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REVIEW

Exercise Prescription Training in Chronic Obstructive Pulmonary Disease: Benefits and Mechanisms

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Abstract: Exercise rehabilitation training has emerged as one of the most promising modalities for enhancing clinical outcomes and overall well-being in patients with chronic obstructive pulmonary disease (COPD). Distinct exercise prescriptions yield different clinical benefits in this population. Endurance training has been demonstrated to significantly improve exercise capacity, alleviate dyspnea, and enhance health-related quality of life metrics. High-intensity interval training offers a time-efficient approach to boosting cardiorespiratory fitness and metabolic function. Resistance training addresses progressive muscle atrophy through targeted myofiber recruitment, thereby augmenting musculoskeletal performance and translating to enhanced exercise tolerance in patients with COPD. Exercise-mediated rehabilitation attenuates COPD progression and mitigates acute exacerbation risks via multifactorial mechanisms such as mitigation of inflammatory responses, reduction of oxidative stress, and improvement of endothelial cell function. Elucidating the pathophysiological mechanisms underlying exercise-induced benefits will pave the way for precision rehabilitation protocols, ultimately advancing COPD disease management paradigms, refining patient-centered outcome measures, and achieving sustainable health optimization in this clinical cohort.

Keywords: chronic obstructive pulmonary disease, exercise training, pulmonary rehabilitation, inflammation, oxidative stress

Introduction

COPD is characterized by progressive airflow limitation, airway inflammation, and alveolar destruction, leading to impaired pulmonary function and significantly reduced quality of life.[1] As the third leading cause of death globally, COPD imposes significant economic and social burdens worldwide.[2,3]

Patients with COPD can benefit from pulmonary rehabilitation (PR), which has been shown to reduce symptoms and improve activity capacity, both of which are essential for enhancing quality of life.[2,3] Physical activity (PA), a key component of PR, encompasses exercise assessment and training therapy. PA is traditionally defined as any skeletal muscle movement that results in energy expenditure.[4] Physical training is a complex behavior influenced by various subjective (such as motivation and self-efficacy) and objective factors (such as environmental conditions and measurement tools).[5] Notably, patients with COPD have significantly lower levels of PA compared to age-matched healthy individuals,[6] highlighting the need for focused attention.

While extensive research confirms that exercise improves the general condition of patients with COPD, the optimal exercise intensity and the mechanisms by which it alleviates COPD symptoms require further investigation. This review summarizes advancements in exercise methods for COPD and explores the primary mechanisms underlying their effects on patients.

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Various Forms of Exercise Training

Endurance training (ET), high-intensity interval training (HIIT), resistance training (RT), flexibility exercise training, and neuromotor exercise training each contribute to overall physical fitness and improved health, particularly for individuals with chronic respiratory conditions such as COPD. ET focuses on enhancing aerobic efficiency, increasing the anaerobic threshold, and improving cardiovascular and pulmonary function.[7,8] HIIT alternates intense exercise bursts with recovery periods, enhancing cardiorespiratory fitness in patients with COPD.[9] It also incorporates inspiratory muscle training (IMT), which improves respiratory muscle strength and tolerance, alleviating some COPD symptoms,[10] though it may not significantly reduce dyspnea.[11] IMT, utilizing a specialized inspiratory muscle trainer, such as Expand-A-Lung and Powerbreathe, can significantly improve exercise capacity, health-related quality of life, and daily physical activity (DPA) in patients with inspiratory muscle weakness, especially when incorporated into a comprehensive exercise training program.[12] RT builds strength and muscle mass, enhancing quality of life,[13] while flexibility training improves joint mobility and reduces muscle damage.[14] Neuromotor training, such as yoga, Tai Chi, split-belt treadmill, and perturbation-based training, promotes balance and coordination, supporting the overall well-being of older adults.[15,16]

A multifaceted exercise program should be tailored to individual capabilities, incorporating a combination of these training modalities to optimize respiratory function and overall health. Regular participation in these exercises can significantly improve exercise capacity, muscle strength, flexibility, and quality of life. For patients with COPD, a comprehensive rehabilitation program integrating these exercise modalities is essential for effective disease management. A summary of the exercise types and recommendations is provided in Table 1.

