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SINGLE-PROLONGED STRESS INDUCED ENDOPLASMIC RETICULUM - DEPENDENT APOPTOSIS IN THE HIPPOCAMPUS IN THE RAT MODEL OF POST-TRAUMATIC STRESS DISORDER

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Background: Post-traumatic stress disorder (PTSD) is a stress-related mental disorder caused by traumatic experience. Some studies showed low hippocampal volumes in PTSD patients. Our previous research indicated apoptosis induced such atrophy in the hippocampus. Endoplasmic reticulum stress -induced apoptosis has been implicated in the development of disorder diseases.
Aim: Our study was to reveal whether apoptosis induced by single-prolonged stress (SPS) in the hippocampus involved in Endoplasmic reticulum-related pathway through observation expression of three important apoptosis-related indicators on ER pathway: Glucose-regulated protein (GRP) 78; calcium/calmodulin/CaM kinaseIIα and caspase-12.
Methods: Wistar rats were killed at 1, 4 and 7 days after exposure to SPS. The apoptotic cells in the hippocampus were assessed by TUNEL method and the free intra¬cellular Ca2+ concentration was measured. Immunohistochemistry, western blotting and RT-PCR were used to detection expression of GRP78, Ca2+/Calmodulin/ CaM kinase IIα and caspase-12.
Results: Our results showed that apoptosis exactly occurred in hippocampus of SPS rats. Both GRP78 and Caspase-12 were significantly up-regulated during early PTSD. They reached peak in the 4 days and then returned to normal levels in 7 days after SPS. The free intra¬cellular Ca2+ concentration was significantly higher in 1 day after SPS and decreased in 7 days; However, CaM expression significantly increased, while CaMKIIα expression significantly decreased in the hippocampus 1 day after SPS.
Conclusion: SPS induced the change of GRP78, Ca2+ and caspase-12 in the hippocampus of PTSD rat, indicating that the endoplasmic reticulum pathway was involved in the process of SPS-induced apoptosis.

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