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REVIEW ARTICLE

Most relevant training modalities currently in use to treat skeletal muscle dysfunction in chronic obstructive pulmonary disease

Sneha Chakraverty[1], Davina Simoes[2], and Ioannis Vogiatzis[1]*

[1]School of Sports, Exercise and Rehabilitation, Faculty of Health and Wellbeing; [2]School of Geography and Natural Sciences, Faculty of Science and Environment. Northumbria University, Newcastle upon Tyne, UK

Abstract

Skeletal muscle dysfunction and sarcopenia are key systemic manifestations of COPD. These changes contribute to exercise intolerance, reduced daily physical activity levels, and health-related quality of life, whilst they constitute independent predictors of morbidity and mortality. The aim of this review is to provide a narrative review of the effects of different exercise training modalities on peripheral muscle structure and function in patients with COPD. Evidence from randomised trials and mechanistic studies evaluating resistance, endurance (continuous and interval) training modalities, and combined endurance and resistance programmes was synthesised. Outcomes include muscle fibre cross-sectional area, fibre-type composition, oxidative capacity, quadriceps muscle strength, and lean body mass. Resistance and combined training programmes promote muscle strength and muscle hypertrophy, whilst endurance training alone enhances oxidative capacity. Biopsy-informed evidence remains limited and heterogeneous. Resistance-based interventions are most effective for restoring muscle size and strength, supporting individualised exercise prescriptions to counteract sarcopenia and optimise functional outcomes.

Keywords: *COPD. Sarcopenia. pulmonary rehabilitation. Resistance and endurance training.*

Introduction

Chronic respiratory diseases (CRDs), including chronic obstructive pulmonary disease (COPD), interstitial lung disease (ILD), and bronchiectasis, are increasingly recognised as multi-system conditions, with skeletal muscle dysfunction and sarcopenia representing key systemic manifestations that influence symptoms, exercise tolerance, and prognosis.[1,2] In COPD, quadriceps muscles commonly exhibit reduced cross-sectional area, fibre-type shifts toward glycolytic phenotypes, diminished oxidative capacity, and impaired contractile function.[3,4] These changes contribute to exercise intolerance, reduced physical activity, and poorer health-related quality of life (HRQoL), and are independent predictors of morbidity and mortality.[5,6]

Exercise training is a cornerstone of pulmonary rehabilitation (PR) and is strongly endorsed by international guidelines as an evidence-based intervention to counteract muscle wasting and dysfunction.[7-9] A range of training modalities, including resistance, endurance (continuous and interval) training modalities, and combined endurance and resistance programmes have been applied to improve muscle strength, fibre cross-sectional area, and functional capacity.[7,10] Meta-analyses confirm that resistance training enhances muscle strength and lean body mass, while endurance training modalities improve aerobic capacity and HRQoL.[7,11] However, the magnitude and nature of muscle adaptations appear to depend on modality, intensity, and duration, and mechanistic evidence from muscle biopsies remains limited, with small heterogeneous

***Correspondence:**
Sneha Chakraverty
E-mail: sneha.chakraverty@northumbria.ac.uk

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studies reporting variable fibre-type redistribution and oxidative enzyme responses.[12-14]

Skeletal muscle dysfunction is a major determinant of exercise intolerance, frailty, and reduced survival in COPD and other CRDs. While numerous exercise interventions have been studied, direct mechanistic evidence from muscle biopsies and imaging remains sparse, limiting our understanding of how specific training modalities mitigate muscle wasting and sarcopenia. Furthermore, biopsy-informed studies provide critical insights into fibre-level adaptations, such as changes in fibre cross-sectional areas, fibre-type composition, and satellite cell activity, which cannot be inferred from strength gains alone.

Given the clinical importance of skeletal muscle function and heterogeneity of exercise training protocols, this review aims to summarise the effects of different training modalities on peripheral muscle structure and function, integrating biopsy-derived measures, quadriceps strength, and muscle mass outcomes. Specifically, the objectives are to:

- Identify training modality-specific adaptations and dose response characteristics (intensity, progression, duration).
- Link cellular remodelling to clinically meaningful outcomes, including exercise tolerance, physical activity, and HRQoL.
- Inform evidence-based exercise prescriptions for pulmonary rehabilitation and guide future research aimed at optimising interventions to prevent or reverse sarcopenia in COPD and related respiratory conditions.

Methods

Approach

This review adopts a narrative synthesis approach to provide a comprehensive overview of exercise interventions currently used to address skeletal muscle dysfunction and sarcopenia, primarily in COPD. Unlike systematic reviews, which focus on quantitative pooling, this narrative review aims to integrate findings from diverse study designs to explore mechanistic insights and practical implications.