Table 1 Exercise Types and Prescription Recommendations for Chronic Obstructive Pulmonary Disease

Type of Exercise	Examples	Focus/Benefit	Recommendations
Endurance training (aerobic training)[8]	Walking, running/jogging, swimming, biking, jumping rope, dancing, climbing stairs	Improves aerobic efficiency, lung function, cardiorespiratory fitness, and arterial stiffness	60–80% max work rate, 20–60 min/session, 3–5 times/week; gradually increase work rate and duration; include respiratory muscle endurance training
High-intensity interval training[11,17,18]	Running, dancing, rowing machines, stationary bicycling, climbing stairs	Enhances cardiorespiratory fitness, exercise capacity, and lung function	≥80%[19] max intensity, 2–3 days/week. Include a 5-min warm-up and 5–10 min cool-down; add high-intensity inspiratory muscle training for patients with moderate-to-severe COPD.
Resistance training (strength/ weight training)[13]	Free weights, weight machines, medicine balls, bands, kettlebells, elastic tubing, bodyweight exercise	Builds strength, increases muscle mass, and improves quality of life	60–70% 1RM, increase training load as tolerated; 2–4 sets/session, 8–12 repetitions, 2–3 times/week for ≥8 weeks
Flexibility exercise training[14,20]	Static stretching, dynamic stretching	Enhances flexibility and range of motion; mitigates muscle damage	Smooth and slow movements for 60s daily (10–30 s increments), at least 2 to 3 days/week
Balance and coordination training[15,16,21,22]	Balance balls, heel-to-toe walks, one-leg balance exercises, Tai Chi, dancing, split-belt treadmill, perturbation-based training, Sensorimotor training	Enhances sciatic nerve regeneration; improves balance, exercise tolerance, and health-related quality of life; decreases the risk of falls	≥45 min/session, 2–3 times/week for≥ 8 weeks

Abbreviations: 1RM, One-repetition maximum; COPD, chronic obstructive pulmonary disease.

Biomolecular Mechanism of Exercise Training

Promoting Inflammation Resolution

Systemic and airway inflammation are two major features of COPD and disrupt muscle synthesis, leading to muscle dysfunction.[17] These inflammatory processes significantly influence the progression and severity of COPD, highlighting the importance of managing both systemic and airway inflammation in chronic respiratory conditions.

The effects of exercise on inflammatory markers are complex. A substantial body of evidence suggests that exercise can improve the overall inflammatory response. However, some inflammatory markers remained unaffected by exercise (Table 2).

Although the precise mechanism by which exercise mitigates inflammation in patients with COPD remains unclear, recent research suggests that exercise can regulate inflammation by altering chemerin levels.[39] Chemerin is a novel adipokine that plays a major role in adipogenesis and lipid metabolism.[36] This is particularly important in diseases such as COPD, where the chemerin/CMKLR1 axis plays a role in both inflammation and metabolism. The inhibitory effect of exercise on chemerin expression provides valuable insights into exercise-induced pulmonary rehabilitation, which shows potential for clinical applications in COPD management. Additionally, exercise reduces STAT3 activation in various cell types involved in COPD pathogenesis, including peribronchial leukocytes, parenchymal leukocytes, and airway epithelial cells, thereby alleviating inflammation in patients with COPD.[37] Furthermore, Bufei Yishen Formula III, a classical traditional Chinese Medicine, exerts therapeutic effects by modulating pulmonary function, reducing inflammation, and enhancing immune regulation in chronic respiratory diseases. A study demonstrated that, when combined with exercise rehabilitation, it may synergistically reduce lung inflammation by inhibiting the EGFR/MAPK pathway[40] (Figure 1).

Effects of Exercise Training on Oxidative Stress

The effects of exercise training on oxidative stress in patients with COPD are complex and influenced by various factors, such as the type and intensity of the exercise and the severity of the disease. While some studies suggest that exercise may trigger oxidative stress by increasing airflow and exposure to pollutants, other research indicates that exercise has minimal effects on oxidative stress in COPD, with no significant changes observed in the ferric-reducing ability of plasma or thiobarbituric acid reactive substances after exercise.[41] Notably, most studies support the view that exercise positively mitigates oxidative stress in patients with COPD, as demonstrated by the findings summarized in Table 3.

