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CHANGE IN EXPRESSION OF CALRETICULIN IN MEDIAL PREFRONTAL CORTEX OF RATSWITH POSTTRAUMTIC STRESS DISORDER

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Posttraumtic stress disorder (PTSD) is an anxiety disorder caused by traumatic experience, exhibiting three major clinical symptoms: re-experience, avoidance and numbing, and hyperarousal (APA,DSM-IV, 1994). It is thought to involve a dysregulation of medial prefrontal cortex (mPFC) activity in response to fear. The mPFC plays an important role in regulating the stress response (RJ. Ursano et al, Textbook of Disaster Psychiatry). Studies have reported that the calcium signal in the brain cell of PTSD rats is disordered (BING XIAO et al, 2009). Calreticulin, helping to deal with misfolded proteins during the endoplasmic reticulum stress (ERS) response as molecular chaperone, is a calcium binding protein.In this study,detection of the expression level of calreticulin in mPFC of rats with PTSD provides experimental evidence reveled part pathogenesis of PTSD and Single prolonged stress (SPS) method as an established animal model for PTSD was used. A total of 75 male Wistar rats were divided randomly into five groups: SPS1d,SPS4d,SPS7d,SPS14d, and the control group. The calreticulin expression in mPFC was examined using immunohistochemistry, western boltting and reverse transcription polymerase chain reaction (RT-PCR). Immunohistochemistry showed calreticulin widely distributed throughout the mPFC, mainly in the cytoplasm, appeared as buffy particle. Protein level of calreticulin of SPS group gradually increased and peaked at SPS 7d. The mRNA expression of calreticulin was upward trend and peaked at SPS4d. ERS induced by SPS stimulation made misfolded protein accumulation increase, which made calreticulin separate from calcium and increase to deal with misfolded protein. Then intracellular free calcium r increased, which exacerbated ERS and induced cell apoptosis. Expression change of calreticulin caused cell apoptosis, which may be closely related to changes of emotion,cognition of PTSD rats.

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