

Exercise training in COPD: muscle O₂ transport plasticity

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¹Dept of Internal Medicine, University of Utah, Salt Lake City, UT, USA. ²Geriatric Research, Education, and Clinical Center, VA Medical Center, Salt Lake City, UT, USA. ³Dept of Medicine, University of California, San Diego, La Jolla, CA, USA. ⁴Dept of Nutrition and Integrative Physiology, University of Utah, Salt Lake City, UT, USA.

Corresponding author: Ryan M. Broxterman (ryan.broxterman@utah.edu)

 This single-page version can be shared freely online. Copyright 6The authors 2011 For permissions contact permissions contact permissions contact This article has supplementary material available from igersiournal.com Received: 10 Nov 2020 Accepted: 5 Jan 2021 Accepted: 5 Jan 2021 This in increased Y_{mop}peak in patients with COPD (However, it is unknown how these determinants of V_{mop}peak in patients with COPD. Adaptations to 8 weeks of single-leg knee-extensor exercise training were used to determine muscle 0₂ transport due training were used to determine muscle 0₂ transport and utilisation at maximal exercise pre- and post-training. Training increased Y_{mop}peak in bOCOPD (by ~26% from 271429 to 3424358 mcl-min⁻¹) and controls (by ~32% from 418437 to 5538441 mL·min⁻¹), restoring Y_{mop}peak in COPD to only ~80% of pre-training control V_{mop}peak. Muscle diffusive 0₂ transport increased similarly in both COPD (by ~38% from 66:60.9 to 9.11:0.9 mL·min⁻¹ mmHg⁻¹), with the patients with COPD (438454 versus 491451 mL·min⁻¹) at ~70% of pre- training control V_{mop}peak. Mostle diffusive 0₂ transport increased significantly only in controls (by ~26% from 10.4±0.7 to 14.1±0.8 mL·min⁻¹, meHg⁻¹), with the patients with COPD (438454 versus 491451 mL·min⁻¹) at ~70% of pre- training control values. Winter plasticity in muscle convective 0₂ transport. 	Check for updates	 Shareable abstract (@ERSpublications) Patients with severe COPD exhibited attenuated plasticity in muscle convective oxygen transport, and therefore muscle V'_{O₂peak}, in response to exercise training that extended beyond simply disuse and would be expected to contribute to muscle dysfunction. https://bit.ly/38uAGfS Cite this article as: Broxterman RM, Wagner PD, Richardson RS. Exercise training in COPD: muscle O₂ transport plasticity. <i>Eur Respir J</i> 2021; 58: 2004146 [DOI: 10.1183/13993003.04146-2020].
Copyright @The authors 2021, Fr permissions contact permissions contact permissions contact permissions contact defersion as supplementary material available from defersion as contact excepted: 5 Jan 2021 Abstract Both convective oxygen (O_2) transport to, and diffusive transport within, skeletal muscle are markedly uptake (V _{mo2} peak) respond to exercise training in patients with COPD. Therefore, the purpose of this situ COPD. Convective oxygen (O_2) transport to skeletal muscle O_2 transport determinants of V _{mo2} peak in patients with COPD. Adaptations to 8 weeks of single-leg knee-extensor exercise training were measured in eight patients with ever COPD (mean±scx forced expiratory volume in 1 s (FEV_1) 0.9±0.1 L) and eight patients with ever eused to determine muscle O ₂ transport and utilisation at maximal exercise pre- and post-training. Training increased V _{mo2} peak in both COPD (by ~26% form 271±29 to 342±35 mL·min ⁻¹) and controls (by ~32% from 418±37 to 553±41 mL·min ⁻¹), restoring V _{mo2} peak in COPD to only ~80% of pre-training control V _{mo2} peak, Muscle diffusive O ₂ transport increased similarly in both COPD (by ~38% from 6.6±0.9 to 9.1±0.9 mL·min ⁻¹ ·mmHg ⁻¹) and controls (by ~36% from 688±57 to 86±69 mL·min ⁻¹). Reaving patients with COPD (438±45 versus 491±51 mL·min ⁻¹) at ~70% of pre- training control values. While muscle diffusive O ₂ transport in COPD was largely restored by exercise training, V _{mo2} peak remained constrained by limited plasticity in muscle convective O ₂ transport.		This single-page version can be shared freely online.
	Copyright ©The authors 2021. For reproduction rights and permissions contact permissions@ersnet.org This article has supplementary material available from erj.ersjournals.com Received: 10 Nov 2020 Accepted: 5 Jan 2021	Abstract Both convective oxygen (O ₂) transport to, and diffusive transport within, skeletal muscle are markedly diminished in patients with COPD. However, it is unknown how these determinants of peak muscle O ₂ uptake (V_{mO_2} peak) respond to exercise training in patients with COPD. Therefore, the purpose of this study was to assess the plasticity of skeletal muscle O ₂ transport determinants of V_{mO_2} peak in patients with COPD. Adaptations to 8 weeks of single-leg knee-extensor exercise training were measured in eight patients with severe COPD (mean±sɛm forced expiratory volume in 1 s (FEV ₁) 0.9±0.1 L) and eight healthy, well- matched controls. Femoral arterial and venous blood samples, and thermodilution-assessed leg blood flow were used to determine muscle O ₂ transport and utilisation at maximal exercise pre- and post-training. Training increased V'_{mO_2} peak in both COPD (by ~26% from 271±29 to 342±35 mL·min ⁻¹) and controls (by ~32% from 418±37 to 553±41 mL·min ⁻¹), restoring V'_{mO_2} peak in COPD to only ~80% of pre-training control V_{mO_2} peak. Muscle diffusive O ₂ transport increased similarly in both COPD (by ~38% from 6.6±0.9 to 9.1±0.9 mL·min ⁻¹ , mHg ⁻¹) and controls (by ~36% from 10.4±0.7 to 14.1±0.8 mL·min ⁻¹ ·mHg ⁻¹), with the patients reaching ~90% of pre-training control values. In contrast, muscle convective O ₂ transport increased significantly only in controls (by ~26% from 688±57 to 865±69 mL·min ⁻¹), leaving patients with COPD (438±45 versus 491±51 mL·min ⁻¹) at ~70% of pre- training control values. While muscle diffusive O ₂ transport in COPD was largely restored by exercise training, V'_{mO_2} peak remained constrained by limited plasticity in muscle convective O ₂ transport.