Plasma Coenzyme Q10 Predicts Lipid-lowering Response to High-Dose Atorvastatin.

Pacanowski MA, Frye RF, Enogieru O, Schofield RS, Zineh I.

University of Florida College of Pharmacy, Department of Pharmacy Practice and Center for Pharmacogenomics; Gainesville, FL, USA.

BACKGROUND: Coenzyme Q10 (CoQ10) is a provitamin synthesized via the HMG-CoA reductase pathway, and thus may serve as a potential marker of intrinsic HMG-CoA reductase activity. HMG-CoA reductase inhibitors (statins) decrease CoQ10, although it is unclear whether this is due to reductions in lipoproteins, which transport CoQ10.

OBJECTIVES: We evaluated whether baseline plasma CoQ10 concentrations predict the lipid-lowering response to high-dose atorvastatin, and to what extent CoQ10 changes following atorvastatin therapy depend on lipoprotein changes.

METHODS: Individuals without dyslipidemia or known cardiovascular disease (n=84) received atorvastatin 80 mg daily for 16 weeks. Blood samples collected at baseline and after 4, 8, and 16 weeks of treatment were assayed for CoQ10.

RESULTS: Individuals with higher baseline CoQ10:LDL-C ratios displayed diminished absolute and percent LDL-C reductions at 8 and 16 weeks of atorvastatin treatment (P<0.001 to 0.01). After 16 weeks of atorvastatin, plasma CoQ10 decreased 45% from 762±301 ng/ml to 374±150 ng/ml (P<0.001). CoQ10 changes were correlated with LDL-C and apolipoprotein B changes (r=0.27-0.38, P=0.001-0.02), but remained significant when normalized to all lipoproteins. CoQ10 changes were not associated with adverse drug reactions.

CONCLUSION: Baseline CoQ10:LDL-C ratio was associated with the degree of LDL-C response to atorvastatin. Atorvastatin decreased CoQ10 concentrations in a manner that was not completely dependent on lipoprotein changes. The utility of CoQ10 as a predictor of atorvastatin response should be further explored in patients with dyslipidemia.

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