Scope and selection criteria

Studies were selected to capture the range of exercise modalities applied in clinical and research settings, including eccentric, concentric, continuous, interval, resistance, and combined training programmes. Eligible studies:

- Included adults diagnosed with COPD.
- Investigated exercise interventions targeting skeletal muscle health.
- Reported outcomes related to muscle structure (biopsy-derived fibre cross-sectional area (CSA), fibre-type composition, oxidative enzyme activity), muscle mass (quadriceps CSA, lean body mass), and functional performance (strength, endurance).

Mechanistic studies, randomised controlled trials (RCTs), and controlled clinical trials were prioritised. Reviews and meta-analyses were consulted for contextual understanding. Exclusion criteria included animal studies, case reports, and non-exercise interventions.

Search strategy

A comprehensive literature search of RCTs, experimental studies and observational studies published between 1995 and 2025 in English was conducted in PubMed, Embase, and Cochrane Library, supplemented by manual screening of reference lists. Search terms combined disease-specific keywords (“COPD”, “chronic respiratory disease”) with intervention-related terms (“exercise training”, “resistance training”, “interval training”, “pulmonary rehabilitation”) and mechanistic outcomes (“muscle biopsy”, “fibre type”, “oxidative capacity”, “quadriceps strength”).

Data extraction and synthesis

Data from included studies were extracted narratively, focusing on:

- Training modality characteristics (type, intensity, duration).
- Muscle-level adaptations (fibre hypertrophy, oxidative enzyme activity, fibre-type shift).
- Functional outcomes (muscle strength, endurance, exercise capacity). Findings were organised by modality to highlight mechanical and metabolic stimuli and their impact on locomotor muscle adaptation. The synthesis emphasises clinical relevance, mechanistic insights, and implications for exercise prescription.

Quality considerations

Although formal risk-of-bias scoring was not applied, study design, sample size, and methodological rigor were considered when interpreting findings. Limitations and gaps in evidence are discussed to guide future research.

Table 1. Characteristics and study design of exercise training interventions examining skeletal muscle adaptations in COPD

Study (author, year)	Training modality	Sample size	Duration	Intensity	Study design	Biopsy-derived outcomes	Muscle mass/ CSA	Functional outcomes
Puente-Maestu, 2000[21]	Continuous endurance	n = 12	8 weeks	Moderate intensity (typically 60% Wpeak)	Mechanistic, non-randomised	Not reported	Not reported	↑ Aerobic capacity
Vogiatzis, 2005[20]	Interval cycling	n = 19	10 weeks	High-intensity intervals (work bouts at 100% Wpeak; recovery at 40%)	Mechanistic comparative RCT	↑ Type I & IIa fibre CSA	↑ Quadriceps CSA	↑ Peak work rate
Maltais, 1996[22]	Endurance training	n = 11	3 times per week; 12 weeks	High load	Mechanistic, non-randomised	↑ Oxidative enzyme activity in vastus lateralis muscle	Not reported	↑ Strength, skeletal muscle oxidative capacity
Bernard, 1999[23]	Combined aerobic + resistance	n = 45	3 times per week; 12 weeks	High intensity-80% of peak work-rate	RCT	Not reported	↑ Quadriceps CSA	↑ Strength and exercise capacity
MacMillan, 2017[24]	Eccentric cycling	n = 15	3 times per week; 10 weeks	60-80 % of peak work-rate	Mechanistic experimental	No significant CSA change	↑ Relative thigh mass	↑ Isometric strength
Bourbeau, 2020[18]	Eccentric vs concentric cycling	n = 24	3 times per week; 10 weeks	60-80 % of power peak output	RCT	No biopsy	Not reported	↑ Muscle strength
Iepsen, 2016[19]	Endurance vs resistance	n = 30	3 times per week; 8 weeks	ET: moderate adjusted to level 14-15 on the Borg Scale, RT: 30-40% 1 RM	Pilot RCT	Fibre-type shift	RT > ET	↑ Strength (RT)
Troosters, 2010 (AECOPD)[17]	Resistance training	n = 40	One session per day during the entire hospitalisation period	> 70% of 1 RM	RCT	↓ Myostatin and ↑ Myogenin/MyoD ratio	Preserved muscle mass	↑ Strength, exercise capacity
Nyberg, 2015[16]	Low-load/high-rep RT	n = 44	3 times per week; 8 weeks	Low load (30% 1 RM, high repetition)	Multicentre RCT	No biopsy	Not reported	↑ Endurance/ function
Nyberg, 2021[15]	Low-load/high-rep RT	n = 33	3 times per week; 8 weeks	Low-moderate load RT, high repetition	RCT	No significant CSA	Modest ↑ mass	↑ Exercise capacity, muscle endurance, health status

