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Pulmonary Rehabilitation and Physical Activity in COPD

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Abstract

Introduction and Purpose: Chronic obstructive pulmonary disease (COPD) is a type of progressive lung disease characterized by chronic respiratory symptoms and airflow limitation. The purpose of this study was to present pathogenesis, symptoms, diagnosis and treatment of COPD and principal strategies and types of exercises of pulmonary rehabilitation in patients with COPD.

Description of State of Knowledge: Chronic obstructive pulmonary disease (COPD) leads to progressive respiratory dysfunction, muscle loss, and decreased exercise capacity, significantly impairing quality of life. Pulmonary rehabilitation (PR) is a key non-pharmacological intervention, combining exercise training, education, and self-management to improve functional capacity and reduce symptoms. Various training modalities have demonstrated benefits in enhancing endurance, muscle strength, and quality of life in COPD patients. Individualized programs based on patient capacity and symptom burden are essential for optimizing rehabilitation outcomes.

Conclusions: Pulmonary rehabilitation relieves dyspnoea and fatigue, improves emotional function and enhances the sense of control that individuals have over their condition. These improvements are moderately substantial and clinically significant. Rehabilitation serves as an essential component of the management of COPD and is beneficial in improving health-related quality of life and exercise capacity.

Keywords: Chronic obstructive pulmonary disease, COPD, pulmonary rehabilitation

1. Introduction and purpose

Chronic obstructive pulmonary disease (COPD) is expected to become an increasingly significant global cause of long-term illness and mortality in the coming decade [1].

In COPD, restricted airflow increases the effort needed for breathing and leads to changes in breathing mechanics, including both static and dynamic lung hyperinflation. These changes alter the shape of the chest and diaphragm length, pushing respiratory muscles outside their optimal range for generating force. Additionally, muscle loss is linked to multiple molecular mechanisms, including inflammation, activation of the hypoxia-inducible factor-1 pathway,

oxidative stress, reduced oxidative enzyme activity, and a lower number of capillaries [2]. Muscle dysfunction in the lower limbs is also influenced by factors such as genetic predisposition, cigarette smoking, elevated CO_2 levels, acidosis, hormonal and metabolic imbalances (including low testosterone and vitamin D), poor nutrition, medications, coexisting diseases, disease exacerbations, systemic inflammation, lack of physical activity, and aging [3]. Compared to healthy individuals of the same age, COPD patients often show a higher energy cost during physical activity, with early lactic acid buildup and decreased maximum work capacity and oxygen uptake [4]. As physical inactivity worsens, deconditioning sets in, increasing the sensation of breathlessness. Many patients gradually become confined to their homes, withdrawing from social life, which often contributes to depression and anxiety, further diminishing their quality of life (QOL) [5].

2. Pathogenesis of COPD

The primary etiology of chronic obstructive pulmonary disease (COPD) is an inflammatory response triggered by inhaled toxic agents, with tobacco smoke being the predominant source. The immune system is activated through pattern recognition receptors (PRRs), primarily Toll-like receptors (TLRs), sensing pathogen-associated molecular patterns (PAMPs) in the smoke. The stimulation of PRRs enhances the secretion of cytokines and chemokines, which mediate inflammation. These molecules facilitate the infiltration of neutrophils, macrophages, and leukocytes into the airway and lung tissue. Additionally, proinflammatory cytokines, primarily TNF-alpha and IL-1, induce a systemic response, leading to elevated body temperature, increased production of acute-phase proteins by the liver, and enhanced leukocyte proliferation in the bone marrow [6]. The accumulation of activated neutrophils at the site, secreting proteases such as elastase, results in the proteolysis of extracellular matrix components. Protein degradation is exacerbated by a deficiency of antiproteases, primarily α1-antitrypsin. Increased extracellular matrix degradation, lung-maintenance failure, and alveolar cell apoptosis have all been implicated in the pathogenesis of emphysema in COPD [7]. The persistent immune response stimulates the activation of interstitial fibroblasts, which synthesize extracellular matrix (ECM) at the site of damage, and epithelial cells, which contribute to basement membrane production. This activation is primarily mediated by transforming growth factor-beta (TGF-β) [6]. Elevated levels of VEGF and IL-13 lead to the enlargement of bronchial mucus glands and hyperresponsiveness of bronchial smooth muscles [8]. Airway remodeling, along with the infiltration of inflammatory cells and increased mucous secretion, is the primary cause of airflow obstruction in COPD. Additionally, the

emphysematous destruction of lung tissue reduces elastic recoil, impairing the ability of the airways to remain open during exhalation [6].