The beneficial effects of exercise on oxidative stress in the diaphragm of patients with COPD involve multiple pathways. Clinical and animal studies by Lei et al[10] showed that sequential IMT alleviates oxidative stress by modulating the SOCS5/JAK2/STAT3 signaling pathway. Irisin, a muscle-derived hormone released during exercise, may protect against oxidative stress by activating the Nrf2 and HO-1 pathways, potentially mitigating cigarette smoke-induced emphysema in COPD[45] (Figure 1).

Others

Exercise has been shown to enhance muscle strength, endurance, and overall exercise capacity in patients with COPD by modulating the expression levels of myokines, including interleukin (IL)-6, IL-15, myostatin, irisin/FNDC5, insulin-like growth factor-1, and semaphorin 3A, thereby effectively addressing skeletal muscle dysfunction.[46,47] Additionally, moderate-intensity exercise improves mitochondrial function in the skeletal muscles of patients with COPD by increasing PGC-1α expression, enhancing ATP production through mitochondrial complex I, and regulating antioxidant capacity via the Keap1–Nrf2–ARE pathway.[48]

Endothelial dysfunction is another common issue in individuals with COPD and appears to be related to the severity of airflow limitation.[49] This dysfunction can significantly impair patients' abilities to engage in physical exercise but can be effectively mitigated through structured exercise programs. Notably, the benefits of exercise on endothelial function appear to be independent of supplemental oxygen during training, suggesting that exercising in regular room air might be even more beneficial for improving peripheral endothelial function. This effect might stem from the stimulatory impact of local hypoxic conditions, which trigger beneficial vascular adaptation. Exercise increases the production and bioavailability of nitric oxide (NO), which helps regulate shear stress and improves vascular function.[50] Furthermore,

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Table 2 Summary of Studies on the Impact of Exercise on Inflammation in COPD

Study ID	Design	Sample Size	Participants	Intervention	Biomarkers Related to Inflammation Change
F. Rodrigues et al[23]	Cross-sectional	21	Patients with COPD, clinically stable (past 4 weeks), exercise-capable, and able to complete health status questionnaires	Treadmill/Cycle ergometer until exhaustion; low-intensity activities maintained, no high-intensity exercise in prior 48 h	No significant changes in IFN-γ, IL-1β, IL6, IL8, TNF-α, TGF-β and iNOS at rest, 1 h, and 24 h post-exercise
Paul D. Loprinzi, PhD et al[24]	Cross-sectional	238	Former or current smokers with self-reported COPD and complete data on study variables	ActiGraph 7164 accelerometers worn on right hip for 7 days (LPA, MVPA, TPA)	LPA, MVPA, and TPA inversely associated with WBC and neutrophil count
Olaf holz1, Stefan roepcke et al[25]	Prospective clinical	46 (moderate COPD: 23; age- and sex-matched healthy controls: 23)	Current smokers with a smoking history of at least 10 pack-years	Peak work capacity and constant-load exercise	IL6, sICAM1, CRP ↑; MPO ↓; Healthy smokers: IL6, CRP ↑ > COPD; COPD: IL6, MPO, CRP, sICAM1, VWF, LTB4 changes/min > controls. Recovery: IL6 ↑ in healthy smokers only
Gilson Pires Dorneles et al[26]	Cross-sectional	41 (moderate:14; severe: 14; very severe: 13)	Patients with COPD	6MWT, patients walked on a 30-m track (10-m markings). Stopped for symptoms (eg, chest pain, respiratory distress, muscle pain). Distance was measured using a 20-m tape	IL6: ↑ in very severe > severe (post-test). IL4: ↓ in severe < very severe/moderate (pre-test); ↑ in moderate > severe (post-test). Very severe: IL4 pre-post ↑ effect
Ryrsø CK[27]	Cross-sectional	38 (RT: 15; ET: 15; healthy sedentary participants: 8)	Patients with moderate-to-severe COPD and healthy controls.	RT and ET, both groups completed 35 min of supervised exercise three times a week for 8 weeks.	TNF-α ↑; IL-1β, IL6, IL8, and IL18 unchanged
Juliana S. Uzeloto et al[28]	RCT	23 (PEG: 15; RPG: 8)	Participants with stable COPD	PEG or RPG	IL-8 ↓; IL-13, TNF-α↑ in $CD4^+$ T lymphocytes
Shehab M et al[29]	RCT	80 (AE group: 40; no exercise training: 40)	Patients with moderate severity of COPD	Aerobic exercise on a treadmill	TNF-α, IL-4, IL-6, and CRP ↓ in exercise group
Bruna S. de Alencar Silva et al[30]	RCT	48 (EBG: 14; ETG:18; MG:16)	Clinically stable patients with moderate to severe COPD	ETG Group: Used specially prepared chairs with elastic tubes (resistance levels 200–204); EBG Group: Utilized elastic bands with resistance categorized by color: yellow (lowest) to black (highest); MG Group: Employed traditional weight machines for upper limb exercises	IL-6: ↓ after 12 weeks, group differences and interaction; TNF-α: ↓ after 12 weeks, no group differences; IL-10: ↓ in EG vs MG; IL-15: ↓ after 12 weeks, no group/interaction effects; IL-10/TNF-α Ratio: Higher in EG vs MG

