

# Abstract

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

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**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  $762 \pm 301$  ng/ml to  $374 \pm 150$  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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