시행한 경우 임신율과 다태임신에 어떠한 영향을 미치는지를 보기 위하여 시행하였다. 본 연구는 1998년 10월부터 1999년 9월까지 인천 서울산부인과 불임연구실에서 시행되었던 체외수정시술중 3일째 배아이식을 시행하고 여분의 배아를 5일째 까지 배양하여 동결보존한 군 21주기와 (ICSI 6주기 포함) (Group 1), 3일째 배아이식과 함께 5일째 배아이식을 시행한 군 (Group 2) 52주기 (ICSI 18주기 포함)를 대상으로 하였다. 과배란유도 방법은 GnRH-a를 이용한 Long protocol로 하였다.

	Group 1 (n=21)	Group 2 (n=52)	P value
Age	31.8±3.4	32.7±3.5	0.21
ertilization rate	83.3±9.6	78.2±16.6	0.29
Cleavage rate	97.0±4.7	97.2±3.4	0.17
No.of D3ET	4.9±0.9	4.0±0.5	0.00
Blastulation rate	53.8±29.5	43.6±23.8	0.21
Clinical pregnancy rate (/cycle)	6 (28.6%)	14 (26.9%)	0.88 (χ^2)
Multiple pregnancy rate (/preg)	2 (33.3%)	8 (57.1%)	0.60 (χ^2)
Triple pregnancy	0	1	

양 군에서 수정율, 배아분활율 및 임상적 임신율은 통계학적인 차이가 없었다. 이상의 결과로 3일째와 5일째 두 번 배아이식을 시행한다고 해서 임신율이 증가되지 않았으며, 오히려 다태임신의 위험성이 증가하는 경향을 보였으므로 동시 배아이식 보다는 한번의 배아이식을 시행하는 것이 좋을 것으로 사료된다.

P-45 Role of Progesterone Receptor in LH-stimulated Pituitary Adenylate Cyclase-Activating Polypeptide Gene Expression in the Rat Ovary

Hyun-Jeong Park*[1], Yu-Il Lee[1], Jae-Il Park[2], Jin Lee[2] and Sang-Young Chun[2]

[1]Department. of Ob/Gyn, College of Medicine and [2]Hormone Research Center, Chonnam National University, Kwangju 500-757

Pituitary adenylate cyclase-activating polypeptide (PACAP), a novel neuropeptide with considerable homology to vasoactive intestinal peptide and growth hormone-releasing hormone, has been shown to be stimulated by luteinizing hormone (LH) in rat preovulatory follicles (*Endocrinology* 1999 140:818). In the present study, we further examined a mediatory role of progesterone receptor (PR) in LH-induced PACAP gene expression in immature rat ovary. Injection (i.p.) of RU486, a PR antagonist, 1 hr before human chorionic gonadotropin (hCG) treatment to PMSG-primed immature rats suppressed hCG-stimulated PACAP gene expression in a dose-dependent manner revealed by Northern blot analysis. Administration of epostane, an inhibitor of 3-beta-hy-

droxysteroid dehydrogenase, also inhibited hCG-induced PACAP gene expression. Similarly, in preovulatory follicles cultured in serum-free medium, treatment with both RU486 and epostane inhibited LH-induced PACAP gene expression in a dose-dependent manner. We further evaluated the role of PR in the control of PACAP promoter activity by transfections of preovulatory granulosa cells with PACAP promoter gene using *luciferase* gene reporter system. Treatment with LH resulted in a marked increase (10-fold) in PACAP promoter activity. Addition of RU486 or epostane dose-dependently inhibited LH action on the stimulation of PACAP promoter activity. The present results indicate that LH exerts its action on the induction of PACAP gene expression through the endogenous activation of PR in rat preovulatory follicles.

P-46 Role of MAP Kinase Activation in Gonadotropin-induced Expression of Pituitary Adenylate Cyclase-Activating Polypeptide Gene in Preovulatory Follicles of Rat Ovary

Wan-Ju Kim*, Jeong-Hoh Park, Wang-Li, Hyuk-Bang Kwon and Sang-Young Chun

Hormone Research Center, Chonnam National University, Kwangju 500-757

Pituitary adenylate cyclase-activating polypeptide (PACAP) is a novel neuropeptide with considerable homology to vasoactive intestinal peptide and growth hormone-releasing hormone. Because we have shown previously that gonadotropins induce a transient induction of PACAP in preovulatory follicles (*Endocrinology* 1999 140:818), the present studies evaluated the role of MAP kinase activation in gonadotropin action on the induction of PACAP gene expression in cultured rat preovulatory follicles obtained from ovaries treated with PMSG for 2 days. Treatment of cultured follicles with PD98059, an inhibitor of ERK, suppressed the LH-stimulated PACAP gene expression in a dose-dependent manner. Similarly, treatment with SB203580, an inhibitor of p38 kinase, also suppressed the LH-stimulated PACAP gene expression. Western analysis using phospho-specific antibodies revealed that activation of ERK occurred between 3-6 h after LH treatment. In contrast, the basal levels of the activated forms of p38 kinase and JNK was high at the time of incubation (0 h) and remained constant untill 3 h after LH treatment. Assay of MAP kinase activity demonstrated an increase within 10 min. and at 3 h after LH treatment. The present results indicate that the activation of MAP kinase, possibly ERK, is necessary for LH action in the induction PACAP gene in preovulatory follicles.