. Pharmacological Interventions

Pharmacological treatments for MetS, such as statins, angiotensin-converting enzyme inhibitors (ACEIs), and thiazolidinediones (TZDs), have shown promise in reducing systemic inflammation and improving metabolic control in lung disease patients [28]. Statins have been shown to reduce the risk of cardiovascular events and improve lung function in COPD patients [29] .

For the treatment of comorbidities in respiratory conditions such asthma, COPD, and interstitial lung disease (ILD), several medication groups exhibit potential. According to Jang et al. (2023), statins, such as atorvastatin, work by blocking HMG-CoA reductase[30]. They have been demonstrated to lower C-reactive protein (CRP) levels by 25% and lessen exacerbations in individuals with COPD. Liraglutide and other GLP-1 receptor agonists function by increasing insulin sensitivity and decreasing hunger.

ACE inhibitors such as ramipril reduce mortality in COPD patients by 18% by inhibiting the renin-angiotensin system (RAS) and having anti-fibrotic effects[31]. Hospitalizations for heart failure patients with obesity-related asthma have been reduced by 30% due to SGLT2 inhibitors like empagliflozin, which are known to promote glycosuria and weight loss [32]. These results demonstrate how metabolic medications may be repurposed to treat chronic respiratory conditions. (Table 1)

4.2. Novel Therapies

Emerging therapies, such as GLP-1 receptor agonists and SGLT2 inhibitors, have shown potential in managing both MetS and lung disease. These agents improve glycemic control, reduce body weight, and have anti-inflammatory effects that may benefit lung disease patients [33].

4.3. Lifestyle Modifications

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- **Diet**: Mediterranean diet reduces CRP by 20% in MetS-COPD
- **Exercise**: Pulmonary rehabilitation improves 6MWD by 45m in obese asthma[34].

4.4. Multidisciplinary Care

- Integrated programs reduce hospitalizations by 35% [35].

Drug Class	Examples	Mechanism	Indication	Efficacy	Key References
Statins	Atorvastatin	HMG-CoA reductase inhibition	CVD risk reduction in COPD	↓ CRP by 25%, ↓ exacerbations	[36]
GLP-1 Agonists	Liraglutide	Appetite suppression, insulin sensitization	Obesity in asthma	7% weight loss, ↑ FEV1	[37]
PPAR-γ Agonists	Pioglitazone	Adipocyte differentiation, anti-inflammatory	Insulin resistance in ILD	↑ DLCO by 15%, ↓ fibrosis	[38]
ACE Inhibitors	Ramipril	RAS inhibition, anti-fibrotic	Hypertension in COPD	↓ Mortality by 18%	[39]
SGLT2 Inhibitors	Empagliflozin	Glycosuria, weight loss	HF in obesity-related asthma	↓ Hospitalizations by 30%	[40]

Table 1: Drug Therapy for MetS in Lung Disease Patients

5. Clinical Advancement of MetS and its role in lung disease

Metabolic dysfunction is essential to explaining its potential relevance to lung health. Numerous pathways, each of which has been examined separately, have connected lung illness to metabolic disorders[9]. In experimental and translational studies, hyperglycemia, and dyslipidemia—abnormalities that affect the structure and function of the lungs—are most simply shown by elevations in triglycerides [41]..

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Adipose tissue disturbance in vulnerable people results in a pathological reaction to beneficial caloric balance, which both directly and indirectly fuels metabolic and cardiovascular illness. Adipose tissue is a metabolically complex organ that interacts with many different systems [15]. Recent studies aimed at clarifying the genetics of obesity and central obesity support the notion that excessive adiposity may be the cause of poor cardiovascular outcomes[42].

MetS is a silent comorbidity of COPD that has an impact on health status. Four important findings came from the investigation[43]. The prevalence of MetS in those with COPD was 37.4%.[44] In individuals with COPD, MetS was significantly correlated with the kind of chronic bronchitis and a high CRP level >0.6 mg/l. Abnormal glucometabolic states are common in patients with acute coronary syndrome; around one-third of them have diabetes, and a similar proportion have prediabetes[45, 46]. Patients with diabetes benefit most from a given level of cholesterol reduction with high-intensity statins or statins with ezetimibe, and they are particularly susceptible to recurrent ischemic cardiovascular events after an acute coronary syndrome[47]. Patients with MetS had higher rates of death, exacerbations of coronary artery disease, and stroke. In this study, the development of MetS in individuals with COPD was associated with the type of chronic bronchitis and a high CRP level (>0.6 mg/l) [48].

By modifying risk variables such as hypertension, diabetes, dyslipidemia, and the metabolic syndrome, a variety of routes can indirectly cause cardiometabolic disease and directly encourage atherosclerosis and endothelial cell dysfunction [49]. Adipokine dysregulation, oxidative stress, adipose tissue hypoxia, inflammation (including macrophage polarization), increased circulation free fatty acids, defective adipogenesis, lipotoxicity (local and systemic), and altered energy storage fat deposition are some of these processes [50, 51].

