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Quadriceps concentric-eccentric force and muscle architecture in COPD patients vs healthy men

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ABSTRACT

The aim of this study was to compare quadriceps concentric and eccentric strength and muscle architecture in chronic obstructive pulmonary disease (COPD) patients vs healthy men. Thirty-five COPD patients (age = 65 ± 4 yrs, forced expiratory volume (FEV_1) = 63 ± 10%; FEV_1/forced vital capacity (FVC) = 57 ± 13% of predicted) and 25 age-matched healthy men (age = 65 ± 4 yrs, FEV_1 = 114 ± 17%; FEV_1/FVC = 101 ± 6% of predicted) (CON) participated in the present cross-sectional study. Concentric and eccentric isokinetic peak-torque was measured at low and high angular-velocity. *Vastus lateralis* pennation angle, fascicle length and muscle thickness were recorded using ultrasound device. Similar eccentric peak-torque was found in COPD and CON at low (2.57 ± 0.55 and 2.80 ± 0.60 $N \cdot m \cdot kg^{-1}$, $p = 0.128$ respectively) and high (2.44 ± 0.51 and 2.58 ± 0.46 $N \cdot m \cdot kg^{-1}$, $p = 0.259$) angular-velocity. Lower concentric peak-torque was found in COPD than in CON ($p < 0.05$). Smaller pennation angle (13.8 ± 3.4 vs 16.2 ± 3.9°) and muscle thickness (17.1 ± 2.8 vs 20.3 ± 3.0 mm) were found in COPD patients vs healthy men, with no difference in fascicle length. In COPD patients only, FEV_1 and FEV_1/FVC were negatively correlated with the eccentric-to-concentric peak-torque ratio ($r = -0.465$ and $r = -0.414$, respectively); irrespective of the testing-modality, FEV_1 and FEV_1/FVC were moderately correlated with peak-torque ($p < 0.05$). The preserved eccentric strength in COPD patients could be accounted for both mechanical and neural adaptations caused by the disease severity.

1. Introduction

Muscle wasting is a widely spread comorbidity in chronic obstructive pulmonary disease (COPD) patients, which contributes to the worsening of their quality life (Couillard & Prefaut, 2005). Such a phenomenon particularly affects the lower-limbs muscles, leading to a progressive muscle atrophy (Kim, Mofarrahi, & Hussain, 2008; Sanders, Kneppers, van de Bool, Langen, & Schols, 2016) and it is related to an increased amount of connective tissue, usually linked with a sort of fibrosis (Gosker et al., 2003). In addition, a shift in lower-limb muscle fibres towards type-II phenotype (Couillard & Prefaut, 2005; Kim et al., 2008; Sanders et al., 2016) has been reported, whose prevalence is related to the disease severity (Gosker, Zeegers, Wouters, & Schols, 2007). These morphological changes were indirectly confirmed by a greater muscle fatigability in severe vs moderate COPD or vs healthy people (Boccia et al., 2016), which correlated with the disease severity (Boccia et al., 2015). Such a fatigability, together with the respiratory diseases, leads to a reduced exercise tolerance in COPD patients, which resulted in a very sedentary lifestyle (Couillard & Prefaut, 2005; Rinaldo et al., 2017).

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Despite the acknowledged muscle wasting, less is known about lower-limb muscle architecture in COPD patients. Muscle architecture encompasses muscle thickness, pennation angle, fascicle length, as well as fascicle curvature or inner muscle compartments that can, separately or together, influence the muscle functions. Fascicles in pinnate muscles are attached to the tendon aponeurosis or bony structures with a certain angle and the increases in pennation angle lead to larger cross-sectional area, which, in turn, leads to greater muscle strength (Blazevich, Gill, & Zhou, 2006). Additionally, the increases in pennation angle, together with the increases in in-parallel myofilaments, act on the physiological cross-sectional area, incrementing its size (Lieber & Blevins, 1989). Furthermore, muscles with longer fascicles are able to work at high shortening-speeds and increase their elongation and flexibility (Blazevich et al., 2006). It was also shown that quadriceps cross-sectional area was correlated with the quadriceps maximal voluntary contraction, in both young and elderly people (Strasser, Draskovits, Praschak, Quittan, & Graf, 2013). However, muscle architecture can be affected by age (Strasser et al., 2013) gender (Kubo et al., 2003) and inactivity (de Boer et al., 2008), which all lead to decreases in muscle thickness and pennation angle, with negative consequences for strength and contraction velocity. Since the reduced tolerance to physical activity shown in COPD patients (Couillard & Prefaut, 2005; Rinaldo et al., 2017), a drop in pennation angle and muscle thickness due to disuse (de Boer et al., 2008) may be expected to be related to the observed atrophy (Couillard & Prefaut, 2005), even if no data on lower-limb muscle architecture in COPD patients have been previously reported. On the contrary, resistance training has been shown to be a powerful method to improve the muscle functions through favourable muscle architecture exercise-induced adaptations (Coratella, Milanese, & Schena, 2015b). Therefore, comparing muscle architecture between COPD patients and healthy people could add further to the mechanisms underlying the lower-limbs muscle wasting in COPD patients.