3. Symptoms

The most common symptoms are those associated with decreased lung function, such as dyspnea, cough, and sputum expectoration. Less common systemic symptoms include chest pain and heart palpitations. It is important to recognize that COPD symptoms fluctuate depending on the season and time of day, with the most severe symptoms typically occurring in the morning. Both respiratory and systemic symptoms can disrupt sleep, leading to hypoxemia and hypercapnia, which may contribute to the development of comorbid conditions [9]. COPD may also lead to systemic alterations driven by oxidative stress and circulating inflammatory mediators, resulting in muscle wasting, weight loss, and hormonal dysfunction [10]. Due to muscle degradation and respiratory impairment, physical activity is significantly restricted in patients with COPD [11]. COPD is also frequently associated with psychological conditions such as anxiety and depression. Collectively these symptoms have a substantial impact on patients' quality of life and daily functioning [9].

4. Diagnosis

Currently, the gold standard in the diagnosis of chronic obstructive pulmonary disease (COPD) remains spirometry, particularly the assessment of the FEV_1/FVC ratio (with values <70% indicating airflow limitation). This test is fundamental not only for confirming the presence of airway obstruction but also for predicting clinical outcomes such as mortality and hospitalization rates. Additionally, it serves as a basis for considering non-pharmacological treatment options, including lung volume reduction procedures or lung transplantation [1]. However, spirometry does not always correlate with the full spectrum of symptoms experienced by patients, nor does it consistently reflect the disease’s impact on quality of life or the frequency of exacerbations [12]. The severity of COPD (from stage I to IV) is classified based on the level of airflow limitation, measured using the forced expiratory volume in one second (FEV_1). In clinical practice, a valuable complementary tool is the COPD Assessment Test (CAT), which evaluates a range of physical symptoms including cough, sputum production, chest tightness, breathlessness when climbing hills or stairs, limitations in home activities, and energy levels. The CAT has shown good responsiveness to changes in the patient's condition, such as improvement following pulmonary rehabilitation or recovery after an exacerbation.Another commonly used tool is the Modified Medical Research Council (mMRC) dyspnea scale, which ranges from 0 to 4. It is a simple way to assess how

breathlessness affects daily activities. However, one of its limitations is its low sensitivity to clinical changes, for example, in response to treatment.The Clinical COPD Questionnaire (CCQ) offers a more comprehensive view of the disease's impact on the patient by evaluating symptoms, functional status, and mental health. It is also sensitive to clinical improvements after smoking cessation and during or after exacerbation events [13]. In recent years, quantitative chest computed tomography (CT) has gained increasing attention in COPD diagnostics [14]. CT imaging allows for the detection and assessment of structural lung changes and may be particularly useful in patients who are unable to undergo spirometry [15]. Finally, while less specific, chest X-ray (CXR) often serves as the first imaging tool used in the evaluation of suspected COPD. It can demonstrate features such as pulmonary emphysema, a key component of COPD, and provide supportive evidence for diagnosis [16].

5. Treatment

The treatment of chronic obstructive pulmonary disease (COPD) focuses primarily on alleviating symptoms and preventing future exacerbations [1]. It consists of three complementary components: reduction or elimination of risk factors, primarily smoking cessation, symptomatic pharmacological treatment, mainly involving inhaled medications and pulmonary rehabilitation, which supports respiratory function and overall physical capacity. Many patients experiencing COPD exacerbations also suffer from comorbid conditions, which can complicate both diagnosis and treatment. In some cases, events identified as exacerbations may in fact reflect manifestations of underlying comorbidities [17]. In this context, personalized medicine is gaining importance and may play a key role in preventing exacerbations in selected groups of COPD patients [18]. One example of a personalized approach is measuring blood eosinophil levels, which can help predict the likelihood of benefit from the addition of inhaled corticosteroids (ICS) to baseline bronchodilator therapy [1]. Recent evidence also supports the role of macrolide antibiotics in managing COPD. For example, low-dose erythromycin therapy administered over 12 months has been shown to reduce both the frequency and severity of exacerbations in patients with moderate to severe COPD [19]. Similarly, daily azithromycin treatment has proven effective in reducing exacerbation rates and is currently recommended for patients at high risk of recurrent episodes [20]. A cornerstone of both COPD prevention and management is the identification and reduction of modifiable risk factors, particularly smoking cessation [21]. Smoking cessation therapies, including varenicline, bupropion, nortriptyline, nicotine gum, nicotine inhalers, nasal sprays, and patches, can significantly enhance the success rate of quitting [22].

