PHYSICAL EXERCISE AVOIDS SHORT AND LONG TERM MEMORY DEFICITS AND ATENUATES OXIDATIVE DAMAGE INDUCED BY MATERNAL DEPRIVATION

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Maternal deprivation (MD) is one potent stressor during neonatal period and can causes mnemonic deficits and oxidative stress in hippocampus. Considering that physical exercise (PE) can be a neuroprotective agent, our objective was investigating its effects on memory deficits and hippocampal oxidative stress caused by MD. For this, 10 pregnant Wistar rats were used and later 38 male rats arising from their offspring. Half of the animals were submitted to MD. To MD, the mother was removed from the home cage for 3 hours/day during the first 10 days of pups' life. After this, the animals were divided into 4 groups (8-12/each): (i) Control; (ii) PE; (iii) MD; and (iv) MD+PE. The PE consisted in running on a treadmill during 8 weeks, 50 min/day, starting on 45th day of life. After all, short and long-term aversive memory (STM and LTM) were evaluated by inhibitory avoidance task (IA), and then the rats were euthanized; hippocampus were dissected and homogenized to measure the GSH, ROS and TBARS levels. IA statistical analysis was performed using the Wilcoxon test; biochemical results were compared using ANOVA followed by Bonferroni post-hoc. P≤0.05 were considered significant. This study was approved by Animal Ethics Committee of UNIPAMPA (protocol 001/2014). The deprived rats presented STM and LTM deficit (P=0.86 STM; P=0.32 LTM); PE prevented these deficits (P=0.001 STM, P=0.02 LTM). There were no alterations on GSH (P=0.015) and ROS (P=0.226) in the groups. An increase on lipid peroxidation (TBARS) on MD rats was detected (P=0.038). The MD+PE rats did not show differences in TBARS when compared to control rats (P = 0.6275). These results demonstrate that the PE can avoid short and long term aversive memory deficits induced by MD and probably one of the mechanisms involved in its effect is the decrease of oxidative damage caused by MD.

Key words: Maternal Deprivation, Memory, Oxidative Damage.