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Reduced activity of the noradrenergic system in the paraventricular nucleus at the end of pregnancy: Implications for stress hyporesponsiveness

Article in Journal of Neuroendocrinology 17(1):40-8 · February 2005  
DOI: 10.1111/j.1365-2826.2005.01272.x · Source: PubMed

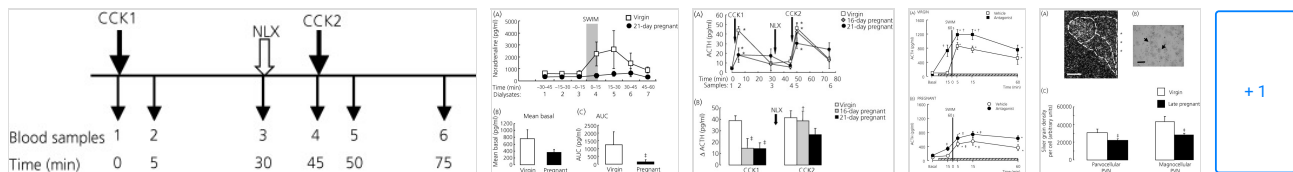
1st **AJ Douglas**  
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Abstract

We investigated whether changes in noradrenaline neurotransmission in the hypothalamus could explain the hyporesponsiveness of the hypothalamic-pituitary-adrenal (HPA) axis in late pregnancy. Noradrenaline release within the hypothalamic paraventricular nucleus in response to swim stress, as estimated by microdialysis and high-performance liquid chromatography, was lower in 20-day pregnant rats compared to virgin rats. Driving a central noradrenergic pathway using intravenous cholecystokinin increased adrenocorticotropic hormone (ACTH) secretion in virgin rats, but the response was significantly less in 16-day and 20-day pregnant rats. Thus, the activity of noradrenergic inputs to the paraventricular nucleus and the HPA axis is attenuated in late pregnancy. The sensitivity of the HPA axis to noradrenaline in pregnancy was investigated by intracerebroventricular administration of an alpha1-receptor antagonist, benoxathian, before and during exposure to swim stress. In virgin rats, benoxathian increased basal and stress-induced ACTH secretion, but in late pregnant rats the benoxathian effects were attenuated, indicating reduced sensitivity of the HPA axis to noradrenaline neurotransmission and/or the inability of the system to become disinhibited at this time. alpha1A-adrenoreceptor mRNA expression in the parvocellular and magnocellular paraventricular nucleus, measured by in situ hybridisation, was decreased in late pregnant compared to virgin rats. Additionally, blocking endogenous opioid inhibition with naloxone pretreatment restored the ACTH secretory response to cholecystokinin in pregnant rats. Thus, in late pregnancy, there is reduced noradrenergic input to the paraventricular nucleus and reduced alpha1A-receptor expression in the paraventricular nucleus, both of which may contribute to the reduced responsiveness of the HPA axis in pregnancy.

Figures



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