Varenicline, a partial agonist of the α4β2 nicotinic receptor, may be offered as a second-line therapy when nicotine replacement therapy proves ineffective [23].Pulmonary rehabilitation is a key component of comprehensive COPD treatment. It can significantly alleviate dyspnea and fatigue, improve emotional functioning, and increase patients' sense of control over their condition [24]. Moreover, it has a positive impact on exercise tolerance, quality of life, anxiety, and depression symptoms, while also reducing healthcare utilization by lowering the frequency of exacerbations and shortening hospital stays [23]. As part of preventive strategies, influenza and pneumococcal vaccinations (PCV13 and PPSV23) are recommended for patients with COPD, especially older adults [1]. These vaccinations have been shown to reduce the risk of severe respiratory infections and may also decrease the frequency and severity of exacerbations [25]. Pharmacological treatment of COPD mainly involves inhaled medications from the following therapeutic classes: long-acting β2-agonists (LABA), long-acting muscarinic antagonists (LAMA), inhaled corticosteroids (ICS) [21]. Depending on the clinical presentation, initial therapy may include combination treatments such as: LAMA/LABA – for patients with severe dyspnea, LABA/ICS – for patients with a high risk of exacerbations and elevated blood eosinophil levels [1]. In mild COPD, short-acting bronchodilators — either β2-agonists or anticholinergics — are used for rapid symptom relief. For long-term management, long-acting bronchodilators are preferred as they: reduce dyspnea, improve exercise capacity and quality of life, lower the frequency and severity of exacerbations [23].

6. Training Modalities

Eccentric training, requiring minimal energy, is ideal for pulmonary rehabilitation to enhance muscle strength. Exercise prescription follows the FITT principles—frequency, intensity, type, and timing—with at least two 20–30 min sessions per week for 8+ weeks recommended for COPD patients [26]. Intensity and duration are key factors in predicting outcomes, with low-intensity training being well tolerated, especially in severe COPD cases. High-intensity and enduring exercise training modalities are frequently applied in pulmonary rehabilitation. Interval or intermittent training might be the right choice for those with a more serious disease and higher symptom burden of illness (dyspnoea, fatigue or incompleteness of the target intensity and/or duration), while high-intensity training improves aerobic metabolism and reduces dyspnoea. Compared to traditional methods, interval training minimizes pulmonary hyperinflation, extends training tolerance, and lowers exertional dyspnoea. Combining high- and low-intensity training helps COPD patients manage symptoms. Resistance training using

heavy loads increases muscle mass and peripheral strength, with less dyspnoea than endurance training [27].

7. Principal Pulmonary Rehabilitation Strategies

7.1 Inspiratory Muscle Training (IMT)

In recent years, Inspiratory Muscle Training (IMT) has gained attention for COPD management [28]. Inspiratory muscle training (IMT) can strengthen these muscles, improve endurance, and ease breathing difficulties. The most common methods involve threshold or resistive loads, typically based on maximal inspiratory pressure (PImax) to enhance inspiratory muscle strength. The top three methods are flow-resistive loading, mechanical threshold loading (MTL), and normocapnic hyperpnoea (low pressure-high flow loading). Starting with >30% of PImax is recommended, gradually increasing as tolerated. Training sessions usually last 30 minutes to 1 hour, 3-7 days a week, often divided into two or three sittings. Most IMT interventions in COPD patients have been performed as wholly or partially supervised daily training for 30 min with controlled training loads using MTL-IMT [29].

