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Anti-inflammatory Effects of High-Density Lipoprotein via Regulation of Nitric Oxide Synthase Expression and Nf-κb Transcription in Activated Human Endothelial Cells

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ABSTRACT

Oxidation of low-density lipoprotein (LDL) and activation of the transcription factor nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) are critical for the inflammatory response for endothelial dysfunction. The objective of this study is to investigate the effects of various doses of HDL on: (a) LDL susceptibility to oxidation; (b) expression of eNOS; and (c) expression of NF-κB p50 and p65. Different concentrations of HDL were incubated in LDL. The reaction rates of LDL susceptibility to oxidation were obtained by kinetic modeling analysis. For determination of eNOS, NF-κB p50 and p65 expression, different HDL concentrations were incubated in lipo polysacharides (LPS)-stimulated human umbilical vein endothelial cell line for 16 hours. Protein was extracted and analysed by western blot and nuclear transcription factor, for example, Co-incubation of LDL with increasing HDL concentrations showed longer lag time and lower reaction rate in a dose-dependent manner compared to controls ($p<0.05$). The eNOS expression at higher HDL concentration was significantly increased when compared to controls ($p<0.05$). HDL significantly decreased the expression of NF-κB p65 but not that of NF-κB p50. HDL protects LDL from oxidation, up regulates eNOS expression and down regulates the expression of NF-κB p65. These in part contribute to the role of HDL in the prevention and retardation of atherogenesis and atherosclerosis-related complications.

Keywords: eNOS, NF-κB, endothelial cells, HDL, LDL oxidation, protein expression

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