Alex R. Jenkins[31]	Prospective cohort	67 (COPD, Phase 1: 40; COPD, Phase 2: 27)	Patients with COPD were recruited with no exacerbations for ≥4 weeks and no antibiotic or steroid treatment during this period	8-week exercise program (cardiorespiratory + muscular strength)	Acute exercise (Phase 1): Fibrinogen, CRP, and neutrophil markers unchanged; leukocytes, neutrophils, and immature neutrophils increased. Pulmonary rehab (Phase 2): Fibrinogen response increased post-exercise
Chun-Hua Wang, Pai-Chien Chou et al[32]	Clinical pilot	26 (control:14; mobile phone:12)	Patients with COPD	Mobile phone-guided ET (80% of the maximal capacity) vs control group	Reduced CRP and IL8 in mobile phone group; no changes in IL6 and TNF-α
Shehab M et al[33]	Prospective cohort	100 (AE: 50; RT:50)	Patients with COPD	AE vs RT (3months)	TNF-α, Il-2, IL-4, IL-6, and CRP were reduced in both groups
Wei Liu et al[34]	RCT	102 (control: 26, TBRS: 25, TC:26, TBRS-TC: 25)	Patients with mild to severe stable COPD	TC, TBRS, TBRS-TC	IL-6, IL-8, TNF-α, and copeptin were statistically significant between groups. Greater reduction in IL8 and TNF-α in TBRS-TC
Xishuai Wang et al[35]	Animal experiment	30 (control:10; COPD:10; COPD +AE:10)	Male ICR mice	COPD: LPS via intratracheal injection; COPD + exercise: LPS via intratracheal injection and low-intensity exercise using a six-lane treadmill	BALF: total cell count, neutrophils, macrophages, TGF-β, IL-1β, TNF-α ↓; IL10 and CXCL1 ↑; lymphocytes, eosinophils, IL17 unchanged; Serum: TNF-α ↓, IL10 ↑
Hidetaka IMAGITA et al[36]	Animal experiment	21 (control: 7; pulmonary emphysema group: 7; emphysema + exercise group: 7)	9-week-old male Wistar rats	Treadmill running in CS and LPS-induced pulmonary emphysema	No significant difference in IL-1β, IL6, TNF-α, IL4, and IL10
Maysa Alves Rodrigues Brandao-Rangel et al[37]	Animal experiment	64 (control:16; exercise:16; COPD:16 COPD+Exercise:16)	Male C57Bl/6 mice	Treadmill aerobic training in CS-induced COPD mice	BALF: IL10 ↑; total cells, lymphocytes, neutrophils, IL-1β, CXCL1, IL-17, and TNF-α ↓; Serum: IL10 ↑; total leukocytes, neutrophils, lymphocytes, monocytes, IL-1β, CXCL1, IL-17, and TNF-α ↓
Panpan Liu, Hongchang Gao et al[38]	Animal experiment		C57BL/6 male mice	Motor-driven treadmill exercise in CS-induced COPD mice	Reversed TNF-α, IL-1β, and IL6 expression

Abbreviations: COPD, Chronic obstructive pulmonary disease; RCT, randomized controlled clinical trial; LPA, light-intensity physical activity, 100–2019 counts per min; MVPA, moderate-to-vigorous physical activity, ≥2020 counts per min; TPA, total physical activity, ≥100 counts per min; sICAM1, soluble intercellular adhesion molecule 1; CRP, C-reactive protein; MPO, myeloperoxidase; VWF, von Willebrand factor; LTB4, leukotriene B4; 6MWT, 6-min walk test; RT, resistance training; ET, endurance training; AE, aerobic exercise; PEG, physical exercise group; RPG, respiratory physiotherapy group; EBG, Elastic band group; ETG, elastic tube group; MG, weight machines equipment group; TBRS, total body recumbent stepper; TC, Tai Chi; TBRS-TC, combined TBRS exercise and Tai Chi; CS, cigarette smoke; LPS, lipopolysaccharide solution.

