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Impact of neonatal hyperglycemia on protein expression involved in survival/death balance in rat brain

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Diabetes is an endocrine disorder of carbohydrate metabolism characterized by hyperglycemia due to deficiency of insulin secretion or action. Recently, the impact of diabetes on the central nervous system (CNS) has been received great attention, but the mechanisms by which hyperglycemia can cause brain damage remain poorly understood. In addition, recent studies have shown that hyperglycemia induces brain damage in adult rat. In this regard, there is a paucity of studies addressing the deleterious effects of hyperglycemia on the rat brain during CNS development. