BRN Reviews REVIEW

Physical Activity and Underlying Muscle Biology in Chronic Obstructive Pulmonary Disease (COPD)

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ABSTRACT

Muscle dysfunction is one of the most studied systemic effects in patients with chronic obstructive pulmonary disease (COPD) and a predictor of mortality. It can be caused by sedentary habit due to muscle underuse. Conversely, muscle dysfunction also contributes to inactivity in COPD. To date, the underlying mechanisms of muscle dysfunction and their impacts on physical activity (PA) levels in COPD were not explored in a literature review. The aims of this review are to summarise and discuss the link between muscle dysfunction and biology with reduced levels of daily PA in patients with COPD. This manuscript covers the many aspects of physical inactivity in COPD, the relationship between muscle dysfunction and muscle biology with physical inactivity in this population, as well as the strategies used to tackle muscle impairment with a possible positive impact on activity levels in COPD and other complementary strategies to increase PA in this population. **(BRN Rev. 2019;5(3):215-29)**

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INTRODUCTION

Although chronic obstructive pulmonary disease (COPD) is primarily considered a lung disease, it associates to systemic consequences and comorbidities, the so-called "systemic effects of COPD" [1]. Indeed, COPD is acknowledged as a systemic disorder. Patients with COPD are often recognised by persistent airflow limitation due to abnormal inflammatory response to inhaled noxious particles or gases (mainly tobacco smoke)[2]; however, common features of COPD also include reduced exercise capacity and lower levels of physical activity (PA) in daily life[3]. Among the systemic effects of COPD, muscle dysfunction, particularly the locomotion muscles, and reduced muscle mass, are among the most studied[4,5]. Muscle wasting is characterised by the loss of muscle mass (and contractile units) and muscle dysfunction is the loss of at least one of the muscle properties: strength and/or endurance. The latter is, in turn, inversely related to the increased fatigability of the peripheral muscles. These muscle properties are commonly evaluated in clinical settings by assessments of muscle strength (e.g., dynamometer) and endurance (e.g., isokinetic). Peripheral muscles of patients with COPD present a shift of the skeletal muscle fibre type I proportion to fibre type II. The oxidative capacity of the muscle is impaired which, in turn, increases the fatigability of the muscle reducing the ability to endure exercise, contributing to muscle dysfunction.

Exercise capacity is impaired in COPD due to multiple factors, including ventilatory limitation, dynamic hyperinflation during exercise, gas exchange abnormalities, impact of comorbid conditions (e.g., cardiovascular comorbidities) and muscle dysfunction. The impaired exercise capacity characteristic of COPD contributes, together with other factors (psychological, behavioural, sociodemographic status), to limit the ability to perform activities of daily life.

According to the World Health Organisation (WHO), inactivity in daily life is one of the leading risk factors for non-communicable diseases, accounting for millions of preventable deaths worldwide[6]. It is established that higher levels of daily PA can postpone and even prevent the appearance of chronic health problems such as cardiovascular diseases or diabetes[7-10]. Moreover, PA is related to morbidity and mortality, especially in chronic lung diseases[11-13]. Additionally, regular PA has been associated to a number of physiological benefits, including reduced metabolic cost of activities, reduced systemic inflammation, reduced body weight and fat mass, increased resistance of myocardial cells to ischaemia and improved insulin sensitivity and angiogenesis[14]. Therefore, the ability to maintain high PA levels plays an important role in the prevention of diseases. This emphasises the importance of assessing and treating physical (in)activity, especially in chronic diseases such as COPD. An active lifestyle should be vigorously encouraged in everyone, including people with chronic health problems.

This review will focus on the underlying muscle biology and its effect on PA levels in COPD. Interventions that can improve PA by tackling muscle dysfunction will be discussed, as well as new strategies that can be complementary to these to achieve improvements in PA levels in patients with COPD.

PHYSICAL (IN)ACTIVITY AND MUSCLE BIOLOGY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Physical activity levels of patients with chronic respiratory conditions are significantly lower than those in the general population[3,15]. Patients with COPD spend more time in the sitting and/or lying position and less time standing/walking[3].

In COPD, patient's activity level is the strongest predictor of all-cause mortality[11], as well as an independent predictor of the risk of hospital admissions due to acute exacerbations and early mortality[12,13]. Besides, sedentary behaviour (spending ≥ 8.5 hours per day in activities of low intensity (≤ 1.5 metabolic equivalent of task [MET])) has also been shown to be an independent predictor of death in this population[16]. Despite the clear association between inactivity and poor outcome, it is still unknown whether and to what extent increasing activity levels of patients with COPD have an impact on survival in this population. It seems, however, reasonable to believe that patients who increase PA or remain physically active may benefit from the positive impact of an active lifestyle and, therefore, have better prognosis than patients with a persistent decline in PA levels over time.

Several factors can contribute to the inactivity characteristic of COPD. These include poor exercise capacity[3,17], to which muscle wasting[18], muscle dysfunction[1,3], hyperinflation[19] and exercise associated-dyspnoea[1,20]; and behavioural and psychological aspects also contribute[2,17] (Fig. 1).

Hyperinflation, a feature of COPD, contributes to inactivity in these patients[21] by imposing additional ventilatory limitation and, consequently reducing inspiratory capacity during exercise. This has a direct impact on ventilation during daily activities, making these patients even more dyspnoeic forcing them to adopt a sedentary habit. This inactivity, together with other factors, contributes to muscle impairment (e.g., reduction in muscle mass and muscle strength – shift in skeletal muscle fibre type I to fibre type II and reduced oxidative capacity of the muscle) due to muscle underuse.

In COPD, muscle dysfunction can be found in both peripheral (mainly muscles involved in locomotion) and respiratory muscles[22]. There are important differences in the underlying pathophysiological mechanisms leading to muscle dysfunction in these two compartments. This review will focus on peripheral muscle dysfunction, mainly of the quadriceps femoris, as the most investigated muscle that has significant impact on exercise capacity and PA levels. Muscle dysfunction and wasting[1,3], particularly in the limb muscles, is one of the most important systemic effects in COPD[4,5,23] and muscle underuse (due to sedentary habits) is a contributing factor to this. Muscle wasting and dysfunction affects muscle strength[4,24,27], exercise capacity[28-31] and is a predictor of poor health-related quality of life (HRQoL)[32], increased health care utilisation[33] and associates with poor survival[34,35] in COPD, independently of the degree of airway obstruction[31].

Several pathophysiological changes have been identified in the skeletal muscle of patients with COPD such as atrophy of muscle fibres[28], fibre type redistribution[36], bioenergetics alteration (reduced oxidative

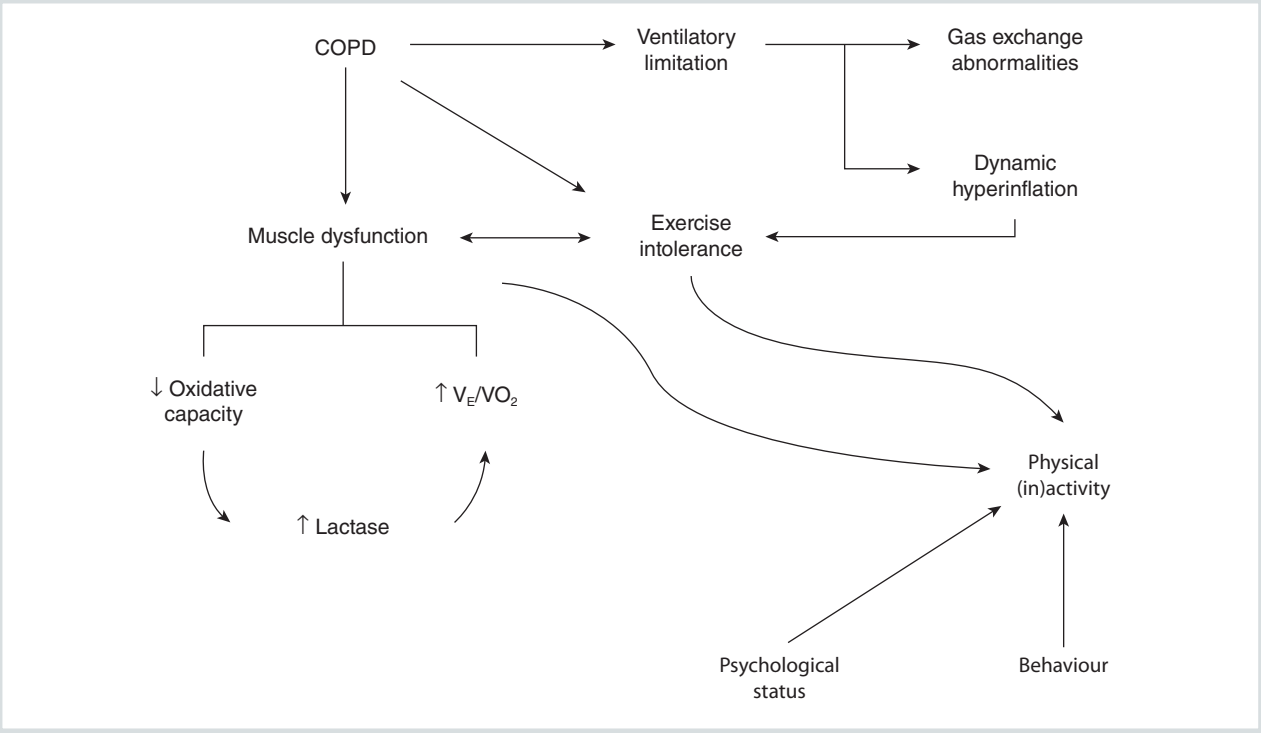

Figure 1. Factors affecting physical activity in patients with chronic obstructive pulmonary disease.
COPD: chronic obstructive pulmonary disease; V_E/VO_2: ventilatory equivalent for oxygen.

capacity)[36], capillarisation defects[37], and altered mitochondrial function[38,39].

A reduction in the fibre to capillaries ratio, muscle cell membrane abnormalities, sarcomere damage and mitochondrial derangements that may impair oxygen delivery to the myofibres are contributing biological mechanisms to muscle dysfunction[23,40]. Among other causes, hypoxia and oxidative stress both induce a loss in muscle mass as a result of the interaction of several molecular mediators (e.g., eukaryotic translation initiation factors [eIFs], AMP-activated protein kinase [AMPK] and DNA damage-inducible transcript 4 protein [DDIT4/REDD1])[41]. In turn, hypercapnia, affecting a subgroup of patients during stability of the disease or during exacerbations, can also affect the muscle through enhancement of proteolytic system activity and/or a reduction in protein anabolism[42]. Furthermore, there are other factors that may contribute to muscle dysfunction such as cigarette smoke and use of systemic glucocorticoids, frequently used as treatment during acute exacerbations of COPD. Cigarette smoke may decrease type I fibre sizes and proportions and can lead to reduced mitochondrial activity, while causing an increase in reactive oxygen species (ROS) levels[43]. Systemic corticosteroids may worsen the type II fibre atrophy characteristic of the peripheral muscle of patients with COPD[40].

The molecular mechanisms leading to skeletal muscle dysfunction/wasting are, to date,

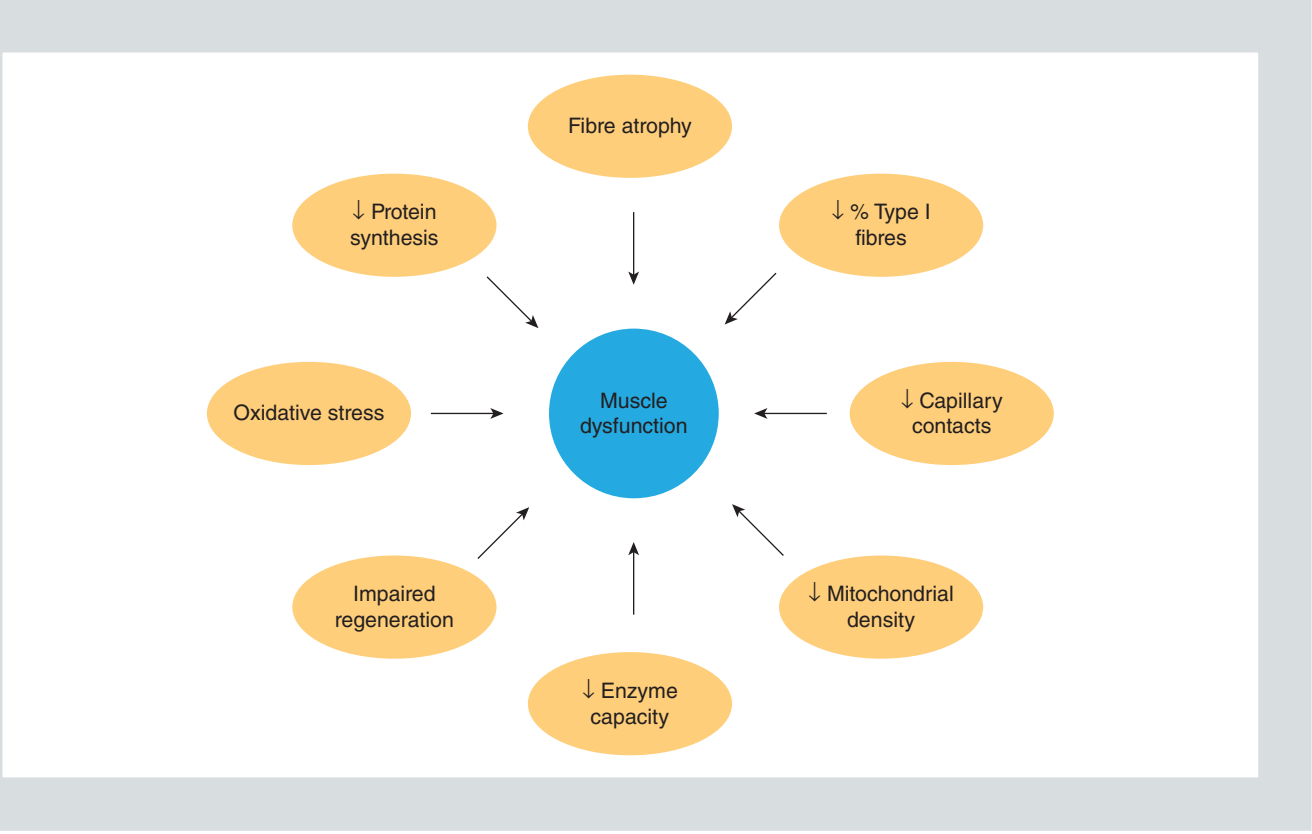

Figure 2. Biological causes of muscle dysfunction in patients with chronic obstructive pulmonary disease.

not fully understood and are likely to be multi-factorial (Fig. 2). Beside muscle underuse[44], several factors have been described as putative mechanisms leading to muscle dysfunction/atrophy, namely systemic inflammation[45], oxidative stress[46], cell hypoxia[47], hypercapnia, low levels of anabolic hormones (testosterone) and growth factors (growth hormone [GH], insulin-like growth factor I [IGF-I]), use of glucocorticoids, vitamin D deficiency, impaired energy balance[5], accelerated ageing and cellular senescence[48-52], and nutritional depletion[53].

Changes in the skeletal muscle of patients with COPD make the limb muscle more susceptible to fatigue and increase the ventilatory requirements, likely due to increase lactate metabolism, consequently decreasing exercise capacity of the subjects. This reduced capacity can lead to inactivity and sedentary behaviour in daily life[3,16,44] conforming a "vicious cycle" of inactivity – muscle dysfunction – increased breathlessness – more inactivity[54] (Fig. 3 and Fig. 4). Therefore, muscle dysfunction can contribute to inactivity. Similarly, as mentioned earlier, inactivity can also contribute to muscle function impairment.

INTERVENTIONS TO IMPROVE PHYSICAL ACTIVITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The negative impact of physical inactivity in COPD highlights the need for interventions

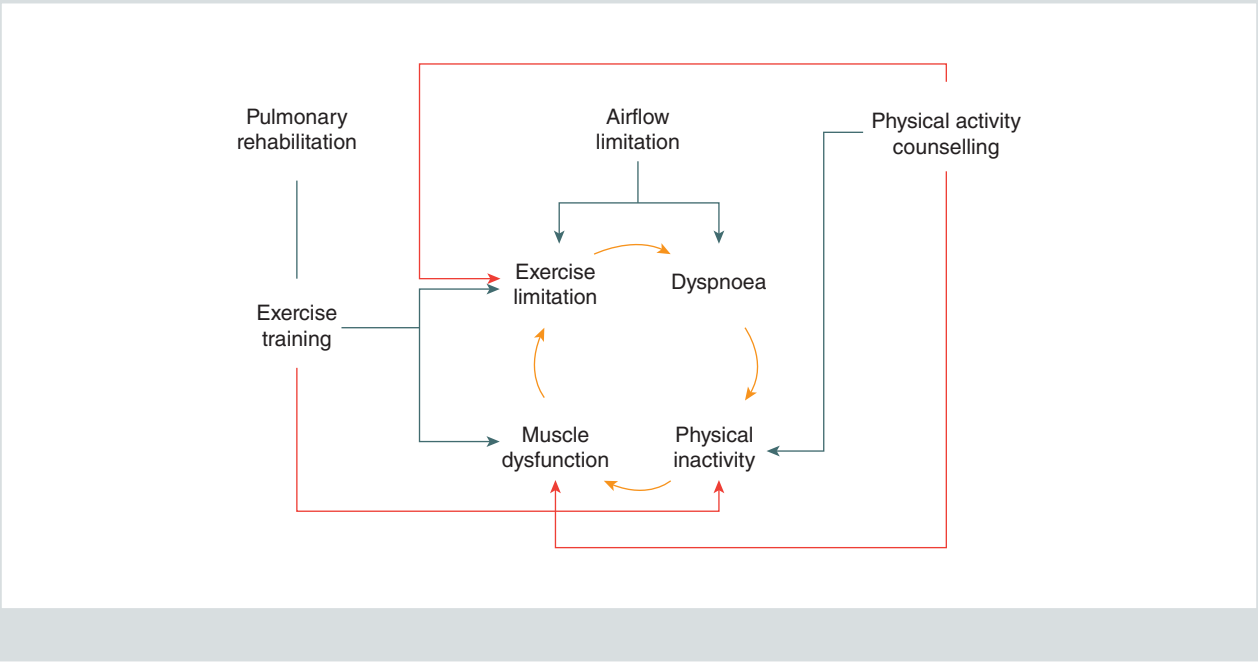

Figure 3. Vicious cycle of chronic obstructive pulmonary disease and interventions to try to break this cycle. Full arrows represent strong evidence and dotted arrows represent a weak evidence of the benefits with the interventions on the represented outcomes.

