NF-κB inhibitor BAY11-7085 suppresses the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 in TNF-α-stimulated human endometriotic stromal cells

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목적: The cell adhesion molecules such as intercellular cell adhesion molecule (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) are representative examples of the molecules involved in the complex events of the crosstalk. A number of mediators including cell adhesion molecules such as ICAM-1 and VCAM-1, pro-inflammatory cytokines such as TNF-α, interleukin-1 (IL-1), IL-6 and IL-8, and chemokines such as monocyte chemotactic protein-1 (MCP-1) play a key role in the pathogenesis of endometriosis. BAY11-7085 is a pharmacological inhibitor of NF-κB transcription factor, a crucial regulator of inflammation.

방법: To explore effect of BAY11-7085 on the expression of these critical molecules involved in the pathophysiology of endometriosis in TNF-α-stimulated human ectopic endometrial stromal cells (HEESCs) isolated from patients with endometriosis.

결과: BAY11-7085 did not affect HEESCs viability up to a dose of 10 μM. Treatment of HEESCs with BAY11-7085 for 48h significantly inhibited TNF-α-induced proliferation of HEESCs. HEESCs treated with BAY11-7085 showed markedly suppressed TNF-α-induced mRNA expression of ICAM-1 and VCAM-1 as assessed by quantitative realtime RT-PCR. BAY11-7085 treatment also significantly decreased the TNF-α-induced cell surface and total protein expression of ICAM-1 and VCAM-1 as examined by flow cytometry, immunofluorescent microscopy and western blot. In addition, treatment of HEESCs with BAY11-7085 inhibited TNF-α-induced secretion of IL-6, IL-8 as analyzed by ELISA.

결론: NF-κB inhibitor BAY11-7085 is a potential anti-endometriotic agent.