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The Signal Transduction Pathways Involve Shear Stress-induced Expression of IL-8 mRNA in Human Endothelial Cells

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1 Introduction

The objective of this study was to investigate the possible signal transduction pathways involved in the low shear stress-induced IL-8 mRNA expression in cultured human umbilical vein endothelial cells (HUVECs).

2 Materials and Methods

Light Cycler™ System was employed to assay the expression of IL-8 mRNA of HUVECs quantitatively. The expression of IL-8 mRNA in HUVECs stimulated by low shear stress (4.20 dyne/cm^2) was compared with that stimulated by the same shear stress pretreated with one kind of inhibitors respectively. The inhibitors included cAMP-dependent protein kinase A (PKA) inhibitor KT5720, cGMP-dependent protein kinase G (PKG) inhibitor KT5823, phospholipase C (PLC) inhibitor Neomycin, protein kinase C (PKC) inhibitor Calphostin C, intracellular Ca^{2+} chelator BPTPA/AM, extracellular Ca^{2+} chelator EGTA, Ca^{2+} channel blockor Verapamil, tyrosine protein kinase inhibitor Tyrphostin-25, and G protein inhibitor GDP-βS.

3 Results

(1) Pretreatment HUVECs with Tyrphostin-25, Calphostin C, KT5720, or Neomycin inhibited the low shear stress induced IL-8 mRNA expression partially; (2) EGTA, or BATPA/AM, or Verapamil also partially suppressed up-regulated of IL-8 gene induced by the low shear stress; (3) G-protein involved in the low shear stress induced IL-8 mRNA expression; and (4) pretreatment HUVECs with cGMP-dependent protein kinase inhibitor KT5823 had no effect on IL-8 mRNA expression induced by the low shear stress.

4 Conclusion

(1) Several signal molecules, for example, tyrosine protein kinase, protein kinase C, Ca^{2+}, PKA, and PLC were involved in the different extent of IL-8 mRNA expression induced by low shear stress; (2) activation of G-protein is necessary in the process; and (3) PKG is not likely involved in the process.

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