262 THE INVOLVEMENT OF PLATELET ACTIVATING FACTOR IN THROMBOCYTOPENIA AND FOLLICULAR RUPTURE DURING GONADOTROPIN-INDUCED SUPEROVULATION IN IMMATURE RAT. X.M. Li, N. Sagawa, Y. Ihara, A. Okagaki, M. Hasegawa, H. Itoh, K. Inamori, T. Mori and C. Ban*, Depts. Gynecol. Obstet., Kyoto Univ., Kyoto, * Dept. Obstet. Gynecol., National Hospital of Osaka, Osaka.

We studied changes in the platelet count of immature rats after the ovulation induction by PMSG and hCG. The effect of PAF antagonist, Y24180, on the platelet count and the number of ova shed was also examined.

Platelet count in either the ovarian vein or the inferior caval vein was significantly decreased by the administration of PMSG and hCG. When both ovaries of rats were extirpated, PMSG and hCG did not decrease the platelet count. The subcutaneous administration of Y24180(0.5-5 mg/kg) decreased the number of ova shed and blocked the thrombocytopenia induced by PMSG and hCG. Such an inhibitory activity of Y24180 on both ovulation and thrombocytopenia was reversed by the supplementation of PAF(2-20 ug/kg). Indomethacin (IDM:0.5-2 mg/kg/12 hours) also reduced the number of ova shed but did not block the thrombocytopenia. Moreover, the inhibition of ovulation by IDM was not reversed by PAF. These results indicate that PAF is involved not only in the platelet activation but also in the follicular rupture by a mechanism different from that of prostaglandins.

263 Detection of gonadotropin-releasing hormone in the rat ovary: studies using the reverse transcription-polymerase chain reaction. M.Oikawa,N.Hoshi,T.Fjino,T.Tanaka,S.Fujimoto,Dept.Obst.and Gynec. Hokkaido Univ. sch. Med.,Hokkaido.

Gonadotropin-releasing hormone (GnRH) exerts direct effects on ovarian cells through specific receptors.Because treatment with GnRH antagonists alter ovarian functions in hypophysectomized rats,the presence of endogenous ovarian GnRH-like peptide(s) has been postulated. In an attempt to detect the ovarian expression of GnRH or related genes at the RNA level,we used the reverse transcription-polymerase chain reaction (RT-PCR) to amplify GnRH message levels.Total RNA from rat ovaries was converted to cDNA using reverse transcriptase and amplified in PCR using a pair of specific primers complementary to the rat GnRH cDNA.The DNA products were subcloned into plasmid vectors and their sequence determined.PCR amplification of cDNA from hypothalamus,granulosa cells and whole ovary yielded a product indentical with the authentic GnRH sequence. Our data demonstrated the presence of mRNA for GnRH in the ovary. Detection of GnRH message in the ovary suggests intragonadal roles of this decapeptide.

264 Inhibitory effect of catecholestrogens on ^{60}Co γ-ray radiation injury in mice. A.Tomatsu, Y.Nakagawa*, M.Suzuki, M.Noguchi, M.Nakanishi, S.Komura**, K.Yagi**, Dept. Obst. and Gynec., Aichi Medi. Univ., Aichi, *Sakashita Hosp., Gifu, **Inst. of Appl. Biochem., Gifu.

We previously reported that lipid peroxide levels in female mice increased after ovariectomy and that the increase in the levels was significantly suppressed by the administration of catecholestrogen 2-hydroxyestradiol ($2\text{-}OHE_2$), a metabolite of estrogen. Since it is known that lipid peroxides are generated by radiation and cause deleterious effects on organs and tissues, it is expected that $2\text{-}OHE_2$ would have a protective effect against radiation injury. The present study was undertaken to examine this problem.

When 10-week-old male BALB/c mice received whole-body irradiation with a single dose of 8 Gy ^{60}Co γ-rays and $2\text{-}OHE_2$ was subcutaneously injected 3 hours before and after the irradiation, 30-day survival rate of the mice was 70%, while the survival rate of the mice administered the other test samples were following; 2-hydroxyestrone, 20%; 2-hydroxyestriol, 20%; 4-hydroxyestradiol, 0%; 2-methoxyestrone, 0%; 2-methoxyestradiol, 0%; 2-methoxyestriol, 0%. The radiation-induced anemia and atrophy of thymus were significantly protected by the administration of $2\text{-}OHE_2$. These results indicate that $2\text{-}OHE_2$ has a potent inhibitory effect on radiation injury.