› Eur J Pharmacol. 2013 Jul 5;711(1-3):1-9. doi: 10.1016/j.ejphar.2013.04.009. Epub 2013 Apr 24.

Ubiquinol rescues simvastatin-suppression of mitochondrial content, function and metabolism: implications for statin-induced rhabdomyolysis

Roger A Vaughan [1], Randi Garcia-Smith, Marco Bisoffi, Carole A Conn, Kristina A Trujillo

Affiliations + expand

PMID: 23624330 DOI: 10.1016/j.ejphar.2013.04.009

Abstract

Statin medications diminish cholesterol biosynthesis and are commonly prescribed to reduce cardiovascular disease. Statins also reduce production of ubiquinol, a vital component of mitochondrial energy production; ubiquinol reduction may contribute to rhabdomyolysis. Human rhabdomyosarcoma cells were treated with either ethanol and dimethyl sulfoxide (DMSO) control, or simvastatin at 5 μM or 10 μM, or simvastatin at 5 μM with ubiquinol at 0.5 μM or 1.0 μM for 24 h or 48 h. PGC-1α RNA levels were determined using quantitative reverse transcriptase polymerase chain reaction (qRT-PCR). Mitochondrial content was determined using flow cytometry and immunocytochemistry. Metabolism was determined by quantification of extracellular acidification rate and oxygen consumption rate. Treatment of human rhabdomyosarcoma cells with simvastatin significantly reduced oxidative, total metabolism, and cellular ATP content in a time- and dose-dependent manner which was rescued by concurrent treatment with ubiquinol. Treatment with simvastatin significantly reduced mitochondrial content as well as cell viability which were both rescued by simultaneous treatment with ubiquinol. This work demonstrates that the addition of ubiquinol to current statin treatment regimens may protect muscle cells from myopathies.