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DYSFUNCTION OF CA^{2+}/ CAM KINASE IIA CASCADES IN BASOLATERAL AMYGDALA OF POSTTRAUMATIC STRESS DISORDER RATS

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Objective: To explore changes of Ca^{2+}-CaM-CaMKIIα in basolateral amygdala of PTSD rats may reveal part of the pathogensis.
Methods: The SPS-method was used to set up the rat PTSD models. A total of 90 male Wistar rats were randomly divided into1d, 4d, 7d, 14d groups of SPS and normal control groups. The intracellular free calcium level in basolateral amygdala was examined by fluorescence spectrophotometer. CaM and CaMKIIα expression in basolateral amygdala were examined by immunohistochemistry, western blotting and reverse transcription-polymerase chain reaction (RT-PCR).
Results: The intracellular free calcium level reached the peak 1 day after SPS stimulation, then gradually decreased to normal level. The expression of CaM 1day after SPS is also the most and then decreased to normal level. In contrast, CaMKIIα expression showed a significant down-regulation 1day after SPS throughout and then gradually increased to normal level. This findings suggest dysfunction of Ca^{2+}-CaM-CaMKIIα in basolateral amygdala of PTSD rats.
Conclusion: Thus, the trauma-induced enhanced anxiety appear to be associated with, and possibly caused by, changes of Ca^{2+}-CaM-CaMKIIα in basolateral amygdala.

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