The effect of Ibuprofen on bFGF release and cell viability in the presence of LPS in HMEC-1 cells

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Aim: Bacterial lipopolysaccharide (LPS) is a major cell wall component of Gram-negative bacteria and it is a key molecule in the initiation of local and systemic inflammation and septic shock. LPS induces endothelial sprouting and causes the release of inflammatory cytokines such as interleukin-1 (IL-1), IL-6, IL-8 and tumor necrosis factor alpha (TNF-α), as well as growth factors including VEGF and bFGF. The aim of this study was to check whether ibuprofen can modulate the level of bFGF in the presence of LPS in human endothelial HMEC-1 cells.

Methods: Human microvascular endothelial cells (HMEC) were treated with 100 µM and 1 mM ibuprofen in the presence of 100 µg/ml LPS. The effect of NSAID and LPS on bFGF proteins was analyzed by ELISA kit (R&D Systems). Cell viability was measured with the use of 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide (MTT) method.

Results: LPS at concentration of 100 µg/ml evoked up-regulation of basic fibroblast growth factor (bFGF) by 53% (p<0.05) and it also enhanced the proliferation of HMEC-1 cells. In our studies ibuprofen was chosen as non-selective cyclooxygenase (COX) inhibitor to study whether it can influence the generation of bFGF in the presence of bacterial lipopolysaccharide. Ibuprofen (at concentration of 100 µM and 1 mM) inhibited in statistically significant manner the secretion of bFGF evoked by LPS by 27% and 62%, respectively. Non-selective COX inhibitor concentration-dependently decreased cell viability. These observed effects also occured when cells were treated with Ibuprofen in the presence of LPS.

Conclusion: Our findings prove that non-selective COX inhibitor weakens proliferative effect of LPS, that may indicate the participation of COX and their products in proliferation of HMEC-1 cells stimulated by LPS. The inhibition of bFGF secretion by ibuprofen can also indicate the participation of COX in the production of growth factor.

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