

199 Increased epidermal growth factor (EGF) bindings to intra-uterine growth retardation rat liver.

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We have been reported that EGF stimulate DNA, glycogen and polyamine synthesis in primary cultured rat liver and may play important roles in fetal growth. We also revealed that the levels of EGF in first voided urine were higher in appropriate for date (AFD) babies than those in light for date (LFD) and heavy for date (HFD) babies. This time, we measured EGF receptor to reveal the control mechanism of EGF level. SFD rat fetuses, 70% weight of AFD, were made by fasting and HFD rat fetuses, 130% weight of AFD, were selected from spontaneous born group. EGF bindings to AFD rat liver membrane markedly increased from day 19 to day 21, but showed no remarkable changes in placenta. At day 21, EGF binding to fetal rat liver were as followed; LFD 380 ± 57 fmol/mg protein, AFD 258 ± 46 , HFD 545 ± 112 , respectively, and showed significantly high EGF binding in LFD and HFD compared to AFD ($p < 0.05$). This data suggest that LFD rat fetus compensate for lack of EGF, on the other hand, HFD rat fetus consume more EGF and resulted in increase of EGF bindings.

200 Fetal growth curve from birth weight and factors related to growth retardation of twin pregnancy. S.Imai, M.Nakayama*, M.Kamiya, I.Shimizu, N.Suehara, T.Takemura, Dept.Obst., *Dept.Pathology, Osaka Med. Center Institute for Maternal and Child Health, Osaka.

Fetal growth curve from birth weight of twins at each gestational age were made in our 259 sets excluding abnormal cases such as fetal major anomaly, intra-uterine fetal death and twin transfusion syndrome. Birth weight of twins was significantly lighter than singleton after 30 weeks gestation. Growth retardation was defined as birth weight was lighter than -1.5 standard deviation. To determine the relationship of factors of growth retardation, we evaluated 309 sets of twins retrospectively. Maternal age, gravidity, parity, induction of ovulation and toxemia of pregnancy had no relationship to fetal growth retardation of twins. In twins both fetuses were well developed, maternal hemoglobin concentration was lower than twins, both or any fetuses were retarded. The incidence of abnormal amniotic fluid volume, monochorionic placenta and abnormal umbilical cord insertion of retarded twin were higher than normally developed twin group. Congenital anomalies were highly associated in the twin, one of which was retarded. Incidence was about 26%.

201 Studies on a physiological role of epidermal growth factor(EGF) in the fetal growth of mice. Y.Kamei, O.Tsutsumi, K.Komatsuzaki, Y.Kuwabara and M.Mizuno, Dept. Obstet. Gynecol., The University of Tokyo, Tokyo.

To study a physiological role of EGF in the fetal growth in mice, we examined the effect of EGF deficiency on the life and growth of fetus employing sialoadenectomy(Sx) on pregnant mice. On day 13 of pregnancy, sham operation or Sx was performed, and 0.9% saline(0.1ml/day) or anti-mEGF antibody(0.1ml/day) was injected sc daily from day 13 to day 18. On day 19 mice were sacrificed and uterus was removed. Number of fetuses(live or dead) was determined and the weight of fetal body, brain, liver and intestine and of placenta was measured. There was no difference in the litter size, however the percent of live pups was reduced from 95 to 80% by Sx and to 71% by Sx + anti-mEGF treatment(Ab). Whereas placental weight was not changed by Sx or Ab, fetal weight decreased from 1.13 ± 0.07 (mean \pm SD) to 1.00 ± 0.14 g in Sx and to 0.93 ± 0.15 g in Ab. Comparison of the weight of fetal organs per body weight revealed that the percent weight of fetal brain was higher in Sx($8.00 \pm 0.38\%$) and Ab($7.90 \pm 0.32\%$) than control($7.20 \pm 0.33\%$), but the percent weight of liver and intestine did not changed. These results suggest that EGF may be necessary for the fetal growth and that EGF deficiency in maternal mice causes asymmetrical intrauterine growth retardation.