Coupled with muscle wasting, COPD patients showed reductions in lower-limb muscle strength, as shown in quadriceps (Seymour et al., 2012), plantar flexors (Gagnon et al., 2013) and dorsi-flexors (Maddocks et al., 2014) muscles. However, the studies that previously reported reductions in muscle strength in COPD patients compared to healthy people, measured it in isometric or concentric modality (Gagnon et al., 2013; Kharbanda, Ramakrishna, & Krishnan, 2015; Maddocks et al., 2014; Seymour et al., 2012). On the contrary, the only study that measured muscle strength in eccentric modality, reported greater eccentric peak-torque normalized per muscle-volume in COPD patients than in healthy people (Mathur et al., 2007). The eccentric contraction benefits from the increased passive tension by the non-contractile proteins and connective tissue (Herzog, Powers, Johnston, & Duvall, 2015), whose greater absolute amount was reported in COPD patients (Gosker et al., 2003). More in detail, the titin-actin interaction plays a key-role in incrementing the force exerted by the sarcomere during an active elongation (Rode, Siebert, & Blickhan, 2009). In addition, COPD patients develop a greater proportion of fast-twitch fibres (Gosker et al., 2007), that it was thought to be primarily involved during the eccentric contraction (Nardone, Romanò, & Schieppati, 1989). However, no further study investigated muscle strength in COPD patients using different contraction modalities, i.e.: concentric vs eccentric. Similarly, given the proven shift in muscle fibres proportion towards type-2 fibres in COPD patients (Couillard & Prefaut, 2005; Gosker et al., 2007), it could be interesting to evaluate if, irrespective of contraction modality, muscle strength is preserved if peak torque is measured at low or high angular velocity.

Because of the inclusion of eccentric exercise in several rehabilitation programs for COPD patients (for a detailed review, see Ellis, Shields, Lim, & Dodd, 2015), investigating if COPD patients can preserve the eccentric strength may increase the interest towards such an exercise modality. Although such possible implications, to the best of the authors' knowledge, no further study investigated if eccentric strength can be preserved in lower-limb muscles in COPD patients. In addition, even if muscle wasting is a very well-known comorbidity related to COPD, no study has described the muscle architecture in lower-limb muscles. Therefore, the aims of the present study were to: i) compare the COPD vs healthy men quadriceps peak-torque both in concentric and in eccentric modalities; ii) evaluate if the severity disease could be correlated with the lower-limb muscle strength and iii) compare the *vastus lateralis* muscle architecture in COPD vs healthy men and investigate the possible correlations with the quadriceps muscle strength.

2. Methods

2.1. Experimental approach to the problem

The present study was designed as a cross-sectional investigation. The participants were involved in two separate testing sessions, separated by at least three days. In the first one, the pulmonary functions were measured and the participants familiarized with the isokinetic dynamometer. In the second one, first the architectural and then the strength measurements were assessed. The participants were instructed to refrain from any form of vigorous physical activity for the entire duration of the study.

An *a-priori* sample size calculation, based on a large effect size (ES) = 0.65 in differences in muscle strength recorded in literature (Seymour et al., 2009; Seymour et al., 2012), one tail, $\alpha = 0.05$ and required power (1-β) = 0.8, resulted in a total of 60 participants. An *a-posteriori* power calculation, based on the confirmed large ES in difference in concentric peak torque, $\alpha = 0.05$ and the size of the two groups (COPD = 35 and healthy = 25), resulted in power (1-β) = 0.79.

The diagnostic criteria for the COPD were based on the GOLD document, for which the threshold is a post-bronchodilator ratio of forced expiratory volume in 1 s (FEV_1) to forced vital capacity (FVC) < 0.7 (Vestbo et al., 2013). In addition, the diagnosis was based on the clinical data which confirmed that the FEV_1/FVC ratio was lower than the 5th percentile of the reference population (Celli, MacNee, & ATS/ERS Task Force, 2004). The participants were categorized using the GOLD stages according to the severity of the airflow limitation (Vestbo et al., 2013).

2.2. Participants

Thirty-five patients moderate (N = 12) and severe (N = 23) COPD men (COPD) (age = 65 ± 4 yrs, FEV_1 = 64 ± 13% of