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혈관내피세포에서 TNF-α에 의한 Fractalkine 발현에 대한 Epigallocatechin-3-O-gallate (EGCG)의 억제효과

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Epigallocatechin-3-O-gallate Decreases Tumor Necrosis factor-α-induced Fractalkine Expression in Endothelial Cells with NF-kB Suppression

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Epigallocatechin-3-O-gallate (EGCG), the main catechin in green tea, has been shown to have anti-oxidant, anti-atherosclerotic, and anti-inflammatory activities. Fractalkine is one of the chemokines involved in inflammation and the early atherosclerosis process, and it is a unique in that it functions as a chemoattractant as well as an adhesion molecule in endothelial cells activated by proinflammatory cytokines. In the present study, we investigated the mechanism by which EGCG affects fractalkine expression in TNF-a-induced human umbilical vein endothelial cells (HUVECs). EGCG decreased TNF-a-induced fractalkine mRNA and protein expression in HUVECs in a dose- and time-dependent manner. EGCG decreased TNF-a-induced phosphorylation and nuclear translocation of the NF-κB p65 subunit in HUVECs. The DNA binding activity of the NF-κB p65 subunit was also decreased in HUVECs treated with both EGCG and TNF-a in comparison to HUVECs treated with TNF-a alone. EGCG inhibited monocytes adhesion to HUVECs stimulated by TNF-a. These results demonstrate that EGCG prevents vascular TNF-a-induced endothelial fractalkine expression.

Key Words : 혈관내피세포, 염증반응, EGCG
Endothelial cells, Inflammation, EGCG