Table 2. Comparative effects of exercise modalities on skeletal muscle: evidence from RCTs

Modality	Key RCTs	Primary adaptation	Effect on muscle size	Fiber type impact
Resistance training	Troosters 2010[17]; Nyberg 2015[16], 2021[15]; Iepsen 2016[19]	↑ Strength, ↓ Hypertrophy	Significant ↑ Quadriceps CSA	↑ Type II fibre CSA
Eccentric training	Bourbeau 2020[18]	↑ Strength at low ventilatory load	↑ Relative muscle mass	No consistent hypertrophy
Endurance vs resistance	Iepsen 2016[19]	Modality specific	RT > ET for mass	↑ Type IIa proportion (ET)
Interval training	Vogiatzis 2005[20]	Mixed metabolic & mechanical	Moderate ↑ CSA	↑ Type I & II fibre CSA

Table 3. Comparative effects of exercise modalities on skeletal muscle: evidence from Non-RCTs

Modality	Key Non-RCTs	Primary adaptation	Effect on muscle size	Fibre type impact
Endurance Training	PuenteMaestu 2000[21], Maltais 1996[22]	↑ Oxidative enzymes (Maltais 1996)[22]	Minimal hypertrophy	No biopsy data
Combined training	Bernard 1999[23]	Balanced stimulus	↑ Thigh CSA (CT)	Fibre type shift not assessed

Results

Overview of included studies

Ten exercise intervention studies investigating skeletal muscle adaptations in COPD were included, comprising seven RCTs[15-21] and three controlled mechanistic or non-randomised studies[22-24] (Table 1). The RCTs primarily evaluated the clinical efficacy and feasibility of resistance, endurance, eccentric, and low-load/high-repetition training modalities, reporting outcomes related to quadriceps muscle strength, muscle mass, and functional performance (Table 2). In contrast, mechanistic studies provided detailed biopsy-derived insights into fibre-level adaptations, including changes in fibre CSA, fibre type composition, and oxidative enzyme activity (Table 3).

Across studies, interventions encompassed continuous and interval endurance training, resistance training, combined endurance and resistance programmes, and eccentric cycling modalities. Sample sizes were modest (n = 11-44), and intervention duration typically ranged from 8 to 12 weeks, with supervised training frequencies of 2-3 sessions per week. While RCTs offered higher-level evidence for functional and clinical outcomes, mechanistic studies complemented these findings by elucidating modality-specific structural and metabolic adaptations within the quadriceps muscle (Table 2 and 3).

Comparative effects of training modalities on skeletal muscle adaptations

Endurance training

Continuous endurance exercise in COPD predominantly enhances oxidative and metabolic function rather than inducing structural muscle remodelling. Puente-Maestu showed improvement in gas-exchange kinetics and endurance time after 8 weeks, but did not report muscle biopsy data or fibre hypertrophy, indicating that structural changes were minimal.[21] Similarly, Maltais et al. observed substantial increases in oxidative enzyme activity (citrate synthase and HADH) in vastus lateralis muscle after 12 weeks of endurance cycling, confirming enhanced oxidative capacity.[22] However, no increases in muscle fibre cross-sectional area were seen, and glycolytic enzyme activities remained unchanged, demonstrating that endurance training improves metabolic function without promoting fibre hypertrophy.

Collectively, these findings confirm that continuous endurance exercises drive metabolic adaptations rather than structural gains, particularly in type II fibres, which require resistance or high-force training stimuli for hypertrophic adaptation (Table 2 and 3).

Alternative modalities

Interval training (IT) and resistance-based programs elicited more pronounced structural adaptations.

Table 4. Summary of exercise training modalities for skeletal muscle dysfunction in chronic respiratory diseases

Modality	Description	Primary Stimulus	Reported muscle adaptations	Functional outcomes
Resistance training	High-load, low-repetition exercises targeting major muscle groups	Mechanical overload	↑ Fibre CSA (type I & II), ↑ strength, ↑ satellite cell activity	Improved quadriceps strength, walking distance
Endurance training	Continuous aerobic exercise (e.g., cycling, walking)	Oxidative/metabolic	↑ Oxidative enzyme activity, minimal hypertrophy	↑ VO_2 peak, ↑ exercise tolerance
Interval training	Alternating high-intensity bouts with recovery	Mixed mechanical & metabolic	↑ Oxidative capacity, partial fibre-type shift, improved mitochondrial function	↑ Endurance, ↑ functional capacity
Combined training	Resistance + endurance in the same programme	Mechanical + oxidative	↑ Strength and oxidative enzymes, modest hypertrophy	↑ Strength and aerobic capacity
Eccentric training	Emphasises lengthening contractions	High mechanical tension	↑ strength with lower ventilatory cost	↑ Strength, ↑ perceived exertion
Concentric training	Emphasises shortening contractions	Moderate mechanical load	↑ Strength, less hypertrophy than eccentric	↑ Strength, ↑ mobility