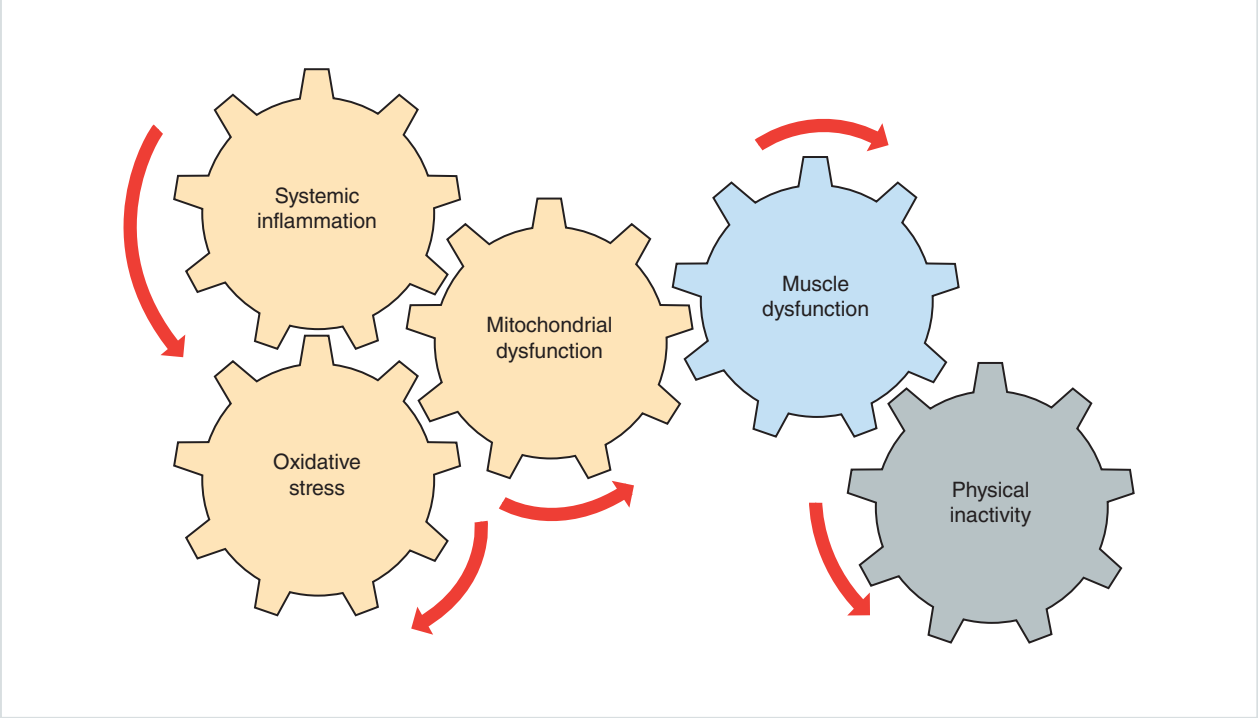

Figure 4. Gear scheme of muscle biology influencing physical activity in chronic obstructive pulmonary disease.

aiming to improve PA levels in this population. Reductions in the exercise capacity contribute to the ability to perform activities of daily living. Symptoms of dyspnoea and muscle fatigue are the main manifestations of patients during exercise. These symptoms preclude patients to engage in an active lifestyle[55]. Numerous features have been recognised as limiting the ability to exercise in patients with COPD: ventilatory limitation, dynamic hyperinflation, gas exchange abnormalities, comorbidities such as cardiovascular disease, and muscle dysfunction/wasting to name a few[55]. Therefore, interventions aiming at modifying these factors may lead to an improvement in exercise capacity with the potential to improve PA levels[56].

On the other hand, daily PA is also related to behavioural and psychological factors[57]. This is highlighted by the fact that interventions that are successful in improving exercise capacity, such as exercise training, not always are effective in converting these improvements into higher levels of PA in daily life[57,58]. Therefore, it seems that exercise training combined with interventions promoting PA should be recommended for COPD patients. This can be necessary even in early phases of the disease.

It is important to note that exercise capacity and PA levels constitute different concepts. Physical activity is defined as "any bodily movement produced by skeletal muscles that results in energy expenditure"[59]. Therefore, simple activities such as walking on level ground or upstairs, sweeping the floor, etc. are examples of PA. Conversely, exercise capacity is described as "the maximum amount of physical exertion that a patient can sustain"[60], for instance on a walking test or cardiopulmonary exercise test. Although these two concepts are interrelated, PA levels are conditioned by multiple factors and exercise capacity is one of them. However, other factors are equally relevant to warrant adequate PA levels, namely psychological and behavioural aspects. The levels can be modified (in)directly by interventions that improve muscle function and exercise capacity, and/or with strategies aiming to change behaviour towards PA.

