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Decrease in CoQ by statin treatment does not attenuate exercise-induced adaptations in the myocardium

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Background & Aim: A side effect of cholesterol lowering statins is lowered coenzyme Q (CoQ), a component of the mitochondrial Electron Transport Chain (ETC) with antioxidant properties. Many statin patients take a CoQ supplement to counter CoQ lowering. Is this necessary? The ETC CoQ content is far greater than all other electron carriers. The purpose of this study was to test the hypothesis that the decrease in CoQ associated with high-dose statin treatment is not sufficient to affect cardiac function or the ability of the heart to adapt to exercise training.

Methodology: Female S-D rats were divided into four groups (8-10/group): sedentary (S), S+Simvastatin (S+ST) (10 mg/kg/day orally), exercised on a treadmill for 5 days/wk up a 6° incline for 5 weeks (E) or E+ST. By week 5 animals were running 1 hr at 30 m/min (approximately 80% Vo2max). Twenty-four hours after the last session, myocardial hemodynamic performance and oxygen consumption was evaluated using an isolated perfused working heart system. After storage at -80°C, ventricular oxidized and reduced COQ9 and COQ10 were measured by high performance liquid chromatography with electrochemical detection.

Findings: COQ9 represented approximately 91% of the total myocardial COQ pool in all groups. Compared to S, total COQ9 was decreased 30.0% in S+ST and 23.8% in E+ST ($P<0.05$). Approximately 56% of COQ9 was in reduced form in all groups; whereas COQ10 was more reduced ($P<0.05$) in the statin-treated groups. Exercise training resulted in similar increases ($P<0.05$) in E and E+ST for cardiac hypertrophy, cardiac output, and external cardiac work. Furthermore, myocardial oxygen consumption was coupled to ATP demand similarly in all groups.

Conclusion & Significance: The magnitude of COQ decrease by simvastatin treatment is not sufficient to affect mitochondrial oxidative phosphorylation, cardiac function or the ability of the heart to adapt to the challenge of exercise training. COQ supplementation is not necessary.

Biography

Joseph W Starnes is the Director of the Cardiac Metabolism Lab at the University of North Carolina at Greensboro. Work in his laboratory centers on understanding function, metabolism, and intrinsic adaptations of the heart. He is particularly interested in how exercise and certain pharmaceutical drugs protect the heart so that it can perform well under normal and very stressful conditions well into old age. The primary experimental model used in my lab is the laboratory rat. He studies the function and metabolism in the intact heart using an isolated perfused working heart system. This system is similar to a heart-lung apparatus. He also carries out studies on the heart's mitochondria because they play a crucial role in deciding whether a heart cell will survive or die.

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