7.2 Downhill Walking (DW)

Downhill walking (DW) is an exercise modality with a high eccentric component that combines quadriceps endurance training and aerobic exercise. Due to its low metabolic demand and high eccentric muscle fatigue, it is a well-suited training method for individuals with COPD. DW helps induce skeletal muscle stress while minimizing ventilatory demand [30]. A study found that 90% of COPD patients experienced muscle fatigue after 20 minutes of downhill walking, compared to 60% after treadmill walking. DW also resulted in 17% less oxygen consumption and 9% less ventilation than treadmill walking. The development of low-frequency fatigue (LFF), which is linked to better recovery from exercise, can be induced by eccentric training like DW. In COPD patients, inducing LFF has been shown to improve functional exercise capacity and quality of life (QoL). Thus, while DW does not directly address COPD symptom progression, it improves overall QoL and exercise capacity. After 3 months of exercise therapy, sustained improvement was observed [31].

7.3 Eccentric Resistance Training (RT)

Eccentric muscle work is notable for its reduced energy requirement, allowing high loads on muscles with small energy requirements for muscle contraction. Mechanical load is essential for muscle maintenance and adaptation. Resistance training (RT) tends to result in lower oxygen consumption and dyspnoea scores, making it more tolerable for COPD patients. For

RT in COPD patients, the prescription should involve 1–3 sets of 8–12 repetitions at 60-70% of the one-repetition maximum (or a load that induces fatigue after 8-12 reps), with sessions 2-3 days/week [32]. To improve dyspnoea levels, it should longer training durations (over 8 weeks). There is an obvious reduction in breathlessness in neither supported nor unsupported training. Single limb exercise training (exercising one limb at a time) can be considered an alternative exercise strategy for individuals with severe respiratory disease [33]. Low-load/high-repetition single limb exercise involves training one upper or lower limb at a time, alternating between sides in each set. This strategy reduces ventilatory demand and oxygen consumption by utilizing a smaller volume of muscle mass at once. For individuals with severe COPD, this approach, especially when using elastic bands, can improve peripheral muscle strength and extremity work capacity. Eccentric cycling is an advanced form of stationary cycling, requiring less metabolic effort while providing higher power output [9]. It causes minimal muscle soreness, dyspnoea, and muscle fatigue, making it a promising option for individuals with respiratory conditions [34]. Single-legged cycling is an alternative strategy of endurance training that minimizes ventilatory load by targeting smaller muscle groups and supporting the same muscle load while still providing endurance benefits similar to regular cycling. Compared to two-legged cycling at 70% peak power, single-legged cycling at 35% peak power results in similar metabolic demands but with lower ventilation and less dyspnoea. This approach can enhance endurance exercise efficacy and improve work capacity in COPD patients. After practicing three times weekly for 7 weeks, VO2 peak improved more significantly in the one-legged cycling group (15 minutes per leg) compared to the two-legged cycling group (30 minutes total). Single-legged cycling has been shown to enhance peak cycling power, VO2 peak, and peak minute ventilation, as well as improve 6-minute walk distance and health-related quality of life [35].

7.4 Neuromuscular Electrical Stimulation (NMES)

Neuromuscular Electrical Stimulation (NMES) uses a battery-powered stimulator to contract muscles via skin electrodes, primarily targeting the quadriceps. It offers a rehabilitation alternative for patients unable or unwilling to perform traditional exercise [36]. Stimulation intensity is adjusted until a strong visible contraction is achieved or reaches the patient's maximum tolerable level. It commonly targets the thigh and calf muscles. A standard NMES program includes 30–60 minutes of stimulation, 3–5 times per week for 4–6 weeks. A long contraction period followed by an extended rest may enhance NMES effectiveness by maximizing mechanical stress and stimulating contractile protein synthesis [37]. Given its low

metabolic demand, NMES is especially beneficial for severely deconditioned or bed-bound COPD patients who struggle with traditional resistance training [38]. While NMES avoids respiratory strain, limitations include skin discomfort and inconsistent reproducibility [39].

7.5 Whole-Body Vibration Training (WBVT)

Vibration training involves voluntarily exposing the body to dynamic frequencies at specific joint angles for a set duration. WBVT is a therapeutic lung rehabilitation method that enhances neuromuscular performance through sinusoidal tonic vibrations by stimulating muscle contractions. Unlike traditional resistance training, where muscle contractions are voluntary, WBV relies on stretch reflexes to activate the muscles. This type of training does not require direct muscle control, with the user only needing to manage posture and movement. For patients with advanced COPD, WBV has proven to be an effective and feasible exercise modality. It can significantly improve functional exercise capacity, particularly when combined with endurance and strength training [40]. In COPD patients, WBVT increases blood circulation by nearly 14% and enhances vascular endothelial growth factor levels, promoting blood vessel formation. It also improves muscle elasticity, coordination, and exercise capacity, with greater benefits observed in stable COPD patients compared to those with severe COPD. Its benefits are primarily linked to increased respiratory muscle contraction, which enhances ventilation, blood flow, and breathing capacity [41].

