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Current Treatment of COPD - A Review

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ABSTRACT: A progressive respiratory disorder known as chronic obstructive pulmonary disease (COPD) is characterised by persistent respiratory symptoms and restricted airflow. In order to treat COPD, it must be possible to lessen symptoms, enhance quality of life, and avoid exacerbations. The current methods used to treat COPD are summarised in this abstract.

The cornerstone of COPD treatment is pharmacological therapies. Airflow is improved and bronchospasm is relieved with bronchodilators, such as short- and long-acting beta-agonists (LABAs), and anticholinergic. For individuals who experience frequent exacerbations and eosinophilic inflammation, inhaled corticosteroids (ICS) and LABAs are frequently administered together. However, ICS use is individualised depending on exacerbation risk and unique patient characteristics. The management of COPD must include non-pharmacological measures. The best intervention to decrease disease development continues to be quitting smoking. To prevent lung function deterioration and hospitalisations, acute COPD exacerbations require immediate care. During exacerbations, short courses of oral corticosteroids and antibiotics are frequently administered, with clinical and microbiological considerations. In some individuals with persistent respiratory failure, long-term oxygen therapy may be an option. A multimodal strategy combining pharmaceutical and non-pharmacological therapies is required for the management of COPD. The best results come from individualised treatment programmes that take into account the severity of the disease, the likelihood of an exacerbation, and specific patient variables. The range of treatments accessible to COPD patients is constantly expanding thanks to on-going research and improvements in therapeutic approaches. The variability of the illness and the difficulty in creating standardised treatment regimens are two difficulties in understanding and treating COPD. The development of pharmaceutical and non-pharmacological therapies, however, as a result of continuous research and improvements in therapy strategies, has improved results for COPD patients and broadened the spectrum of accessible treatment choices.

Keywords: COPD, Smoking, Bronchodilators, Pulmonary vasculatures, inflammatory.

INTRODUCTION

The chronic respiratory disorder known as chronic obstructive pulmonary disease (COPD) is characterised by persistent airflow restriction. It is a disease that may be prevented and treated, has airway obstruction, and is distinguished by enduring respiratory symptoms. About 16 million persons in the US are thought to have COPD. Patients who smoke or have a history of tobacco use, individuals who are older than 40, and men have the highest incidence of COPD. The goals of treating COPD are to alleviate symptoms, enhance quality of life, improve lung function, and lessen the frequency and severity of exacerbations. A combination of pharmaceutical and non-pharmacological therapies is used to treat COPD (Vestbo *et al*., 2013). A disease that can be prevented and treated, chronic obstructive pulmonary disease (COPD) is characterised by ongoing respiratory symptoms and reduced airflow. In 2016 it was classified as the third most common cause of death worldwide, while in 2017 it was the fourth most common cause of death in the US (Celli *et al.*, 2019). The goal of pharmacological treatment for chronic obstructive pulmonary disease (COPD) is to enhance overall health status and exercise tolerance while preventing and controlling symptoms and reducing the frequency and severity of exacerbations. However, lowering exposure to these risk factors, such as avoiding or quitting smoking early, can prevent COPD. Dyspnoea, coughing, and/or sputum production are the most typical respiratory symptoms linked to COPD. In addition to the everyday symptom load, COPD patients may experience episodes of acute respiratory symptoms

worsening (often referred to as "exacerbations"), which make up the majority of the overall COPD burden on healthcare systems (Montuschi, 2006).

SCOPE

The scope of this review article on the treatment of chronic obstructive pulmonary disease (COPD) would likely include the following aspects:

1. Pharmacological Therapies: The review would go over the pharmacological therapies that are currently being used to manage COPD, such as bronchodilators (such as short- and long-acting beta-agonists and anticholinergic) and the use of inhaled corticosteroids in conjunction with LABAs for people who experience frequent exacerbations and eosinophilic inflammation.

2. Non-pharmacological Measures: With a particular emphasis on quitting smoking as the key intervention to slow the progression of the disease, the review would examine the significance of non-pharmacological therapies in the management of COPD. Though the abstract gives no specifics, it might also describe alternative non-pharmacological methods.

3. Acute Exacerbation Management: Taking into account clinical and microbiological factors, the review would cover the immediate care necessary for acute COPD exacerbations, including the administration of brief courses of oral corticosteroids and antibiotics.

