

In vitro ubiquinol increases cellular oxygen consumption in peripheral blood mononuclear cells from patients with metabolic stress

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Introduction:

The objective of the current study was to investigate the effects of *in vitro* ubiquinol (coenzyme Q10) administration on cellular oxygen consumption in peripheral blood mononuclear cells (PBMCs) from patients undergoing coronary artery bypass grafting. Ubiquinol (Coenzyme Q10) is a mitochondrial molecule that is essential for adequate aerobic metabolism. This population was evaluated to capture pre- and post blood sampling for patients in a state of severe metabolic stress.

Methods:

Patients scheduled for coronary artery bypass grafting with cardiopulmonary bypass were enrolled at Beth Israel Deaconess Medical Center, a tertiary care center in Boston, USA, between January 2015 and July 2015. Blood was drawn and PMBCs were isolated from the patient before and after surgery. Cells were then randomized to either treatment with placebo or 1 µg/mL ubiquinol. The complete mitochondrial respiration profiles were measured using XF Cell Stress Mito Kit (Seahorse Bioscience) to reveal the key parameters of cellular oxygen consumption. Wilcoxon Signed Rank test was used to analyze differences in oxygen consumption rate between groups.

Results:

Basal cellular oxygen consumption was available on 23 patients pre-operatively and 17 patients post-operatively. The mean age was 71 (SD: 7), and 22/26 (85%) were male. We found a significant difference in post-operative relative basal (1.1 mL/min/mg difference [0.9, 1.6] $p < 0.001$) oxygen consumption and maximal (4.2 mL/min/mg difference [0.3, 7.0] $p = 0.01$) oxygen consumption between the ubiquinol and placebo group. There were no significant differences in pre-operative basal (1.0 mL/min/mg [-0.9, 2.2] $p = 0.08$) or maximal (0.5 mL/min/mg [-4.3, 7.3] $p = 0.56$) cellular oxygen consumption between the ubiquinol and placebo.

Conclusions:

In a sample of cardiac surgery patients, *in vitro* administration of ubiquinol enhanced post-operative cellular oxygen consumption. These findings suggest ubiquinol may have potential as a mitochondrial resuscitator in states of metabolic stress, akin to typical physiology for critically ill patients in a state of shock.