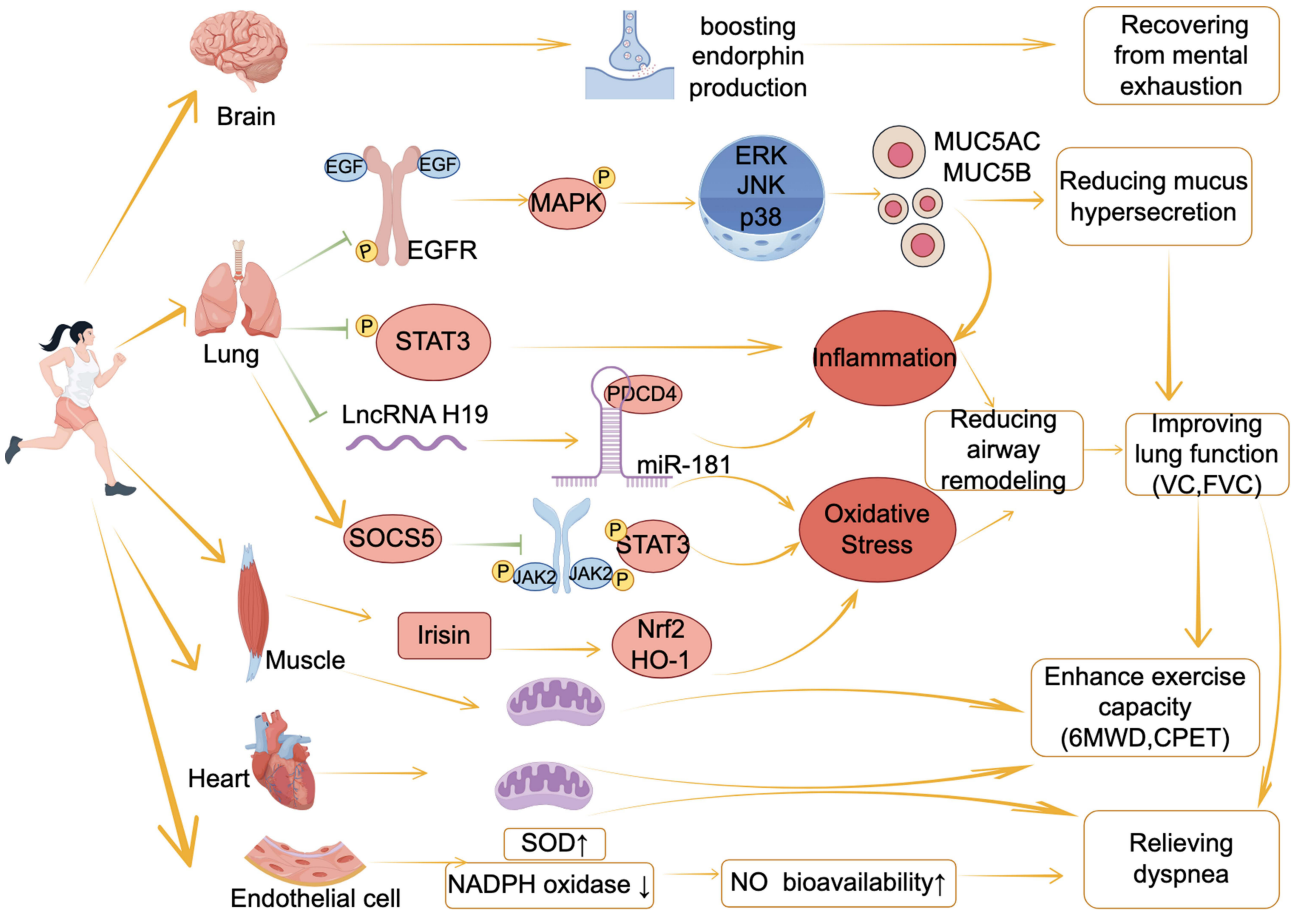

Figure 1 Mechanisms of exercise-induced improvement in COPD symptoms (created using Figdraw).
Abbreviation: COPD, chronic obstructive pulmonary disease.

