Neural mechanisms involved in the noxious stress-induced inhibition of ovarian estradiol secretion

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Stress is known to change the secretion of ovarian steroid hormones via the hypothalamic-pituitary-ovarian axis. Our previous study showed that sympathetic inhibitory regulation of ovarian estradiol secretion became pronounced when the hypothalamic-pituitary-ovarian axis was inhibited by chronic estradiol treatment in rats. The present study aimed to clarify the neural mechanisms involved in the responses of ovarian estradiol secretion induced by noxious stress stimulation. The rats were anesthetized on the day of estrus, and the ovarian venous blood was collected intermittently. The secretion rate of estradiol from the ovary was calculated from differences in the estradiol concentration between ovarian venous plasma and systemic arterial blood plasma, and from the flow rate of ovarian venous plasma. Hindpaw pinching or electrical stimulation of a tibial nerve (10 V, 10 Hz) decreased the estradiol secretion rate from the ovary. The decrease responses of the ovarian estradiol secretion rate were abolished by bilateral severance of the ovarian sympathetic nerves. The efferent activity of the ovarian sympathetic nerves was increased following hindpaw pinching or the tibial nerve stimulation. These noxious stress-induced responses of increase in ovarian sympathetic nerve activity and decrease in ovarian estradiol secretion rate were observed in decerebrate rats but were abolished in spinal rats. These results indicate that noxious stress decreases the ovarian estradiol secretion, and the response is due to reflex activation of ovarian sympathetic nerves. The main integration center for this ovarian hormonal response is located in the brain stem.

Optogenetic and chemogenetic dissection of the essential role of the parabrachial nucleus in the nociception-emotion link

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The central amygdala (CeA) plays a kernel role in integrating the aversive sensory information and sending appropriate command signals to the behavioral, autonomic and endocrine circuits. The question here is how neurons in the central amygdala collect information as to the tissue damage, injury and inflammation and how this information affects the amygdala network activities and subsequent behaviors. A large majority of neurons in the CeA, particularly those in its capsular part (CeC), receive direct monosynaptic excitatory inputs from the lateral parabrachial nucleus (LPB; Sugimura et al., 2016), to which a large majority of ascending projection neuron in the superficial layer of the dorsal horn project (Todd, 2010). A selective stimulation of the LPB-CeC fibers in mice, in which channelrhodopsin2 (ChR2)-expression vector had been injected into the LPB, led to Pavrovian associative and operant non-associative threat learning. Chemogenetic activation of the CeA alone was sufficient to induce nociceptive behaviors in healthy rats. In isolated slice preparations, selective stimulation of the LPB-CeC fibers resulted in a brief excitation followed by potent long-lasting feed-forward inhibition in the CeA network. To conclude, the nociceptive information carried by the LPB-CeC pathway determines the CeA network activities and then the adaptive behaviors. [Contribution by Y. Takahashi, Y.K. Sugimura, Y. Miyazawa, M. Sugimoto and A. M. Watabe is acknowledged; Supported by Kakenhi, PSRFPU (MEXT) and RPECP (AMED)].

Neurovascular coupling during nociceptive processing in the spinal cord of the rat.

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Background: Functional magnetic resonance imaging (fMRI) is based on neurovascular coupling, which allows inferring neuronal activity from hemodynamic changes. Spinal fMRI has been used to examine pain processes, although spinal neurovascular coupling has never been investigated. Also, fluctuations in mean arterial pressure (MAP) occur during nociceptive stimulation and this may affect neurovascular coupling. In addition, anesthesia may significantly alter neuronal activity and neurovascular coupling.
Aim: The aim of this study was to examine neurovascular coupling in the rat spinal cord during nociceptive stimulation and to determine the influence of MAP fluctuations and anesthesia on neuronal activity and neurovascular coupling.
Methods: Local field potentials (LFP) and spinal cord blood flow (SCBF) were recorded concurrently in the lumbar enlargement, where activity was evoked by electrical stimulation of the left sciatic nerve in the following conditions: isoflurane-anesthetised, intact vs spinalised (n=6); decerebrated, with vs without isoflurane anesthesia (n=6).
Results: In the intact condition, stimulation of graded intensity produced proportional changes in SCBF and LFP that were paralleled by similar MAP changes. However, spinalisation almost abolished MAP changes ($p<0.001$), while SCBF and LFP responses remained similarly coupled before and after spinalisation ($p>0.3$). In decerebrated rats, anesthesia had no effect on the LFP ($p=0.53$) and SCBF ($p=0.57$), which remained similarly coupled with or without anesthesia ($p=0.39$).
Conclusion: This indicates that spinal hemodynamic changes reflect neuronal activity even when large fluctuations in MAP occur and under the influence of isoflurane anesthesia. This supports the use of fMRI to investigate spinal cord functions.