Prenatal glucocorticoid administration accelerates kidney development in the fetal rats.

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The hypothesis of the "development origins of health and diseases" addresses the risk of chronic kidney disease in adulthood. Although prenatal glucocorticoid (GC) therapy has shown to prevent infant respiratory injury in the neonate, the effects of the kidney functions remains unknown. This study aimed to investigate whether prenatal GC administration is associated with fetal kidney maturation.

Dexamethasone (DEX) was administered to pregnant rats for 2 days on days 17 and 18 or days 19 and 20 of gestation, and the kidney tissues of 19- and 21-day fetuses and 1-day-old neonates were analyzed. The expression of kidney development-related markers (alpha-SMA, aquaporin1and 2) were evaluated by immunohistochemistry and glomeruli and tubules numbers were calculated by H-E staining in the fetal and neonatal rats with prenatal GC.

The expression of kidney development-related markers (alpha-SMA, aquaporin1and 2) were evaluated by immunohistochemistry and glomeruli and tubules numbers in the kidney tissue were measured by H-E staining in the fetal and neonatal rats with prenatal GC. In non-treated group, a kidney size in the neonate was significantly increased compared with that of the fetus. DEX did not changed a kidney size in the fetal and the neonate. The glomerular number was not changed by DEX, but tubular number was significantly increased in 19-day and 21-day fetal kidney tissues.

The expression of alpha–SMA which decrease with growth of mesangial cells was significantly decreased in the fetuses with DEX. Furthermore, DEX increased the expression of aquaporin 1 and 2 in tubuli of the fetal kidney.

In conclusion, these results indicate that prenatal GC administration may contribute to the development of glomeruli and tubules in the immature fetal rat.