7.6 Contractile Fatigue After Exercise

Ventilatory limitations might prevent patients with better preserved oxidative metabolism from reaching a sufficient training intensity to induce a similar overload [42].

7.7 Nutritional Support and Anabolic Stimulation

Adequate nutrition is essential for COPD patients. Studies show that combining dietary supplementation with an 8-week pulmonary rehabilitation program can improve body weight, muscle mass, respiratory and limb muscle strength, exercise capacity, and quality of life [43]. Dietary supplementation is particularly beneficial when combined with anabolic stimuli like exercise training, especially for patients with advanced COPD and poor body composition. Drugs like testosterone, growth hormone analogues, megestrol acetate, L-carnitine, creatine, antioxidants, and vitamin D may help counteract muscle loss and dysfunction of limb muscles in COPD patients [44].

7.8 Oxygen Supplementation During Exercise

Oxygen therapy enhances peripheral muscle oxygen levels, reduces dyspnoea, and boosts exercise capacity in COPD patients with hypoxaemia, likely enabling them to engage in training at higher intensities. It is strongly recommended for those with severe resting hypoxaemia, aiming for oxygen saturation >90% or arterial oxygen pressure >55 mmHg. Even in patients who don't qualify for long-term oxygen therapy or lack exercise-induced hypoxaemia, using oxygen during training may still offer benefits [45].

7.9 Heliox Supplementation

Heliox (HX), a gas mixture of 79% helium and 21% oxygen, is used in COPD patients to reduce airflow resistance during exercise. It helps decrease dynamic lung hyperinflation and improves cardio-circulatory functions. Heliox also enhances the flow-volume loop size and slows the increase in end-expiratory lung volume (EELV) during training, preventing respiratory muscle overload and functional weakening [46].

7.10 Incremental cycle ergometry

Exercise tests that have an incremental phase lasting between 8 to 12 minutes are considered effective and yield valuable diagnostic data. Key performance measures from the test—maximum work rate, peak heart rate, or peak oxygen uptake (VO_2 peak)—can be used to establish appropriate training intensities for an endurance program [47].

7.11 Constant Work Rate Test

In this test, the patient cycles at 70% of their peak work rate and continues until they reach exhaustion. The duration for which the patient can maintain this effort is recorded and used as the key outcome measure [48].

7.12 6-min walk test

The 6-Minute Walk Test (6MWT) requires a 30-meter hallway but does not involve any exercise equipment. It assesses the distance a patient can walk briskly on a flat, hard surface over a 6-minute period, moving back and forth around cones. Strict standardization of the testing procedure is essential to reduce potential bias from the test administrator. Typically, oxygen saturation, heart rate, perceived breathlessness, and leg fatigue (rated using the Borg scale) are recorded at the beginning and end of the test, along with the total walking distance (in meters) achieved during the 6 minutes [49].

7.13 Incremental shuttle walking tests

The Incremental Shuttle Walking Test (ISWT) is a field-based test where patients walk back and forth on a 10-meter course, guided by audio signals that gradually increase the walking

pace each minute. The test ends when the patient can no longer reach the turnaround point in time. The total distance walked is recorded and reflects maximal exercise capacity [50].
A variation of this is the Endurance Shuttle Walking Test (ESWT), where patients walk at 85% of the peak speed reached during the ISWT until exhaustion. The primary outcome is walking time. The ESWT is particularly sensitive to improvements after pulmonary rehabilitation [51].

7.14 Sit-to-stand tests

This test measures either the number of sit-to-stand repetitions a patient can perform from a standard chair within 30 or 60 seconds, or the time it takes to complete a set number of repetitions, such as five in a row. It can also provide insight into functional status and neurophysiological performance [52].

7.15 Peripheral muscle strength testing

Since COPD is associated with muscle dysfunction, evaluating peripheral muscle function is essential. Muscle strength is typically measured as the maximal voluntary isometric force. The quadriceps femoris muscle is most commonly assessed, as it serves as a key indicator of lower limb strength [53].