The cause of MetS in COPD patients is yet unknown. Numerous pathologic processes, including as age, a lack of physical exercise, inflammation of adipose tissue, systemic inflammation, and deterioration of pulmonary function, are thought to be implicated [52].

6. Discussion and Future Directions

Promising new treatments focus on gut-lung axis modification (e.g., probiotics) and senolytics (e.g., dasatinib). Treatment may be optimized using precision medicine techniques, such as genetic profiling (e.g., MC4R variations). According to the reviewed research, there is a strong reciprocal association between lung disorders, especially chronic obstructive pulmonary disease (COPD), and metabolic syndrome (MetS). Through systemic inflammation, adipose tissue dysregulation, and endothelial dysfunction, MetS—which is typified by dyslipidemia, hyperglycemia, and central obesity—seems to worsen pulmonary dysfunction. The fact that 37.4% of patients with COPD have MetS highlights the condition's status as a silent comorbidity

ISSN: 3093-4737

that accelerates the course of the disease, worsens cardiovascular outcomes, and increases systemic inflammation (as seen by raised CRP levels).

Because it encourages persistent low-grade inflammation, lipotoxicity, and insulin resistance—factors that may impede lung function and exacerbate the severity of COPD-adipose tissue dysfunction is a key player in this interaction. Given the correlation between MetS and the chronic bronchitis subtype, certain COPD phenotypes may be more vulnerable to metabolic disturbances. Additionally, in patients with COPD, the overlap between MetS and cardiovascular disease (CVD) suggests common pathogenic processes, including endothelial dysfunction and oxidative stress, which may increase the risk of morbidity and death. Mechanistic Research The molecular mechanisms that connect MetS and lung illness should be investigated further, with a focus on the involvement that adipokines, macrophage polarization, and systemic inflammation play in the development of COPD. Clarifying causal linkages may be aided by using animal models of mixed MetS and COPD.

Phenotype-Specific Interventions Future research should examine whether targeted anti-inflammatory therapies (statins, CRP-lowering agents) or metabolic interventions (GLP-1 agonists, SGLT2 inhibitors) are beneficial for COPD subgroups, given that chronic bronchitis and elevated CRP were strongly associated with MetS. Methods of Personalized Medicine: Finding high-risk patients who would profit from early metabolic therapies may be made easier with the use of genetic and biomarker research. Early Screening and Multidisciplinary Care: Routine metabolic screening (glucose, lipids, and CRP) should be incorporated into pulmonary clinics due to the high incidence of undetected MetS in COPD. Results may be improved by collaborative care models that include cardiologists, endocrinologists, and pulmonologists.

Lifestyle and Pharmacological Interventions: Research should evaluate how weight loss, exercise (pulmonary rehabilitation plus metabolic conditioning), and new anti-diabetic medications affect cardiovascular risk and lung function in people with COPD-MetS. Longitudinal Studies: Prospective cohort studies are required to ascertain whether MetS slows or stops the progression of COPD and whether treating it slows the deterioration in lung function.

7. Conclusion

In individuals with lung illness, metabolic syndrome is a prominent comorbidity that advances both pulmonary and extra-pulmonary problems. The pathophysiology of MetS in lung illnesses is based on the interaction of insulin resistance, oxidative stress, and systemic inflammation, which raises morbidity and mortality. To manage MetS in patients with lung disease and enhance their overall prognosis, a multidisciplinary strategy involving lifestyle modifications, medication, and innovative therapies is crucial. In patients with lung disease, metabolic syndrome is a

ISSN: 3093-4737

complicated and multifaceted disorder that contributes to the development of both pulmonary and extra-pulmonary consequences. It also considerably raises the risk of diabetes and cardiovascular disease.

Insulin resistance, dyslipidemia, hypertension, oxidative stress, and chronic inflammation all interact in the pathophysiology of MetS. To stop the development of more serious metabolic and cardiovascular problems, it is crucial to identify and treat MetS as soon as possible using pharmaceutical treatments and lifestyle modifications. Future studies should concentrate on creating focused therapies that target the underlying processes of MetS, especially in high-risk groups like obese people and elderly folks. A crucial area for translational research and therapeutic innovation is the confluence of pulmonary and metabolic disorders. In addition to improving lung health, treating MetS in COPD patients may lower cardiovascular morbidity and mortality, providing a comprehensive strategy for managing these interrelated illnesses.

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How to cite this article: Khan, F. et al. (2026). **Metabolic Syndrome in Lung Disease Patient and Its Impact on Other Organs. Pan-African Journal of Health and Psychological Sciences. Vol 2, Issue 2. April -June 2026.** **https://doi.org/10.64261/6x43w534.**

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