IMPROVING EXERCISE CAPACITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (TACKLING THE MUSCLE WITH PULMONARY REHABILITATION)

The primary treatment to muscle dysfunction in COPD is exercise training in the context of a pulmonary rehabilitation (PR) programme[56,61]. Pulmonary rehabilitation offers numerous benefits, including: improvement in muscle strength and exercise capacity, reduction of exercise-induced dyspnoea and muscle fatigue, improvement of anxiety and depression levels and HRQoL[56,62].

The cornerstone of PR is exercise training, which usually involves both aerobic and resistance training. Exercise training is a mechanical stimulus that consists of repeated bouts of muscle stress promoting functional adaptation and remodelling not only to the skeletal muscle, but also to various systems[63]. Skeletal muscle overload is an important training principle indicating that the level of the training load must be sufficient to stress the muscle in order to obtain physiological training

adaptations (e.g., change in fibre type, increase in the number of mitochondria, enhance oxidative capacity)[64].

In healthy elderly subjects, aerobic and resistance training increase fibre size, capillarisation and alter the proportion of fibre type in favour of a more oxidative profile[65]. Therefore, these evidences provide a rationale for exercise training in patients with COPD to counteract structural muscle abnormalities developed with the disease. The improvements in muscle strength and endurance, as well as the increase in muscle size/volume, changes in fibre type proportion and in capillarity of muscle fibres may differ according to the modality of the selected training[63].

Table 1. Summary of different interventions effect on the muscle

Intervention	Muscle effects
Resistance training	– Increase proportion of fibre type IIa – Increase satellite cells (maintenance/ regeneration of muscle mass)
Aerobic training	– Increase cross-sectional area and capillarisation of fibres type I – Increase the oxidative regulation (mitochondrial respiration)
Combined training	– Increase in mean fibre size and capillarisation
NMES	– Increase cross-sectional area
HIIT	– Decrease proportion of fibre type IIx – Increase cross-sectional area and capillarisation – Increase the oxidative regulation (mitochondrial respiration)

HIIT: high-intensity interval training; NMES: neuromuscular electrical stimulation.

Resistance training (strength training)

The recommendation for resistance training consists of two to three sessions per week, including two to four sets of 8 to 12 repetitions of each exercise, at loads ranging and progressing from 30 to 90% of one repetition maximum (1RM)[5,66]. Following these guidelines, substantial effects can be achieved maximising the physiological benefits of the training[67]. The exercise programme may enhance the fatigue resistance of the muscle by promoting a muscle-fibre type shift, increasing muscle fibres hypertrophy, improving mitochondrial function and glucose transportation and reducing sarcopenia[67].

A conventional resistance training[56] reduces patient's symptoms[68], increases exercise capacity[69], muscle strength[68,70-72] and fat-free mass[72]. Furthermore, the satellite cells that are responsible for maintenance of muscle mass and regeneration of injured muscle fibres may increase in response to resistance training. However, it is still unclear whether this training may have a wider anti-inflammatory effect on these patients[71]. When considering muscle structural changes, the resistance training has shown a significant increase in the proportion of type IIa fibres[69,71], and a decrease in type I fibre proportion[69] (Table 1). In addition, when this training modality was applied in isolation, it did not show significant increase in capillarisation, nor in the cross-sectional area of the quadriceps[69]. These findings demonstrate that resistance training alone does not promote more oxidative changes in the muscle of COPD patients, which seems to be a key factor when considering increments in daily life activities. Indeed, this intervention alone did not show a positive impact on PA levels in COPD[73].

Aerobic and combined (aerobic and resistance) training

It is common knowledge that aerobic training improves exercise capacity, symptoms and quality of life in patients with COPD[56]. The American College of Sports Medicine (ACSM) recommends that aerobic training is prescribed with a frequency of three to five times per week, with high level of intensity of continuous exercise (60% maximal work rate) or with a modified Borg score of 4 to 6 (moderate-to-severe) for 20 to 60 min per session[56,66].

Aerobic training can induce neuromuscular adaptations that generate greater sarcolemma excitability, better muscle efficiency, and higher recruitment of slow-twitch fibres[74]. These changes decrease the oxygen consumption kinetics during exercise, making the cardiovascular, respiratory and muscular systems response to exercise faster. This leads to a smaller oxygen deficit during activities, contributing to increase the endurance time to high intensity exercise[75], and, potentially, having a positive impact on daily activity levels.

Moreover, training with moderate intensity was shown to increase the oxidative regulation of mitochondrial respiration that influences muscle endurance[5,76,77] and activity of oxidative enzymes (e.g., citrate synthase [CS], 3-hydroxyacyl CoA dehydrogenase [HADH]) while decreasing the activity of enzymes linked to the anaerobic metabolism (e.g. lactate dehydrogenase [LDH], phosphofructokinase [PFK] and creatine-kinase [CK]). This explains, in part, the positive effect of exercise training on the oxidative capacity of the muscle[75,77].