7.16 Endurance training

Endurance training is the most common exercise approach for COPD patients, aiming to enhance aerobic capacity. While high-intensity training offers greater physiological benefits, many with severe COPD struggle to tolerate it due to symptoms like breathlessness and fatigue. As a result, alternative methods like interval training are gaining popularity, especially for advanced cases [54]. Endurance in COPD patients can be assessed using field tests like the 6-minute walk test (6MWT), incremental and endurance shuttle walking tests (ISWT, ESWT), 4-meter gait speed, 6-minute stepper test, sit-to-stand test (STST), and the cardiopulmonary exercise test (CPET), the gold standard for measuring VO_2 max. Endurance training improves hyperinflation, dyspnea, heart rate recovery, and peripheral muscle function. Recommended exercises include walking or cycling, targeting muscles such as the biceps, triceps, deltoids, latissimus dorsi, and pectorals [55].

7.17 Continuous versus interval training

Both continuous and interval training are effective in improving exercise capacity, quality of life, and peripheral muscle adaptations in patients with COPD. They lead to similar changes in muscle fibre composition, including a reduction in anaerobic fast-twitch (type IIb) fibres

and an increase in aerobic slow-twitch (type I) fibres, which enhances muscular endurance. However, interval training is often better tolerated in patients with more severe COPD, as it reduces symptoms like dyspnoea, lowers ventilatory and metabolic stress, and allows for longer total exercise durations. This improved tolerability can enhance motivation and long-term adherence to training. Tailoring exercise intensity—such as using 70% of peak workload and aiming for a perceived exertion of 4 to 6 on the Borg scale—can help optimize both training effectiveness and feasibility [56].

7.18 Cycle-based versus walking-based endurance training

Cycling-based endurance training minimilising dyspnoea and reducing the risk of oxygen desaturation during high-intensity exercise but it often requires access to specific equipment. Walking is a more accessible and cost-effective training option, especially in low-resource settings. Supervised, progressive walking programs may lead to greater improvements in walking endurance than stationary cycling. For patients unable to walk continuously for 10 minutes, reducing intensity by around 10% or incorporating longer walking intervals of 1–2 minutes may enhance feasibility and adherence. The choice of training should consider patient ability and resource availability [57].

7.19 Strength training

Resistance training can help reverse peripheral muscle dysfunction and ease the impact of COPD [58]. It is more effective than endurance training in increasing muscle mass and strength and typically causes less dyspnoea, making it more tolerable for many patients [59]. Combining resistance training with interval endurance training is especially beneficial for those with severe ventilatory limitations. Guidelines ATS/ERS recommend 2–4 sets of 6–12 repetitions at 50–85% of one-repetition maximum, performed 2–3 times per week. A simple method to set the right load is to choose a weight that leads to muscle fatigue within 6–12 reps.

7.20 Breathing retraining (or breathing exercises)

Breathing retraining aims to alter respiratory muscle recruitment in order to reduce dyspnoea and improve respiratory muscle performance, work on breathing, ventilation, lung volume, functional performance and activities in daily living. For example pursed-lip breathing, diaphragmatic breathing and expiratory muscle strengthening. These exercises can improve exercise tolerance in COPD patients, especially those unable to do regular exercise training [60].

8. Conclusion

Chronic obstructive pulmonary disease (COPD) leads to progressive respiratory dysfunction, muscle loss, and decreased exercise capacity, significantly impairing quality of life. Patients with COPD often experience varying degrees of activity limitation due to skeletal muscle dysfunction. However, physical activity remains crucial for all individuals with COPD. Exercise training and physical activity interventions should be integral, complementary components of pulmonary rehabilitation, each serving a unique purpose. Exercise training focuses on improving physical fitness and exercise capacity, particularly in the initial phase of rehabilitation. In contrast, physical activity interventions aim to promote long-term behavioural changes that encourage a more active lifestyle. Combining these approaches is essential for optimal patient management. Nonetheless, further research is needed to determine the best strategies for integrating these interventions—specifically regarding timing, duration, intensity, patient preferences, and selection criteria—to support sustainable, health-enhancing behaviours in individuals with COPD.

Disclosures

Author's contribution

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