a randomized, double-anonymized, sham-controlled study confirmed that the application of noninvasive positive pressure ventilation during high-intensity exercise acutely modulates endothelial functions in patients with COPD-related heart failure.[51]

Table 3 Effects of Different Exercise Interventions on Oxidative Stress in COPD

Study	Participants	Exercise Type	Changes in Oxidative Stress Markers	Main Conclusion
Mayalen Valero-Breton et al[42]	Patients with COPD	12-week eccentric (ECC) and concentric (CONC) cycling training	**CONC**: ↑ TAC, ↓TBARS, ↓ GPX activity; **ECC**: ↓ GPX, no significant changes in TBARS, SOD, or CAT	CONC training significantly improved antioxidant capacity; ECC had weaker effects on oxidative stress
Denisse Valladares-Ide et al[43]	Patients with COPD	Moderate-intensity CONC (CONC-M), moderate-intensity ECC (ECC-M), high-intensity ECC (ECC-H)	**MDA**: ↓ after CONC-M (−28%), ECC-M (−14%), ECC-H (−17%); **GPX**: No change; **EBC H_2O_2, NO^{2-}, pH**: No change	Exercise decreased oxidative stress in the plasma of patients with COPD at moderate stage of the disease

(*Continued*)

Table 3 (Continued).

Study	Participants	Exercise Type	Changes in Oxidative Stress Markers	Main Conclusion
Wang et al[35]	LPS-induced COPD mice	Aerobic exercise	↓ MDA, MPO; ↑ SOD, GSH	Exercise reduced oxidative stress and improved antioxidant capacity
Renata Tiscoski Nesi et al[44]	CS-induced COPD mice	Swimming	Prevented increases in hydroxyproline content, SOD activity, CAT activity, 2-thiobarbituric acid reactive species, and carbonyl group content in lung tissue	Exercise mitigated CS-induced oxidative damage
Yirou Lei et al[10]	Patients with COPD + CS-induced COPD mice	NIPPV, IMT, and sequential NIPPV + IMT	↓ ROS, MDA; ↑ SOD, GSH	Sequential NIPPV + IMT reduced oxidative stress and improved COPD outcomes

Abbreviations: TAC, total antioxidant capacity; TBARS, barbituric acid reactive substances; GPX, glutathione peroxidase activity; SOD, superoxide dismutase; CAT, Catalase; MDA, malondialdehyde; EBC, exhaled breath condensate; H_2O_2, hydrogen peroxide; LPS, lipopolysaccharide; GSH, glutathione; CS, Cigarette smoke; NIPPV, noninvasive positive pressure ventilation; IMT, inspiratory muscle training.

The lactate threshold—the point where lactate production surpasses clearance—occurs at lower exercise intensities in patients with COPD due to impaired oxygen delivery, leading to early lactic acid buildup, breathlessness, and fatigue. Tailored exercise training can raise this threshold, allowing patients to exercise at higher intensities before symptoms arise, improving their physical capacity and disease management.[52] Excess post-exercise oxygen consumption (EPOC), the elevated oxygen uptake post-exercise, is less pronounced in patients with COPD due to limited lung capacity and impaired oxygen use. Managing intensity is key to optimizing EPOC while minimizing respiratory strain. Activities like walking, cycling, and aquatic exercises are ideal, as they promote recovery while accommodating reduced respiratory function.[53]

Current research supports the idea that exercise directly influences pulmonary function by modulating inflammation and oxidative stress. Additionally, exercise enhances skeletal muscle and endothelial function, thereby significantly influencing the pathophysiology of COPD. This multifaceted effect underscores the importance of incorporating physical activity into therapeutic strategies to improve health outcomes in individuals with this debilitating respiratory condition.