4. Long-Term Oxygen Therapy: The possible use of long-term oxygen therapy for people with persistent respiratory failure would be discussed in the review.

5. Multimodal Treatment Approach: In order to effectively manage COPD, the review would emphasise the necessity for a multimodal therapy approach that blends pharmaceutical and non-pharmacological therapies. It would emphasise the value of customised treatment plans depending on the severity of the disease, the likelihood of an exacerbation, and patient-specific variables.

6. Treatment Advancements and Challenges: The review would go over the growing variety of treatment options that COPD patients have access to as a result of continuous research and advancements in therapy strategies. It would recognise the disease's diversity and the difficulties in developing standardised treatment plans.

The review article will give an overview of the current techniques to treating COPD, including pharmacological and non-pharmacological therapies, acute exacerbation therapy, and the value of individualised treatment plans. It would also recognise the improvements in the management of COPD and the difficulties brought on by the condition.

ETIOLOGY

Prolonged exposure to hazardous chemicals or particles results in COPD. The most common cause of COPD worldwide is cigarette smoking. Other potential contributing factors are alpha-1 antitrypsin deficiency (AATD), second-hand smoking, and occupational and environmental exposures (Singh *et al*., 2019).

PATHOPHYSIOLOGY

COPD is an inflammatory disease that affects the pulmonary vasculature, lung parenchyma, and airways. Protease-antiprotease imbalances and oxidative stress are thought to play a role in the process. Emphysema is the name given to one of the structural alterations associated with COPD, in which the lungs' gas-exchanging surfaces known as alveoli are destroyed, causing obstructive physiology. Emphysema results from an inflammatory reaction to an irritant (such as smoking). The recruitment of neutrophils and macrophages results in the release of many inflammatory mediators. The air cavities are destroyed by oxidants and excessive proteases. Airways collapse during expiration as a result of the loss of elastic recoil caused by the protease-mediated degradation of elastin.

A rare form of emphysema called alpha-1 antitrypsin deficiency is characterised by a loss in antiproteases, which puts the lung parenchyma at risk for damage caused by proteases. The mutant protein's misfolding, which can build up in the liver, is what causes AATD. When COPD patients present with liver injury, AATD should be taken into consideration. In contrast to emphysema brought on by smoking, AATD primarily affects the lower lobes.

The forced expiratory volume (FEV1) is reduced by the inflammatory response and airway blockage, while airflow restriction and decreased gas exchange are brought on by tissue damage. Imaging examinations frequently reveal hyperinflation of the lungs, which results from air getting trapped when the airways collapse during exhalation. Increases in carbon dioxide (CO2) levels are also brought on by the inability to adequately exhale. A common symptom of illness progression is impairment of gas exchange. CO2 retention is caused by a decrease in ventilation or an increase in physiologic dead space. Diffuse vasoconstriction brought on by hypoxemia may cause pulmonary hypertension.

Acute COPD exacerbations are frequent and typically brought on by a trigger (such as viral or bacterial pneumonia, irritants in the environment, etc.). A rise in inflammation and air embolism frequently necessitates corticosteroid and bronchodilator therapy.

Impact of exacerbations on patients. Patients with COPD have been demonstrated to have considerably lower health-related quality of life when exacerbation frequency, severity, and recovery time increase. In order to lessen the severity of the respiratory symptoms and reduce the length of the exacerbation, antibiotics and/or oral corticosteroids are frequently prescribed for the treatment of COPD exacerbations. There is proof that treating a COPD exacerbation with oral antibiotics has an impact on subsequent occurrences, and treating exacerbations with oral prednisolone has a comparable impact with lower 30-day relapse rates. However, no study has yet looked into whether the length of an exacerbation's symptoms is related to how long it takes for the following exacerbation to occur (MacLeod *et al*., 2021; Solem *et al*., 2013).

