AAT 유전자와 mTOR pathway 관련 유전자들의 발현이 조절되며, 체외배양과정의 적절하지 못한 배양 조건에 의해 이러한 유전자들의 발현 양상이 변화됨을 알 수 있었다. 따라서 체외배양 조건의 최적화와 특정 배양액의 개발에 있어 관련 유전자의 발현 양상 변화에 대한 연구가 유용할 것으로 생각된다.

O-18(기초) Peroxisome Proliferator-Activated Receptor-g (PPAR-g) Inhibits TGF-b Induced Decidualization in Human Endometrial Stromal Cells

Chang HJ[1], Lee JH[1], Kim MR[1], Park DW[2], Hwang KJ[1]

[1]Department of Obstetrics and Gynecology, Ajou University School of Medicine,
[2]Department of Biology, College of Natural Science, Ajou University

Background & Objectives: It is well recognized that the endometrial decidualization is crucial for implantation and maintenance of pregnancy. TGF-b plays an important role in endometrial stromal cell in decidualization. PPARs are members of the nuclear receptor superfamily, regulates gene expression in a variety of cells. Recently PPAR-g is accepted by the one of the novel receptor that is associated with implantation and menstrual physiology. We investigated the effect of PPAR-g agonist on TGF-b induced decidulaization.

Method: The endometrium was obtained from mid to late proliferative phase women who had no gynecologic diseases by currettage. Then, the endometrial stromal cells cultured for 48 hours under the following hormonal conditions (100 nM P4 and 1 nM E2). TGF-β1 (2.5 ng/ml) or PPAR-g agonist (Rosiglitazone®; 50 nM) were added when necessary. We evaluated the TGF-β1 signaling pathway- ERK, COX-2, PGE2, and ERK inhibitor (PD98059) were added. Western blotting, ELISA were utilized to detect the proteins quantitatively.

Results: TGF-b induced the endometrial decidualization with increasing expression of the p-ERK, COX-2 and prolactin, PGE2, but leaded to down-regulation of PPAR-g expression. The PPAR-g agonists down-regulated the p-ERK, COX-2 and prolactin expression and PGE2 releasing in cultured endometrial stromal cells. When endometrial stromal cells treated with ERK inhibitor (PD98059), stromal cell decidualization was decreased with down-regulation of COX-2, PGE2.

Conclusions: TGF-b lead to endometrial stromal cells decidualization through ERK pathway. Peroxisome Proliferator-Activated Receptor-g (PPAR-g) inhibit TGF-b induced decidulaization by ERK pathway down-regulation in human endometrial stromal cells.