ALTERATIONS ON THE LEVELS OF PROTEINS INVOLVED IN BRAIN OXIDATIVE STRESS INDUCED BY NEONATAL HYPERGLYCEMIA IN RATS

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Introduction: Recently, the consequences of diabetes on the central nervous system have received great attention. However, the mechanisms by which hyperglycemia affect the central nervous system remain poorly understood. In addition, recent studies have shown that hyperglycemia induces oxidative damage in the adult rat brain. In this regard, no study has assessed oxidative stress as a possible mechanism that affect the brain normal function in neonatal hyperglycemic rats. Furthermore, our recent studies have shown that neonatal hyperglycemia induces oxidative damage in rat brain. **Objectives:** We assessed protein levels that probably are involved in the role of oxidative stress in the brain of rats with neonatal hyperglycemia. **Material and Methods:** Seven-day-old Wistar rats were subject to a single administration of streptozotocin (100mg/Kg body weight) and rats with glycemia above 200mg/dL are considered hyperglycemic, while controls received saline. Five days after injection of streptozotocin, animals were killed. We assayed catalase, gluthatione peroxidase, superoxide dismutase, Nrf2, total Akt and phosphorylated Akt protein levels by western blot method. T test was used to statistical analyses. **Results:** No significant changes were detected in superoxide dismutase, glutathione peroxidase and total Akt protein levels. On the other hand, neonatal hyperglycemic rats presented increased catalase and decreased Nrf2 and p-Akt protein levels in the brain when compared to control group. **Conclusion:** Neonatal hyperglycemia was able to alter catalase protein levels, an important enzyme involved in the brain redox status. Also, neonatal hyperglycemia promoted alterations in brain signaling pathways. So, these results suggest that oxidative stress could represent a mechanism to explain the harmful effect of neonatal hyperglycemia on the central nervous system. Protocol number CEUA: 25395.

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