By improving the oxidative capacity of the muscle (and reducing the lactate production during exercise), exercise training improves the ventilatory equivalent for carbon dioxide (V_E/VCO_2)[78,79]. Patients require significant less ventilation to perform the same level of exercise after exercise training. In turn, this reduction in the minute ventilation prevent, or at least improves, the potential development of dynamic hyperinflation[80]. Moreover, the reduction of lactate production results in improvement in muscle fatigue[77,78]. All these factors lead to improved exercise capacity with potential benefits in PA levels.

As for the changes in muscle structures of COPD patients, the aerobic training alone demonstrated no changes in fibre type proportion, but it did show an increase in both the capillarisation and the cross-sectional area of fibres type I (the more oxidative fibres)[81].

Opposite to healthy elderly subjects, patients with COPD show a reduced muscle redox capacity after endurance training[82] with an abnormal increase in plasma tumour necrosis-α (TNF-α) levels in response to exercise bouts[83]. The abnormal redox response to training appears to be worse in patients with reduced muscle mass[84]. In patients with severe COPD, three weeks of endurance training performed five days/week increased quadriceps nitrosative stress, impacting the antioxidants systems[85]. It is known that ROS relates to a reduction in muscle strength and endurance[84]. This suggests that chronic endurance exercise of relatively high intensity, particularly in patients with reduced muscle mass, may generate further disruption of the nitroso-redox balance leading to maladaptive muscle alterations. Moreover, it may also help to guide

the debate on the rationale for interventions increasing the redox potential of muscle cells in the subset of COPD patients with low body mass index (BMI).

Studies combining both trainings of resistance and aerobic exercises maximize the benefits of both modalities with better impact on relevant clinical outcomes (i.e. survival, exercise capacity, muscle strength, quality of life)[86,87] through benefits from muscle structural and functional changes in response to both training modalities. The effects on muscle structure are an increase in fibre size and capillarisation, without significant results on metabolic characteristics and oxidative stress[88]. However, the studies combining resistance with aerobic exercise training that explored changes at structural level used different strategies of training. More trials are needed to investigate these physiological and structural changes in the muscle after PR programmes[88].

Effects of exercise training on physical activity

Unfortunately, until now, findings from different studies using either aerobic or combined training have conflicting effects on PA levels of patients with COPD. Nearly half of the studies fail to improve PA levels[58]. Interestingly, longer programmes (i.e. with more than three months of duration) have better results when compared to shorter programmes (< 3 months)[58]. A study comparing the effects of three against six months training showed that both programmes were able to increase exercise capacity. However, only after 6 months of training a significant impact on PA levels was achieved[89]. These results suggest that more time is needed to promote a change in behaviour in this population, and it appears that three months of PR is enough to train the muscles but a longer time (e.g., six months) is needed to "train the brain". Similarly, more intense programmes seem to have better results on PA[58,90] than lower intensity programmes. Strategies to deliver high intensities during training are possibly more effective in achieving an effect on PA levels.

High Intensity Interval Training

The high intensity interval training (HIIT) is an alternative training delivery method for severe and very severe patients, as well as for patients with high levels of symptoms that are not able to perform exercise at high intensities for long periods of time (i.e. continuous exercise training)[91]. The HIIT may facilitate the administration of high intensity exercise through the use of intermittent recovery periods. This associates with a reduction in muscle metabolic products that stimulate increased ventilatory demand reducing the end-expiratory lung volume and symptoms[91]. Thus, patients can exercise at higher intensities with less leg fatigue and dyspnoea[91].

Although HIIT has some advantages over conventional training (constant-work rate), the overall findings from HIIT studies in COPD indicate that this method is at least as equally effective as continuous exercise training in producing benefits on exercise capacity and symptoms[91]. At micro-structural levels, the HIIT revealed no changes after exercise training for type I and type IIa fibre distribution, whereas the type IIb fibre distribution was

reduced regardless of the severity of the disease[92-94]. In addition, the muscle capillarisation and the cross-sectional area of fibres type I and IIa increased[92-94], factor that may have contributed the patients to achieve higher levels of activity on their daily life[90]. It is important to highlight that this type of training has been shown to improve daily activity levels of patients with COPD[90].

Other exercise strategies and training modalities

Despite the benefits demonstrated with the "conventional" type of trainings, not all patients respond to PR. Therefore, individual characteristics of the subjects (e.g., low exercise capacity, high reported dyspnoea sensation on daily life, muscle dysfunction) should be considered when prescribing the best exercise training programme for each individual. Hence, new strategies may be implemented in order to enhance the physical training of patients who are not able to improve their exercise capacity with the mentioned exercise training programmes[95].

