Vascular Dysfunction in Chronic Obstructive Pulmonary Disease (COPD): The Role of Mitochondrial-derived Oxidative Stress

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ABSTRACT

Background: This study sought to determine the contribution of mitochondrial-derived oxidative stress in the vascular dysfunction exhibited by patients with COPD.
Methods: Vascular function was assessed with brachial artery flow-mediated vasodilation (FMD) and the hyperemic response to both single and continuous passive leg movement (PLM) in 10 patients with COPD (55-70 years) before and after both the acute and chronic ingestion (4 weeks) of a mitochondrial-targeted antioxidant (MitoQ) (20mg/day).

Results: Baseline % FMD (2.5 ± 0.9 %) in the patients with COPD was significantly enhanced by both the acute (3.3 ± 0.8 %) and 4 week (5.3 ± 1.2 %) MitoQ consumption. Acute MitoQ had no effect on the single PLM-induced change in peak LBF (ΔPeak) or the area under the leg blood flow (LBF) response curve (AUC), but the 4 week MitoQ consumption significantly augmented both the single PLM ΔPeak (190±184 vs. 300±96 ml/min) and LBF AUC (20±14 vs. 50±16 ml). Compared to baseline (ΔPeak = 300±84 ml/min; LBF AUC = 80±34 ml), both ΔPeak and LBF AUC during continuous PLM-were significantly enhanced with both the acute (ΔPeak: 435±96 ml/min; LBF AUC: 105±56 ml) and 4 week (ΔPeak: 622±106 ml/min; LBF AUC: 300±120 ml) MitoQ consumption.

Conclusions: This study reveals that, in patients with COPD, mitochondrial-derived oxidative stress contributes significantly to the vascular dysfunction exhibited by this population. Therefore, targeting mitochondrial-derived oxidative stress may, through an improvement in vascular function, be an efficacious approach to combat cardiovascular disease in patients with COPD.