Challenges in management of COPD. COPD is a heterogeneous, a varied, complex illness with many facets, COPD is influenced by both genetic and environmental factors. Due to the variety of COPD, there is growing interest in establishing various phenotypes according to clinical traits (Donaldson *et al*., 2003; Seifart and Plagens 2007). For instance, the GOLD ABCD classification categorises patients into four groups (phenotypes) depending on the severity of their symptoms and their history of exacerbations. The designation of patient subgroups based on underlying biologic distinctions, such as bacterial colonisation or, more contentiously, eosinophilic inflammation, is known as the idea of COPD endotypes. The term "precision medicine" refers to the individualization of treatment plans through the utilisation of clinical (phenotype) and biologic (endotype) data. Our knowledge of COPD subtypes is currently insufficient, and more research is required to better create unique, targeted therapy approaches rather than a "one-size-fits-all" strategy for COPD treatment. To manage the illness, healthcare providers must collaborate with their patients to identify the ideal mix of pharmaceutical and non-pharmacological therapy approaches, including dietary and lifestyle changes (Garudadri and Woodruff 2018; Agusti *et al*., 2016).

SYMPTOMS

Signs and symptoms of COPD may include:

- Shortness of breath, especially during physical activities
- Wheezing
- Chest tightness
- A chronic cough that may produce mucus (sputum) that may be clear, white, yellow or greenish
- Frequent respiratory infections
- Lack of energy
- Unintended weight loss (in later stages)
- Swelling in ankles, feet or legs

Treatment of Chronic Obstructive Pulmonary Disease (COPD)

1. Pharmacological Interventions

Pharmacological treatments for Chronic Obstructive Pulmonary Disease (COPD) are intended to enhance lung function, lessen exacerbations, and alleviate symptoms.

(a) Bronchodilators: The cornerstone of the current pharmacological therapy of COPD is bronchodilators. Long-acting bronchodilators lessen exacerbations and enhance quality of life in terms of health. Inconsistencies in exacerbation definitions or statistical analysis make it difficult to compare efficacy data for this outcome across trials using various long-acting bronchodilators. Tiotropium and salmeterol have different effects on exacerbations; thus, a clinical trial has been planned to assess their effects. To potentially detect a difference in the effectiveness of the two long-acting bronchodilators, the experiment would need to include 6,800 patients (Dusser *et al*., 2006). Salmeterol and formoterol are examples of selective long-acting 2 -agonists, while ipratropium bromide, oxitropium bromide, and tiotropium bromide are examples of selective short-acting 2 -agonists, anticholinergic antimuscarinic agents such as ipratropium bromide, oxitropium bromide and tiotropium bromide, and methylxanthines such as theophylline. Salmeterol and formoterol are examples of selective long-acting 2 -agonists, while ipratropium bromide, oxitropium bromide, and tiotropium bromide are examples of selective short-acting 2 -agonists.

(b) Inhaled corticosteroids: There is still confusion involving the use of inhaled corticosteroids as a long-term COPD treatment. Inhaled corticosteroids do not affect mortality on their own, but they may slow the deterioration of lung function.

ICS/LABA combination therapy: To lessen inflammation and the risk of exacerbations, ICS and LABA combinations (such as fluticasone/salmeterol or budesonide/formoterol) are advised for patients with more severe COPD and frequent exacerbations. However, the use of ICS depends on the unique patient characteristics, such as the history of exacerbations and levels of eosinophilic inflammation (Tashkin *et al*., 2018).

(c) Theophylline. One of the less expensive bronchodilators is theophylline, a methylxanthine. Theophylline is given intravenously in the form of aminophylline due to its extremely low water solubility. Theophylline and ethylenediamine combine to form aminophylline, which is 20 times more soluble than theophylline by itself. Theophylline has a bronchodilator effect because of its competitive antagonism of adenosine receptors and its relatively non-selective inhibition of cyclic nucleotide phosphodiesterase. Theophylline increases airflow to the lungs by relaxing the smooth muscles of the airways. Additionally, it has anti-inflammatory properties and can improve how well some bronchodilators work.

When patients do not effectively respond to or tolerate other bronchodilator drugs, or in cases of severe COPD, theophylline may be explored as an additional therapy. It is normally only prescribed to individuals with moderate to severe COPD who are still experiencing symptoms after receiving the best possible care from other drugs. Among the many negative effects of theophylline are gastrointestinal distress, headache, tremors, and elevated heart rate. Although they are uncommon, severe adverse effects can happen at higher doses or if blood levels rise too much.

