Abstract

Adverse events that cause stress during the early stages of life may alter the normal development of the brain and neuroendocrine system and increase the vulnerability of the individual to various disorders. Chronic stress and subsequent releasing of stress mediators can lead to oxidative stress and cell damage. The first aim of this work was to determine selected oxidative stress markers in the cerebral cortex, hippocampus, and cerebellum after the exposure of rats to early life stress. To model the stressful situation, we used maternal separation of the offspring for three hours a day during the first three weeks of life. We choose reduced glutathione, protein carbonyls, lipid peroxides and hydroperoxides as typical markers. These markers were determined in the brains of rats aged 22 days. Any significant changes were found in the levels of the studied markers after maternal separation.

Damage to brain cells may also be reflected in behavior. Studies of numerous neuropsychiatric and neurodegenerative diseases have indicated that oxidative stress is a promising candidate for inducing changes at the cellular level. The second aim of this work was to monitor the behavior of rats by the light/dark box test after maternal separation along with administration of N-acetylcysteine (NAC), a drug with known antioxidant effects. The results obtained at 22nd postnatal day did not allow us to clearly determine the effect of separation and NAC on anxiety-like behaviors of young rats. In 90-day old rats, their previous early separation caused anxiety behavior, which was partially reduced in cases where NAC was injected during infancy.

The data obtained suggest that the stress model used in this study does not induce changes in the observed oxidative stress markers in the adolescent rat brain. However, the negative impact of maternal separation manifested by increased anxiety behavior of adult rats. In our experiments, NAC adminstration apparently reduced anxiety.