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RELATIONSHIP BETWEEN INFLAMMATORY PAIN AND CORTICOSTERONE RESPONSE IN PRENATALLY STRESSED RATS IN POSTNATAL ONTOGENY

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Introduction. Prolonged or severe repeated pain is the stress that activates the hypothalamic-pituitary-adrenocortical system (the HPA axis). Available clinical and fundamental data on the impact of inflammatory pain on the HPA reactivity are controversial. The importance of research into this issue is determined by the fact that pain experienced at an early age can change the HPA axis programming and cause behavioral disorders later.

Methods. A comparative analysis of data on the effect of pain response on the plasma corticosterone level during different periods of postnatal development (1-, 7-, 25- and 90-day-old rats) in prenatally unstressed and prenatally stressed rats was performed for the first time using a model of moderate inflammatory pain (2.5% formalin into the plantar pad of a hind paw).

Results. In newborn rats, the corticosterone level was higher in response to an injection of formalin than to that of vehicle, this difference was maintained 24 h after injections. An injection of formalin to 7-day rats evoked a gradual increase in pain response and corticosterone response for 60 min; the higher level of hormone was observed in prenatally stressed compared to prenatally unstressed animals only on the 30th min after injection of formalin; twenty-four hours later, the hormone level remained above the basal level and did not differ between prenatally stressed and prenatally unstressed rats. In 25- and 90-day-old rats, the inflammatory pain response and corticosterone reaction depended on the level of organization of pain response in CNS, sex and prenatal exposure.

Conclusions. Thus, formalin-induced increase in corticosterone level reflects nociceptive input, rather than the stress associated with vehicle injection; relationship between inflammatory pain and corticosterone response changes during postnatal development showing sexual dimorphism.

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