Exercise Training Assessment in Clinical Practice

Acute Exacerbations of Chronic Obstructive Pulmonary Disease (AE-COPD)

GOLD 2023 has highlighted the potential of PR in reducing hospitalization rates among patients with recent exacerbations (within 4 weeks of their previous hospitalization). Initiating exercise training within 3 days of hospitalization likely enhances exercise capacity and improves overall quality of life.[54] A clinical study revealed that Medicare beneficiaries who completed appropriate exercise training within 90 days of being discharged for COPD experienced a reduction in rehospitalization rates within a year.[55] A retrospective study demonstrated that combined respiratory and exercise training significantly lowered the rates of acute exacerbation, rehospitalization, and mortality compared to controls.[56] A randomized controlled trial[57] showed that patients receiving rigorous supervised training had fewer readmissions and a longer time to first readmission than those receiving usual care. Additionally, RT was safer, more feasible, and more effective than ET for improving muscle function in patients with severe AECOPD. However, ET, when performed after the acute phase of AECOPD, can improve functional exercise tolerance.[58]

Dyspnea

Dyspnea is a prominent symptom among individuals with COPD. The severity of dyspnea can be assessed using various tools, including the modified Medical Research Council (mMRC) scale, Borg Dyspnea Scale, Visual Analogue Scale, Numeric Rating Scale, and the Baseline Dyspnea Index/Transition Dyspnea Index. GOLD 2023 indicates that PR improves dyspnea in patients with COPD. Exercise reduces central ventilatory drive, improves dynamic ventilatory mechanics, and enhances respiratory muscle function, thereby alleviating dyspnea.[59] Additionally, a decrease in the diaphragm activation ratio (measured via electromyography) during maximal inspiration explains the reduction in perceived dyspnea, even under prolonged high ventilation and mechanical loading during exercise.[60] Upper extremity exercise and IMT alleviate dyspnea in patients with COPD,[61] with the ET group reporting significantly lower mMRC scores compared to controls.[62] Additionally, a 4-week lower limb resistance training program effectively reduced exertional dyspnea, while both elastic tube and conventional resistance training reduced mMRC scores by 30%.[63] However, the effectiveness of HIIT in reducing dyspnea remains debated. A study[64] indicated a reduction in breathlessness, while a subsequent meta-analysis[11] of 689 participants suggested that HIIT improves pulmonary function, exercise capacity, and quality of life without significantly affecting the sensation of breathlessness. This inconsistency is noteworthy, as the larger sample size of the meta-analysis may provide a more reliable conclusion regarding HIIT's impact. Furthermore, sensitivity analyses indicated that the effect on dyspnea could vary depending on the study design and measurement tools used. Therefore, HIIT may not be suitable for all patients with COPD, particularly those with significant dyspnea. Therefore, a more appropriate exercise method should be selected for patients with COPD who experience significant dyspnea.

Lung Function

Spirometry is an inexpensive, noninvasive, easily accessible, and repeatable diagnostic test widely used for diagnosing and monitoring COPD. Commonly reported parameters in pulmonary function tests include forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC), and the ratio of these measurements (FEV1/FVC). A meta-analysis of 20 RCTs involving 962 patients with COPD demonstrated that HIIT improved peak oxygen consumption (standardized mean difference [SMD] = 0.30, 95% confidence interval [CI]: 0.14–0.46), peak minute ventilation (SMD = 0.26, 95% CI: 0.05–0.47), and peak work rate (SMD = 0.34, 95% CI: 0.17–0.51) in these patients. However, no significant effects were observed regarding the FEV1/FVC ratio or St. George's Respiratory Questionnaire scores ($P > 0.05$). Additionally, no significant difference was found in the FEV1/FVC ratio between the HIIT and control groups.[64] A clinical study revealed that ET combined with resistance training (ERT) and ET alone increased vital capacity (VC), FVC, and forced expiratory flow at 25%–75% (FEF25%–75%), while only ET and ERT increased maximum voluntary ventilation (MVV); however, none of these interventions significantly affected FEV1 or FEV1/FVC in this study.[65] Notably, high-volume RT may impair lung function indices due to reduced engagement of ventilatory and respiratory muscles.[66] Nonetheless, a short-term (12 weeks) high-intensity RT program for patients with moderate to severe COPD resulted in a significant 5.3% increase in FEV1 and a trend towards improved daily peak-flow measurements.[67] Therefore, selecting the appropriate exercise intensity and frequency for patients with varying degrees of severity is crucial. Several other indicators reflecting ventilation and gas exchange function, such as MVV, PEF, and FEF25%–75%, can also be assessed simultaneously.