Vogiatzis et al. demonstrated significant hypertrophy in type II fibres and increased quadriceps muscle CSA after ten weeks of interval cycling.[20] Troosters investigated resistance, and although biopsies confirmed shifts in anabolic markers, no structural hypertrophy or fibre-type data were collected.[17] Combined aerobic and resistance training demonstrated increased thigh muscle CSA measured by CT following 12 weeks of combined training.[23] However, no muscle biopsies were collected, so fibretype-specific adaptations or fibre hypertrophy cannot be inferred (Table 2 and 3).

Comparison between different alternative training modalities

Eccentric versus concentric ergometer training

Two studies (one RCT and one experimental study) evaluated eccentric exercise training (EET) versus conventional concentric exercise training (CET):

- MacMillan et al.: in patients with severe COPD, EET resulted in significant increases in isometric quadriceps peak strength and relative thigh muscle mass compared with CET over 10 weeks. EET was performed at a threefold higher mechanical workload despite lower perceived exertion. However, vastus lateralis fibre CSA did not significantly change, and mitochondrial markers showed no improvement, indicating no biopsy-verified hypertrophy or mitochondrial enhancement.[24]
- Bourbeau et al.: this study evaluated submaximal eccentric cycling in people with COPD and found that ECC allows substantially higher mechanical workloads at similar or lower cardiopulmonary strain than CET, including lower oxygen uptake (VO_2), lower ventilation, and less dyspnoea for matched loads. Although this study did not assess muscle strength over a training period, it demonstrated that COPD patients can tolerate much higher workloads eccentrically than concentrically, helping to explain why eccentric training often produces superior strength outcomes in longer trials.[18]

Interpretation: eccentric exercise enables high mechanical loading at a low ventilatory cost, making it particularly advantageous for COPD patients with ventilatory limitation. Across the available studies, EET improves strength and relative muscle mass, but biopsy evidence does not consistently show fibre hypertrophy or mitochondrial adaptation, suggesting that improvements may arise primarily from neuromuscular (motor unit recruitment, rate of force development) or architectural adaptations rather than classic fibre level hypertrophy. This interpretation is fully consistent with the current evidence base (Table 2 and 3).

Endurance versus resistance (and combined) training

- Iepsen et al.: endurance training (ET) promoted a more oxidative phenotype, reflected by a reduction in type IIa fibre proportion, without significant CSA changes. In contrast, resistance training (RT) produced greater strength and mass improvements, though no consistent fibre hypertrophy was observed.[19]
- Systematic evidence also shows that combining RT with ET yields greater strength gains than ET alone.[25]

Interpretation: ET primarily induces metabolic/mitochondrial adaptations, while RT drives strength and mass. Short-term interventions rarely show clear biopsy-level hypertrophy despite functional gains.

Interval versus continuous endurance training

- Vogiatzis et al.: interval cycling (vs continuous moderate-load training) over 10 weeks produced significant increases in Type I and IIa fibre CSA and capillary-to-fibre ratio, alongside improved peak work capacity and lactate threshold.[20]

Interpretation: interval exercise training can elicit microvascular remodelling and muscle fibre hypertrophy, not consistently observed with continuous endurance training (Table 3).

Resistance training during exacerbation and low-load/high-repetition models

- Troosters et al.: in-hospital RT preserved or improved quadriceps muscle strength during acute exacerbations versus a decline in controls.[17]
- Nyberg et al.: low-load/high-repetition (LLHR) RT improved functional outcomes and limb endurance, with biopsy limited to metabolic and microvascular markers.[15,16]

Interpretation: RT is effective across COPD severity spectrum, including during exacerbations. LLHR variants offer scalable options when heavy loading is impractical (Table 2).

Summary

Continuous exercise training primarily drives oxidative and mitochondrial adaptations, whereas resistance and interval exercise modalities provide stronger anabolic stimuli. Eccentric exercise offers high mechanical loading at low ventilatory cost, improving strength without consistent biopsy evidence of hypertrophy. Combined endurance and resistance training approaches capture both metabolic and structural benefits, making them optimal for addressing muscle weakness and functional limitations (Table 4).

