CHAPTER 11

Cited references


Benediktsson R, Lindsay RS, Noble J, Seckl JR, Edwards CR (1993). Glucocorticoid exposure in utero: new model for adult hypertension. Lancet. 341: 339–341. [This was the first paper to propose the importance of 11β-HSD2 in protecting the developing fetus from adverse effects of glucocorticoids and the consequences on adult susceptibility to disease (glucocorticoid programming).]


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Holmes MC, Abrahamsen CT, French KL, Paterson JM, Mullins JJ, Seckl JR (2006a). The mother or the fetus? 11beta-hydroxysteroid dehydrogenase type 2 null mice provide evidence for direct fetal programming of behavior by endogenous glucocorticoids. Journal of Neuroscience. 26: 3840–3844. [Up until this paper, all models of glucocorticoid programming involved manipulations of the mother, making it impossible to tease apart causal effects of maternal health and behaviour from that of direct actions on the developing fetus.]


Welberg LA, Seckl JR, Holmes MC (2000). Inhibition of 11beta-hydroxysteroid dehydrogenase, the foeto-placental barrier to maternal glucocorticoids, permanently programs amygdala GR mRNA expression and anxiety-like behaviour in the offspring. *European Journal of Neuroscience*. 12: 1047–1054. [This was the first paper to demonstrate that inhibition of 11β-HSD2 alone can cause programming of adult mood behaviours.]


Wyrwoll CS and Holmes MC (2012). Prenatal excess glucocorticoid exposure and adult affective disorders: a role for serotonergic and catecholamine pathways. *Neuroendocrinology*. 95: 1287–1293. [This paper highlights the consequence of deletion of 11β-HSD2 in the placenta to not only affect glucocorticoid access to the developing fetus but also cause dramatic consequences on placental structure and function.]