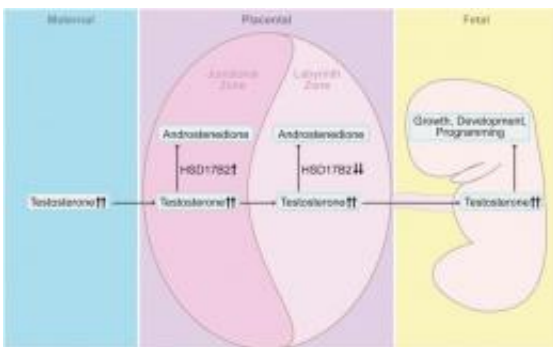


How a low-protein diet predisposes offspring to adulthood hypertension

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Testosterone in maternal plasma, demonstrating elevated levels in response to gestational protein restriction, is transported into placental zones, where part of the testosterone is inactivated by HSD17B2. The rest of the testosterone escapes the inactivation due to the reduced expression of *Hsd17b2*. Thus, excess testosterone goes into fetal circulation, affecting fetal growth and development and presumably predisposing the development of hypertension in offspring. Credit: Image concept: C. Yallampalli. Drawing: Alan Sheffield.

Studies have shown that the offspring of mothers on a low-protein diet are more likely to develop hypertension as adults. Now, Drs. Gao, Yallampalli, and Yallampalli of the University of Texas Medical Branch at Galveston report that in rats, the high maternal testosterone levels associated with a low-protein diet are caused by reduced activity of an enzyme that inactivates testosterone, allowing more testosterone to reach the fetus and increase the offspring's susceptibility to adulthood hypertension.

Fetal programming is a term used to describe the impact of maternal stress on an unborn child's physical characteristics at birth, as well as its long-term health. The placenta is thought to be a major contributor to fetal programming due to its critical roles in hormone production and nutrient transport, as well as its susceptibility to environmental disruptions.

Recently, a study found that protein restriction doubles the plasma testosterone levels in pregnant rats. Elevated testosterone levels are associated with pregnancy-related complications such as preeclampsia and polycystic ovarian syndrome in humans, and emerging evidence suggests that testosterone may play a role in fetal programming of hypertension.

Gao et al. hypothesized that the increased testosterone levels were caused either by increased activity of an enzyme that produces testosterone or by decreased activity of an enzyme that reduces testosterone, specifically *Hsd17b2*, which converts testosterone to a less potent androgen, androstenedione.

The team found that *Hsd17b2* expression in rats was affected by protein restriction in two parts of the placenta. It was increased in the junctional zone, which is responsible for hormone production, but was reduced in the labyrinth zone, which is essential for nutrient transport from mother to fetus and also acts as a protective barrier.

Based on this novel finding, Gao et al. propose that the reduction in *Hsd17b2* expression in the protective labyrinth zone may allow more testosterone to reach the fetus and play a role in fetal programming of hypertension.

The finding that *Hsd17b2* was the only enzyme for testosterone production affected by gestational protein restriction suggests an

important role for *Hsd17b2* in regulating the testosterone levels at the maternal-fetal interface; further research is needed to determine its exact functions.

More information: Gao H, Yallampalli U, Yallampalli C. Gestational protein restriction reduces expression of Hsd17b2 in rat placental labyrinth. *Biol Reprod* 2012; (in press). Published online ahead of print 18 July 2012; [DOI 10.1095/biolreprod.112.100479](https://doi.org/10.1095/biolreprod.112.100479)

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