S3-01

The regulation of *in vitro* maturation of oocytes: consideration of circadian genes

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In vitro maturation (IVM) of human oocytes is an emerging infertility treatment with great promise. Recent studies demonstrate that changes, occurred during growth and maturation of the M Ⅱ of immature oocytes, greatly affect further embryonic development and clinical outcome. Therefore, age, date of menstrual cycle, and cyclicity of the patients are important factors influencing growth of retrieved oocytes as well as number of recovered ones. To be successful, this novel concerning assisted reproductive technology must entail both nuclear and cytoplasmic maturation of oocytes. Follicle size, oocyte diameter, and hormonal treatment affect cytoplasmic maturation of oocytes. In addition, the type of media, hormones, growth factors, energy substrates, protein sources, scavenging of reactive oxygen species and meiosis activating sterols, have been considered improving nuclear and cytoplasmic maturation during *in vitro* maturation. In addition, various functional genes must be considered. Especially, we focused on the new hypothesis regarding the relationship between circadian clock genes and regulation of oocyte maturation.

So far, the success rate of *in vitro* maturation program is still low, because of asynchronous implantation, maturation failure, and no reliable markers for assessing oocyte. Without detailed knowledge of *in vivo* processes that regulate oocyte nuclear and cytoplasmic maturation and embryonic developmental competence it will be quite difficult to understand and to remedy the shortcomings within the *in vitro* maturation systems. For this reason, it is imperative that the analysis of specific gene expression and function of genes during maturation should continue.

S3-02

Candidate Gene Analysis in Korean Patients with Endometriosis

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Endometriosis is defined as the growth of endometrium outside the uterine cavity, usually in the peritoneal cavity, which can cause dysmenorrhoea, dyspareunia, pelvic pain, and infertility. It is a common disease with a prevalence of 0.5 to 5% in fertile and 25 to 40% in infertile women. The most widely accepted aetiological theory is that retrograde flow of menstrual fluid through the Fallopian tubes deposits viable endometrial tissue, which implants on the peritoneal surface. Although retrograde menstruation is very common phenomenon in menstruating women, not all women develop endometriosis. Therefore additional causative factors should be present in a susceptible woman to develop endometriosis. There is increasing evidence of a genetic susceptibility of an individual to endometriosis: familial clustering, concordance in monozygotic twins, increased prevalence among the first-degree relative of women with endometriosis compared to the control group. We have analysed many polymorphisms of genes that are thought to be involved in the pathogenesis of endometriosis. The polymorphisms we have found a significant association with endometriosis in the Korean population are as follows: GSTM1 polymorhism, estrogen receptor dinucleotide polymorphism, and p53 codon 72 polymorphism. Of notice was that estrogen receptor dinucleotide polymorphism and p53 codon 72 polymorphism were associated only with mild (stage I-II) endometriosis, not with severe (stage III-IV) endometriosis. These results suggest a difference in genetic susceptibility to develop endometriosis between mild and severe endometriosis. And the polymorphisms which we have found no association with endometriosis in the Korean population were *Pvu*II or *Xba*I polymorphism of estrogen receptor, GSTT1 polymorphism, NAT2 polymorphism, interleukin-6 gene (-174) promotor polymorphism, interleukin-10 -627 gene promoter polymorphism, MMP-9 exon 6 polymorphism, and progesterone receptor (PROGINS) polymorphism. In conclusion, to reveal the genetic profiles of endometriosis patients will enhance our understanding of the histogenesis of endometriosis.