Theophylline may interact with other pharmaceuticals, including certain antibiotics, antifungal treatments, and therapies that influence the liver enzymes. If theophylline is taken along with other drugs, close monitoring is required (Devereux *et al*., 2015).

(d) Selective β2 –agonists. Selective 2-agonists, sometimes referred to as 2-adrenergic agonists, are a type of drug that is frequently administered to treat Chronic Obstructive Pulmonary Disease (COPD). As a result of these drugs' primary focus on the airways' 2-adrenergic receptors, bronchodilation and increased airflow occur. The primary mechanism of action of selective 2-agonists is stimulation of 2-adrenoceptors on airway smooth muscle cells. An increase in intracellular levels of cyclic adenosine monophosphate

(cAMP) caused by the formation of the drug-receptor complex causes a stimulatory protein (Gs) to bind to guanosine triphosphate (GTP) and activate, in turn, the cAMP-dependent protein kinase (PKA). Myosin light chain kinase is activated by the latter, which results in a decrease in the enzyme's affinity for the calcium-calmodulin complex, a decrease in the production of active myosin light chain kinase, a decrease in the phosphorylation of myosin, and, finally, a decrease in the interaction between actin and myosin filaments, which leads to bronchodilation (Hatipoğlu, 2018).

- SABAs, or short-acting 2-agonists: Short-acting 2-agonists, like albuterol (salbutamol), are used as rescue drugs to treat acute symptoms of bronchospasm and to assist breathing. Typically, they are breathed in using a nebulizer or a metered-dose inhaler (MDI).Long-acting 2-agonists (LABAs): LABAs are used as maintenance therapy to give persistent bronchodilation over a longer length of time. Examples of LABAs are formoterol and salmeterol. For greater effectiveness, they are frequently delivered using inhalers or as a component of combination inhalers (LABA/ICS). Formoterol has a fast-occurring bronchodilator impact comparable to that of short acting 2 -agonists, in contrast to salmeterol, which has a gradual onset of action. Selective 2-agonists may occasionally be used in conjunction with additional drugs to treat COPD more thoroughly. To promote bronchodilation and lessen airway inflammation, combination inhalers that combine a LABA and an inhaled corticosteroid (ICS) are frequently prescribed.

(a) Phosphodiesterase-4 inhibitors. A class of drugs called PDE-4 (phosphodiesterase-4) inhibitors is used to treat Chronic Obstructive Pulmonary Disease (COPD). These drugs function by preventing the activity of the phosphodiesterase-4 enzyme, which lowers inflammation and relaxes the smooth muscles of the airways (Phillips, 2020).

The main PDE-4 inhibitor that has been approved for treatment in COPD is roflumilast. It comes in tablet form and is consumed orally.

PDE-4 inhibitors, like roflumilast, raise cellular levels of cyclic adenosine monophosphate (cAMP), which reduces the activity of inflammatory cells and the inflammation of the airways. By preventing the breakdown of cyclic AMP, selective inhibitors of phosphodiesterase (PDE) type 4 raise intracellular levels of the compound, which results in the relaxing of the smooth muscles of the airways and anti-inflammatory actions on effector cells involved in the pathogenesis of COPD. Small but significant improvements in lung function tests were seen in placebo-controlled studies for up to 6 months with cilomilast 15 mg twice daily and rofl umilast 500 mg once day in individuals with COPD (Chong *et al*., 2017).

(b) Antibiotics: Macrolide antibiotics: Long-term treatment of macrolide antibiotics (such as azithromycin) may be explored in some cases of COPD exacerbations to lessen exacerbation frequency (Bagge *et al*., 2021).

(c) Vaccinations: The influenza vaccination It is advised to get vaccinated against the flu every year to lower the risk of complications and respiratory infections.

Pneumococcal vaccine: According to research, pneumonia and its associated problems can be avoided by receiving vaccinations against pneumococcal infections, such as the pneumococcal conjugate vaccine (PCV13) and the pneumococcal polysaccharide vaccine (PPSV23) (Ji *et al*., 2022).

(d) Triple Therapy: ICS/LABA/LAAC combination therapy: Patients with severe COPD with a history of exacerbations may occasionally be prescribed a combination of inhaled corticosteroids, long-acting beta-agonists, and long-acting anticholinergics (LABA/ICS/LAAC) (Singh *et al*., 2017).