Some of the newer modalities of exercise training use the development of muscle contractile fatigue during PR sessions to promote improvements in exercise capacity and quality of life[64]. Downhill walking[96] and eccentric cycling[97,98] are among these strategies and result in an increase in muscle strength[96,97]. At a muscle structural level, eccentric cycling training did not show changes in proportion of fibre distribution, but did increase the cross-sectional area and generated mitochondrial adaptations in the muscle of the patients[97]. As far as we are aware, no studies assessed the PA levels of patients with COPD submitted to either downhill or eccentric cycling training.

Another alternative modality of aerobic/combined training is the nonlinear exercise training. This type of intervention combines the endurance and strength limb muscle training alternating the number of sets, repetitions and the intensity of the exercise to promote better muscle adaptations[99]. The use of nonlinear periodisation in resistance training for improving muscle function in patients with COPD appears as a promising alternative to the ordinary continuous exercise training[100]. Moreover, modalities applying whole-body vibration seem to have a positive effect on muscle function in COPD[101].

To implement these interventions a detailed baseline evaluation of the patients is required in order to identify the specific training protocol which may maximise the benefits of PR in a particular subject. In addition, the possible increments in terms of daily PA should be the focus of future research when using these modalities.

Neuromuscular Electrical Stimulation (NMES)

The neuromuscular electrical stimulation (NMES) can be used to increase peripheral muscle strength and/or endurance in patients who are unable to achieve adequate intensity during conventional aerobic training (e.g., very severe COPD and/or during severe exacerbations of the disease)[102]. The NMES generates similar effects as resistance training on exercise capacity, muscle strength

and symptoms[103,104]. Although this intervention seems to be able to induce increases in the cross-sectional area[105], no changes were observed in the relative fibre distribution or capillarity[104]. Unfortunately, no changes in PA levels were achieved with NMES in COPD patients[58].

Although this intervention seems to induce increments in the cross-sectional area[105], no changes were observed in the relative fibre distribution or capillarisation[104]. The effect of NMES at micro-structural level is still unclear, partly due to the heterogeneity of the protocols used[103]. Therefore, more studies with higher methodological quality are necessary to investigate the possible changes in muscle structure as well as to highlight which is the profile of the patients that would benefit the most from this type of intervention. The extent of the impact this intervention can have on activity levels of patients with COPD also deserves to be further investigated.

Improving physical activity levels in chronic obstructive pulmonary disease with coaching programmes

It is still challenging to achieve high levels of PA in COPD with the standard exercise training modalities. While PR is the most successful intervention at improving dyspnoea, muscle dysfunction, exercise capacity, HRQoL and health care utilisation and costs[56] in subjects with chronic respiratory diseases, the conversion of these improvements into increments of PA levels is less evident as previously mentioned[58]. Some studies show moderate increments in PA levels while others indicate no effects[58]. Although improving muscle function with exercise training allows the patients to increase their exercise capacity, patients do not necessarily opt to increase their activity levels. Therefore, other strategies are needed to change patients' behaviour towards a more active lifestyle.

Physical activity coaching is becoming a more common strategy to stimulate patients towards these goals[58]. By giving individualised PA targets, delivering tailored messages and using PA monitors that provide objective assessment and feedback on activity levels, coaching strategies are very successful interventions to increase PA in patients with COPD[58]. Interestingly, combining PR with PA coaching programmes had greater impact on PA levels of COPD patients than PR only[106]. Perhaps merging the benefits of the PR with the encouragement provided by a PA coaching programme is the way forward into a more active lifestyle for patients with COPD. This modality has the potential to turn the benefits acquired with the PR programme (in terms of increments in exercise capacity, muscle strength and muscle mass, and improvements in dyspnoea sensation) into more time spent doing PA. Unfortunately, the effects of such programmes on muscle mass and muscle strength, especially at micro-structural levels, are not well described in the literature. Therefore, more studies are welcome to investigate how PA coaching programmes can impact on muscle biology of patients with COPD.

CONCLUSIONS

Muscle dysfunction is one of the most relevant systemic effects in patients with COPD. It is a predictor of mortality in this population and

can be caused by sedentary habit due to muscle underuse (among other causes). Conversely, muscle wasting and muscle dysfunction also contribute to inactivity in COPD. The molecular mechanisms leading to skeletal muscle wasting/dysfunction are likely to be multi-factorial. Pulmonary rehabilitation through the different modalities of exercise training contributes to improve muscle function with a positive impact on exercise capacity in these patients. However, the transformation of these effects into increments in PA is still a challenge with longer and more intense programmes showing the best results. As psychological and behavioural factors seem to influence the predisposition of patients towards a more active lifestyle, strategies aiming at helping patients to change their behaviour towards activity level can play a role in treating the inactivity characteristic of patients with COPD. Therefore, PA coaching programmes, alone or in association to PR, appear as a promising tool to counteract inactivity in COPD.

DISCLOSURES

The authors have nothing to disclose.

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