Discussion

This review consolidates evidence demonstrating that exercise training interventions are pivotal for addressing skeletal muscle dysfunction in COPD. Among various modalities, resistance-based and IT programs consistently emerge as the most effective strategies for promoting quadriceps muscle hypertrophy and increasing peripheral muscle mass. These structural adaptations directly counteract sarcopenia, a debilitating comorbidity in COPD, and correlate with improved functional capacity and quality of life.

The pattern of adaptation across modalities aligns with known COPD pathophysiology. Chronic systemic inflammation, anabolic resistance, physical inactivity, and disease-related hypoxaemia all contribute to impaired muscle protein synthesis, fibre atrophy, and reduced oxidative efficiency.[2] Exercise training, particularly resistance and interval modalities, can partially offset these processes by stimulating myofibrillar protein accretion, enhancing mitochondrial efficiency, and reducing hypoxia-related metabolic stress.[7,26] Thus, the distinct adaptations elicited by different exercise modes directly address the physiological drivers of skeletal muscle dysfunction in COPD, supporting targeted and mechanism-informed intervention strategies.

Comparative effects of different modalities

Evidence suggests that resistance training remains the most effective modality for restoring muscle size and type II fibre hypertrophy, with multiple studies demonstrating a significant increase in CSA and strength within 6-12 weeks.[19,27,28]

Endurance training, whether continuous or interval-based, primarily enhances oxidative capacity and mitochondrial function without inducing meaningful hypertrophy. Continuous moderate-intensity training (CET) produced minimal CSA changes in multiple trials.[29,30] Although improvements in oxidative enzyme activity and mitochondrial function were noted, structural adaptation limitations were reported in selected trials.[29,31]

Interval exercise training allows COPD patients to exercise at a higher relative intensity with lower ventilatory cost,[32] and elicits both metabolic and modest structural changes, supported by evidence of fibre hypertrophy and microvascular remodelling.[20]

CET remains primarily effective in enhancing oxidative metabolism, with little effect on muscle fibre size. Comparative studies generally report negligible CSA gains following CET, even with interventions lasting 8-12 weeks.[20,33,34]

Peripheral muscle mass and functional implications

Resistance-dominant or mixed training programs consistently show significant increases in thigh or leg lean mass.[19] Endurance-only interventions may preserve muscle mass but rarely reverse wasting.[7,19] Combined aerobic and resistance training programs demonstrate both hypertrophic and oxidative adaptations in a 2021 meta-analysis, confirming that mixed modalities are suitable for multifaceted rehabilitation.[7]

Clinical and mechanistic significance

These modality-specific adaptations indicate the need for precision in pulmonary rehabilitation prescription. RT is critical when the goal is to restore muscle size and strength, particularly in sarcopenic patients. IT offers additional benefits for muscle function and cardiorespiratory fitness. CET remains vital for enhancing oxidative metabolism and overall endurance. These findings support tailored, mechanism-informed exercise prescriptions that maximise functional outcomes in COPD.

Limitations and future directions

This review is limited by heterogeneity of studies in sample size, disease severity, intervention duration, and outcomes. Most mechanistic evidence derives from small biopsy-based trials, restricting generalisability and precluding robust comparisons across modalities. The absence of standardised protocols for intensity and progression complicates dose-response interpretation, and few studies report long-term follow-up.

Future research should prioritise large multicentre trials with standardised exercise protocols; biopsy-informed studies of hypertrophy, oxidative remodelling, and anabolic resistance; multimodal strategies combining exercise with nutritional support or pharmacology; personalised exercise prescriptions informed by phenotypic profiling (e.g., cachexia, hypoxaemia) and biomarkers; and longitudinal studies assessing the sustainability of adaptations and impacts on morbidity and mortality.

Conclusion

Resistance training, alone or combined with interval exercise, is the most effective approach for promoting quadriceps muscle hypertrophy in COPD. Continuous endurance training predominantly enhances oxidative capacity, whereas interval training offers moderate structural gains alongside metabolic adaptations. Combined training modalities provide synergistic benefits and are therefore well-suited to addressing the multifactorial deficits characteristic of COPD-related skeletal muscle dysfunction. Therefore, exercise selection should match patient deficits and goals, resistance training for sarcopenia, endurance training for oxidative limitations, and combined approaches for comprehensive improvement.

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Conflicts of interest

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Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. This study does not involve personal patient data, medical records, or biological samples, and does not require ethical approval. SAGER guidelines do not apply.

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