2. Non- Pharmacological Interventions

The management of Chronic Obstructive Pulmonary Disease (COPD) heavily relies on non-pharmacological measures. The main goals of these interventions are to enhance symptoms, functional ability, and overall quality of life for people with COPD through dietary changes, education, and supportive measures.

(a) Smoking cessation. The biggest risk factor for the onset of COPD in susceptible hosts is believed to be smoking. Therefore, quitting smoking is crucial for slowing the loss of lung function and should be started as soon as a diagnosis is made, especially when COPD is discovered in its early stages. One of the few therapies that has been demonstrated to reduce mortality is this one (Miravitlles and Anzueto 2009).

Replacement therapy for nicotine (NRT): When compared to placebo, NRT has a 2-fold higher rate of smoking cessation than that18. The purpose of NRT is to help patients quit smoking by giving them access to addictive nicotine without using dangerous tobacco. Numerous formulations exist, including a nasal spray, chew, lozenge, transdermal patch, inhaler, and transdermal patch (Tashkin *et al*., 2009).

(b) Rehabilitation. Compared to healthy controls, patients with COPD have lower levels of spontaneous physical activity. Regular exercise has been shown to slow the loss of lung function, lower the risk of COPD in smokers who are still smoking, and lower the likelihood that COPD patients may need to be hospitalised. Exercise capacity and dyspnoea are both improved by training the peripheral muscles, which also mitigates the increased oxidative stress brought on by exercise. Changes in a systemic and multidimensional assessment of performance and functional status (body mass index, airflow obstruction, dyspnoea, and exercise capacity index) generated by PR and patient outcomes are closely correlated in severe COPD (Jiloha, 2014; Garcia-Aymerich *et al.*, 2007).

(c) Transcutaneous neuromuscular electrical stimulation. Transcutaneous neuromuscular electrical stimulation (NMES) of the lower leg muscles enhances muscular oxidative capabilities within the PR framework. There have been a few small controlled studies utilising this approach in patients with severe COPD, including some who were bedridden (Clini *et al*., 2008).

(d) Immunization. Exacerbations are known to cause people with COPD to lose lung function more quickly and experience higher rates of morbidity and mortality. The guidelines issued by NICE presently advise giving COPD patients pneumococcal vaccinations in addition to their regular influenza vaccine. The evidence for the pneumococcal vaccine, however, is not as strong as it is for the influenza vaccine. When compared to people who had only received one of the two immunisations, individuals who had received both the pneumococcal and influenza vaccines had lower hospitalisation and death rates.

(e) Oxygen. For patients with advanced COPD who have chronic respiratory failure, LTOT is one of the principal treatments. The main objective of LTOT is to raise resting arterial oxygen pressure (Pa, O2) to 7.9 kPa (60 mmHg). Patients with COPD who are hypoxic who receive ongoing LTOT have a higher survival rate. The source of additional oxygen (gas or liquid), method of distribution, time spent using it, and flow rate at rest, during exercise, and while sleeping should all be specified when prescribing LTOT. Exercise endurance and dyspnoea are improved by oxygen therapy in patients who are normoxaemic at rest, but exercise capacity may not be directly and permanently affected. Adults with COPD have expensive outpatient therapy that includes additional home oxygen (Stoller *et al*., 2010).

(f) Physical activity. For people with COPD, encouraging regular physical activity is crucial. Exercises like walking, cycling, and swimming can enhance cardiovascular health, muscular strength, and general wellbeing. The level of physical exercise should be gradually increased and should be customised to the individual's preferences and capabilities (Shin, 2018).

(g) Nutritional support. For those with COPD, proper nutrition is crucial. Optimising nutritional intake can help you stay at a healthy weight and have enough muscle mass. Supplemental nutrition may be advised in cases of undernourishment or weight loss. Additionally, those who have COPD may benefit from getting enough water to thin mucus discharges and make breathing easier (Collins *et al*., 2019).

(h) Breathing techniques. People with COPD can improve their respiratory control and lessen their sense of being out of breath by using techniques like pursed-lip breathing and diaphragmatic breathing. These breathing exercises, which emphasise slow, deep breathing, can be taught to patients one-on-one or by medical experts as part of pulmonary rehabilitation (Gosselink, 2004).

