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SINGLE PROLONGED STRESS TRIGGERS ENDOPLASMIC RETICULUM STRESS AND ENDOPLASMIC RETICULUM ASSOCIATED DEATH IN RAT HIPPOCAMPAL NEURONS

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The efficient functioning of the endoplasmic reticulum (ER) is essential for most cellular activities and survival. The ER is highly sensitive to stresses that perturb cellular energy levels, the redox state or Ca^{2+} concentration. ER stress and ER stress-induced apoptosis is implicated in the pathophysiology of several neurodegenerative and cardiovascular diseases. We suspect that Single prolonged stress which is extensively used as an established animal model for PTSD triggers endoplasmic reticulum stress and endoplasmic reticulum associated death in rat hippocampal neurons. Grp94 (Glucose-regulated protein 94), is the ER-resident member of the heat-shock-protein 90 (Hsp90) family, highly expressed when ERS happened. Caspase-12 is a protease until now believed to play a central role in the initiation of ER stress-induced cell death. In present study, Changes of GRP94 (Glucose-regulated protein 94) and caspase-12 were detected by immunohistochemistry, reverse transcription-polymerase chain reaction and western blot. Changes of GRP94 and caspase-12 may contribute to the progress of molecular mechanism of PTSD.

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