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Inhibition of cholesterol synthesis increases secretion of apolipoprotein E (apoE) from human astrocytoma and liver hepatocyte cells

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Apolipoprotein E (apoE) genotype is the biggest genetic risk factor Alzheimer's disease (AD). ApoE is the primary lipid carrier in the brain, and is involved in brain cholesterol homeostasis and neuronal repair. It is thought that the e4 isoform increases risk for AD due to inadequate neuronal repair capabilities, and other deleterious effects such as lysosomal dysfunction and aggregation with Abeta peptide.Apomine and simvastatin are potent cholesterol-reducing compounds that mediates their actions through inhibition of HMGCoA-reductase and activation of SREBP2. We have found that under serum-free conditions, these two compounds increase apoE secretion and increase expression of LDLR and ApoER2 (apoE receptors) from human HepG2 liver hepatocyte cells. Addition of LDL to these treatments in serum-free medium suppressed induction of apoE by these compounds.Consistently, we found a strong negative correlation between cellular cholesterol levels and secreted apoE. Our results suggest that apomine and simvastatin regulate apoE expression at both the transcriptional and post-translational levels, and that these effects seem to be indirectly through alterations in cholesterol levels.

Biography

Nilay Patel received his Ph. D. in Neurobiology and Behavior from SUNY Stony Brook. He was a post-doctoral fellow in Dr. Caleb Finch's group at USC, and a Beckman Research Fellow in Dr. Barry Forman's group at City of Hope. He is an Associate Professor in Department of Biological Science at California State University, Fullerton, and Director of CIRM-funded Bridges to Stem Cell Research. His research focuses on identification of novel regulators of apoE gene expression.

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