(i) Environmental modifications. The quality of indoor air can be increased and respiratory irritation exposure can be decreased with changes to the living environment. Tobacco smoke exposure should be avoided, air purifiers should be used, ventilation should be maintained, and exposure to pollutants including dust, chemicals, and allergies should be kept to a minimum (Souto-Miranda *et al*., 2020).

(j) Psychological support. It can be difficult to manage a chronic condition like COPD, therefore psychological assistance is essential. An individual's disease can be more effectively managed and their general well-being can be improved with the help of supportive counselling, education, and stress-management techniques (Sohanpal *et al*., 2020).

CONCLUSIONS

In conclusion, treatment for chronic obstructive pulmonary disease (COPD) involves a combination of pharmaceutical and non-pharmacological approaches. To improve airflow and manage exacerbations in COPD patients, pharmaceutical therapies such bronchodilators and inhaled corticosteroids are employed. Non-pharmacological therapies like quitting smoking are crucial for the management of illnesses. As soon as an exacerbation occurs, corticosteroids and antibiotics should be given to help arrest further decline. Chronic respiratory failure patients may be candidates for long-term oxygen therapy. The options available to those who suffer from COPD are expanding as a consequence of ongoing research and advancements in therapy modalities that have improved treatment outcomes.

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REFERENCES

Agusti, A., Bel, E., Thomas, M., Vogelmeier, C., Brusselle, G., Holgate, S. & Pavord, I. D. (2016). Treatable traits: toward precision medicine of chronic airway diseases. *European Respiratory Journal*, *47*(2), 410-419.

Bagge, K., Sivapalan, P., Eklöf, J., Hertz, F. B., Andersen, C. Ø., Hansen, E. F. & Jensen, J. U. S. (2021). Antibiotic treatment in acute exacerbation of COPD: patient outcomes with amoxicillin vs. amoxicillin/clavulanic acid—data from 43,636 outpatients. *Respiratory research*, *22*(1), 1-9.

Celli, B. R. & Wedzicha, J. A. (2019). Update on clinical aspects of chronic obstructive pulmonary disease. *New England Journal of Medicine*, *381*(13), 1257-1266.

Chong, J., Leung, B. & Poole, P. (2017). Phosphodiesterase 4 inhibitors for chronic obstructive pulmonary disease. *Cochrane Database of Systematic Reviews*, (9).

Clini, E. M., & Ambrosino, N. (2008). Nonpharmacological treatment and relief of symptoms in COPD. *European Respiratory Journal*, *32*(1), 218-228.

Collins, P. F., Yang, I. A., Chang, Y. C. & Vaughan, A. (2019). Nutritional support in chronic obstructive pulmonary disease (COPD): an evidence update. *Journal of thoracic disease*, *11*(Suppl 17), S2230.

Devereux, G., Cotton, S., Barnes, P., Briggs, A., Burns, G., Chaudhuri, R. & Price, D. (2015). Use of low-dose oral theophylline as an adjunct to inhaled corticosteroids in preventing exacerbations of chronic obstructive pulmonary disease: study protocol for a randomised controlled trial. *Trials*, *16*(1), 1-10.

Donaldson, G. C., Seemungal, T. A. R., Patel, I. S., Lloyd-Owen, S. J., Wilkinson, T. M. A. & Wedzicha, J. A. (2003). Longitudinal changes in the nature, severity

and frequency of COPD exacerbations. *European Respiratory Journal*, *22*(6), 931-936.

Dusser, D., Bravo, M. L. & Iacono, P. (2006). The effect of tiotropium on exacerbations and airflow in patients with COPD. *European Respiratory Journal*, *27*(3), 547-555.

Garcia-Aymerich, J., Lange, P., Benet, M., Schnohr, P., & Antó, J. M. (2007). Regular physical activity modifies smoking-related lung function decline and reduces risk of chronic obstructive pulmonary disease: a population-based cohort study. *American journal of respiratory and critical care medicine*, *175*(5), 458-463.

Garudadri, S. & Woodruff, P. G. (2018). Targeting chronic obstructive pulmonary disease phenotypes, endotypes, and biomarkers. *Annals of the American Thoracic Society*, *15*(Supplement 4), S234-S238.

