서 자궁내막증이 없는 환자와 차이가 있을 수 있는 것으로 제시되고 있다. 본 연구는 자궁내막을 포함한 여러 조직에서 맥관형성과 세포 증식에 관여하는 것으로 확인된 섬유아세포 성장인자-2 (fibroblast growth factor-2, FGF-2)와 표피 성장인자 (epidermal growth factor, EGF) mRNA의 발현 양상을 자궁내막증 환자의 자궁내막에서 분석하고 이를 대조군과 비교하고자 고안되었다.

**Method:** 개복 또는 복강경 수술을 통하여 자궁내막증으로 확진된 환자 32명과 자궁내막증이 없으며 가임능력이 확인된 28명의 환자를 대상으로 하였다. 모든 환자들의 자궁내막에서 RNA를 추출, 역전사시킨 다음, 실시간 중합효소연쇄반응을 이용하여 FGF-2와 EGF 유전자의 발현량을 GAPDH의 발현량에 대한 상대적 수치로 표준화하여 비교, 분석하였다.

**Results:** 모든 생리주기를 통합하여 분석한 결과와 증식기, 분비기 결과에 있어서 자궁내막증군과 대조군의 자궁내막에서 FGF-2와 EGF 유전자의 발현에는 유의한 차이가 없었다. 6개의 생리주기별로 나누어 분석한 결과에서도 모든 주기에서 환자군과 대조군간에 FGF-2와 EGF 유전자의 발현에 유의한 차이가 없었다.

**Conclusions:** 본 연구의 결과를 볼 때, 자궁내막증 환자의 자궁내막 내 FGF-2와 EGF의 발현은 가임능력이 확인된 대조군과 차이가 없음을 확인할 수 있었다. 이러한 소견은 자궁내막증의 병인과 병태생리에는 FGF-2와 EGF 이외의 다른 요인들이 관여할 수 있음을 시사하는 것으로 사료된다.

## P-6 p57kip2 was Involved in the Inhibition of Proliferation and the Differentiation of Human Endometrial Stromal Cells

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**Background & Objectives:** Regulation of endometrial growth and differentiation through the activation and inactivation of different cyclin-Cdks (cyclin-dependent kinases) at appropriate times is needed for normal menstrual cycle, implantation and successful pregnancy. This study was to investigate whether p57kip2 could be involved in the normal menstrual cycle and pathogenesis of human endometrium, endometrial hyperplasia and endometrial cancer.

**Method:** The expression and localization of p57kip2 was assessed by semi-quantitative RT-PCR and immunohistochemistry in human endometrium during the normal menstrual cycle and pathological tissues, endometrial hyperplasia and cancer. For induction of in vitro decidualization, isolated endometrial stromal cells (ESC) were cultured with 10 nM 17b-estradiol and 1000 nM progesterone for 14 days. The expression of p27kip1, p57kip2 and prolactin was examined by semi-quantitative RT-PCR.

**Results:** During the menstrual cycle, mRNA level of p27kip1 and p57kip2 is significantly increased in mid-secretory phase. Expression of p57kip2 protein was detected in neither glandular nor stromal cells at proliferative phase, endometrial hyperplasia and cancer. From early to mid-secretory phase, the expression of p57kip2 in glandular cells was significantly increased. In stromal cells of late secretory phase, the intensity of the staining for p57kip2 was much stronger than that of early and mid-secretory phase. mRNA

of p27kip1 and p57kip2 was gradually increased during in vitro decidualization of ESC induced by steroid hormones.

**Conclusions:** These results suggest that p57kip2 may play an important role in endometrial proliferation and differentiation, in growth inhibition of malignant glandular cells, and in decidualization of stromal cells by steroid hormones during the late secretory phase.

## P-7 The Expression of p57kip2 in Mouse Testis During Postnatal Development and Adult Human Testis with Various Defects

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**Background & Objectives:** Regulation of cellular growth and differentiation through the activation and inactivation of different cyclin-Cdks (cyclin-dependent kinases) at appropriate times is needed for normal development of testis. This study was to investigate the expression of CDK inhibitors, p57kip2 during the postnatal growth and differentiation of mouse testis, and in adult human testis with various defects.

**Method:** The expression and localization of p57kip2 was assessed by semi-quantitative RT-PCR and immunohistochemistry in mouse testis during post natal development. And localization of p57kip2 was exained by immunohistochemistry in adult human testis with verious defects, which are non-obstructive azoospermia, spermatogenic hypoplasia, Sertoli cell-only syndrome, and testicular cancer.

**Results:** mRNA expression of p57kip2 was higher in immature (7 and 14 days after birth) testis than pubertal (28 days) or adult (50 days) mouse testis. In 7 days mouse testes, moderate p57kip2 immunoreactivity was largely found in spermatogenic and somatic cells in the seminiferous tubules. In 14 days mouse testes, intensive immunoreactivity of p57kip2 was found in spermatogonia. In pubertal mouse testes, p57kip2 immunoreactivity was very strong in nucleus of some spermatogonia and Leydig cells. Also in adult mouse testes, intensive immunoreactivity of p57kip2 was found in nucleus of some spermatogonia and Leydig cells. In normal human testis, very intensive immunoreactivity of p57kip2 was found in nucleus of many spermatogonia. Also in non-obstructive azoospermic testis, some spermatogonia were strongly stained. In seminiferous tubule of spermatogenic hypoplasia, p57kip2 was weakly expressed in some spermatogonia. However, in the seminiferous tubule of Sertoli cell-only syndrome and testicular cancer patients, there was no visible sign of p57kip2 expression.

**Conclusions:** These results suggest that the role of p57kip2 might be required for the postnatal development of mouse testis and that p57kip2 in human testis is involved in the differentiation of spermatogonia, and in growth inhibition of malignant germ cells.