Influence on the rat fetal liver maturation with antenatal glucocorticoid administration.

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Purpose : The fetal liver is immature, and physiological jaundice often occurs. The symptom of jaundice will become more severe in premature infants. To investigate the maturation and the function in the liver of premature infants is necessary. Antenatal glucocorticoid (GC) administration is the standard of care for women at risk of a preterm birth. The purpose of this study was to examine whether GC administration acts on maturation factors in the fetal liver for development.

Methods : Dexamethasone were administered to pregnant rats for 2 days and the livers of 19-day-old fetuses, 21-day-old fetuses and 1-day-old neonates were analyzed. We evaluated mRNA levels of HNF4α, Ki-67 and Cyclin B as liver maturation factors and UGT1A1 as bilirubin metabolism-related factor by real-time PCR. In histochemistry, cell size of a hepatocyte was confirmed H-E staining.

Results and Discussion : The mRNA levels of HNF4α and UGT1A1 increased with growth. The mRNA expressions of HNF4a and UGT1A1 were increased in fetal liver with antenatal dexamethasone administration. Cell size of a hepatocyte was enlarged with growth, which is accelerated with antenatal GC administration. These results suggest that antenatal GC administration may accelerated maturation, and may increases bilirubin metabolism in the liver of premature infants.