Gosselink, R. (2004). Breathing techniques in patients with chronic obstructive pulmonary disease (COPD). *Chronic respiratory disease*, *1*(3), 163-172.

Hatipoğlu, U. (2018). Chronic obstructive pulmonary disease: More than meets the eye. *Annals of Thoracic Medicine*, *13*(1), 1.

Jiloha, R. C. (2014). Pharmacotherapy of smoking cessation. *Indian journal of psychiatry*, *56*(1), 87.

Ji, Z., Jareño-Esteban, J. J. & de Miguel-Díez, J. (2022). Role of Vaccines in COPD Patients. *Open Respiratory Archives*, *4*(3), 100191.

MacLeod, M., Papi, A., Contoli, M., Beghé, B., Celli, B. R., Wedzicha, J. A. & Fabbri, L. M. (2021). Chronic obstructive pulmonary disease exacerbation fundamentals: Diagnosis, treatment, prevention and disease impact. *Respirology*, *26*(6), 532-551.

Miravitlles, M. & Anzueto, A. (2009). Insights into interventions in managing COPD patients: lessons from the TORCH and UPLIFT® studies. *International Journal of Chronic Obstructive Pulmonary Disease*, 185-201.

Montuschi, P. (2006). Pharmacological treatment of chronic obstructive pulmonary disease. *International journal of chronic obstructive pulmonary disease*, *1*(4), 409-423.

Phillips, J. E. (2020). Inhaled phosphodiesterase 4 (PDE4) inhibitors for inflammatory respiratory diseases. *Frontiers in Pharmacology*, *11*, 259.

Seifart, C. & Plagens, A. (2007). Genetics of chronic obstructive pulmonary disease. *International journal of chronic obstructive pulmonary disease*, *2*(4), 541-550.

Shin, K. C. (2018). Physical activity in chronic obstructive pulmonary disease: clinical impact and risk factors. *The Korean journal of internal medicine*, *33*(1), 75.

Singh, D., Agusti, A., Anzueto, A., Barnes, P. J., Bourbeau, J., Celli, B. R. & Vogelmeier, C. (2019). Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019. *European Respiratory Journal*, *53*(5).

Singh, D., Corradi, M., Spinola, M., Papi, A., Usmani, O. S., Scuri, M. & Vestbo, J. (2017). Triple therapy in COPD: new evidence with the extrafine fixed combination of beclomethasone dipropionate, formoterol fumarate, and glycopyrronium bromide. *International Journal of Chronic Obstructive Pulmonary Disease*, 2917-2928.

Stoller, J. K., Panos, R. J., Krachman, S., Doherty, D. E. & Make, B. (2010). L. term Oxygen Treatment Trial Research Group. *Chest*, *138*, 179.

Sohanpal, R., Pinnock, H., Steed, L., Heslop Marshall, K., Chan, C., Kelly, M. & TANDEM Investigators (2020). Tailored, psychological intervention for anxiety or depression in people with chronic obstructive pulmonary disease (COPD), TANDEM (Tailored intervention for ANxiety and DEpression Management in COPD): protocol for a randomised controlled trial. *Trials*, *21*, 1-12.

Solem, C. T., Sun, S. X., Sudharshan, L., Macahilig, C., Katyal, M. & Gao, X. (2013). Exacerbation-related impairment of quality of life and work productivity in severe and very severe chronic obstructive pulmonary disease. *International journal of chronic obstructive pulmonary disease*, 641-652.

Souto-Miranda, S., Gonçalves, A. C., Valente, C., Freitas, C., Sousa, A. C. & Marques, A. (2020). Environmental awareness for patients with COPD undergoing pulmonary rehabilitation: is it of added value?. *International Journal of Environmental Research and Public Health*, *17*(21), 7968.

Tashkin, D. P. & Strange, C. (2018). Inhaled corticosteroids for chronic obstructive pulmonary disease: what is their role in therapy?. *International journal of chronic obstructive pulmonary disease*, 2587-2601.

Vestbo, J., Hurd, S. S., Agustí, A. G., Jones, P. W., Vogelmeier, C., Anzueto, A. & Rodriguez-Roisin, R. (2013). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *American journal of respiratory and critical care medicine*, *187*(4), 347-365.

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