Exercise Ability

The Six-Minute Walk Distance (6MWD) is a widely utilized clinical tool for evaluating the functional exercise capacity of individuals with COPD. The 6MWD test provides valuable information about the severity of COPD, its impact on exercise tolerance, and the effectiveness of interventions such as pulmonary rehabilitation and pharmacotherapy. ET, RT, combined ET and RT, HIIT, and respiratory training have all been shown to improve 6MWD.[56,64,68] Cardiopulmonary exercise testing (CPET) is a comprehensive, noninvasive procedure that evaluates the integrated functions of the heart and lungs during physical activity. It has consistently been regarded as the gold standard for assessing exercise capacity in patients with COPD,[69] especially those with pulmonary artery hypertension. High-intensity interval cycle ergometer training for 6 weeks resulted in significant reductions in lactate values, minute ventilation, ventilatory equivalent for oxygen, heart rate, oxygen uptake, and carbon dioxide output.[70] Additionally, notable differences were observed between

active (DPA ≥ 30 min a day, 5 days a week) and inactive (DPA < 30 min a day, 5 days a week) patients with COPD in their cardiovascular responses to exercise, such as peak O_2 pulse, double product reserve, and heart rate recovery. However, no significant variations were observed in their ventilatory response or operational volumes during exercise.[71] Therefore, both 6MWD and CPET are convenient and practical tools for monitoring COPD. The 6MWD can be used to assess the benefits of exercise, while CPET results allow for individualized adjustments to training intensity, ultimately optimizing exercise programs for patients.

Health-Related Quality of Life

Various methods are available for evaluating quality of life in patients with COPD, including the St. George's Respiratory Questionnaire (SGRQ), COPD Assessment Test (CAT), Chronic Respiratory Questionnaire (CRQ), and Clinical COPD Questionnaire (CCQ). The use of ET resulted in a significantly lower CAT score compared to the control group in a study.[62] In another study, both the RT and ET groups significantly improved CRQ scores compared to the control group, with no notable differences between the two groups.[70] Conversely, conventional RT was more effective in improving CRQ scores than ET using elastic tubes.[63] Short-term (12 weeks) high-intensity RT significantly improved SGRQ scores in patients with moderate to severe COPD.[67] Additionally, HIIT significantly improved the quality of life in patients with COPD, although SGRQ scores did not significantly differ between the HIIT and control groups.[64] Neuromotor exercise training, specifically sensorimotor training, has significantly reduced pain compared to usual care. It is recommended that the training intensity be moderated to ensure participants do not experience excessive fatigue. Furthermore, a frequency of two to three sessions per week is suggested for optimal results. Additionally, whole-body vibration therapy has demonstrated promise in improving sleep quality and is particularly effective in reducing depressive symptoms. Aerobic exercises like brisk walking, swimming, cycling, and dancing help reduce stress, improve mood, and enhance cognitive function by boosting endorphin production, increasing blood flow to the brain, enhancing prefrontal oxygenation, and elevating circulating neurotrophins.[72]

Conclusion

In conclusion, exercise interventions, including ET, HIIT, and RT, offer significant benefits for patients with COPD by improving exercise capacity, reducing dyspnea, and enhancing quality of life. These modalities not only improve physical function but also reduce inflammation and oxidative stress, potentially slowing disease progression and reducing exacerbation risk. Monitoring inflammatory markers like IL-4, IL-6, IL-8, and IL-10 can help assess intervention efficacy.

To optimize patient outcomes, individualized exercise prescriptions should be tailored to disease severity, comorbidities, and physical capability, with a focus on gradual progression in intensity and duration. Supervision is essential for patients with more severe disease, ensuring safety during exercise. Clinicians should integrate exercise with pharmacological treatments and pulmonary rehabilitation for a holistic management approach.

Future research should aim to elucidate the molecular mechanisms behind exercise-induced improvements, standardize exercise protocols, and evaluate long-term adherence and efficacy. By incorporating exercise as a core component of COPD management, clinicians can significantly improve the functional status and quality of life of their patients.

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Disclosure

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