

# Abstract

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## Ubiquinol-induced gene expression signatures are translated into altered parameters of erythropoiesis and reduced low density lipoprotein cholesterol levels in humans.

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**OBJECTIVE AND METHODS:** Studies in vitro and in mice indicate a role for Coenzyme Q(10) (CoQ(10)) in gene expression. To determine this function in relationship to physiological readouts, a 2-week supplementation study with the reduced form of CoQ(10) (ubiquinol, Q(10) H(2)), 150 mg/d) was performed in 53 healthy males.

**RESULTS:** Mean CoQ(10) plasma levels increased 4.8-fold after supplementation. Transcriptomic and bioinformatic approaches identified a gene-gene interaction network in CD14-positive monocytes, which functions in inflammation, cell differentiation, and peroxisome proliferator-activated receptor-signaling. These Q(10) H(2)-induced gene expression signatures were also described previously in liver tissues of SAMP1 mice. Biochemical and NMR-based analyses showed a reduction of low density lipoprotein (LDL) cholesterol plasma levels after Q(10) H(2) supplementation. This effect was especially pronounced in atherogenic small dense LDL particles (19-21 nm, 1.045 g/L). In agreement with gene expression signatures, Q(10) H(2) reduces the number of erythrocytes but increases the concentration of reticulocytes.

**CONCLUSIONS:** In conclusion, Q(10) H(2) induces characteristic gene expression patterns, which are translated into reduced LDL cholesterol levels and altered parameters of erythropoiesis in humans.

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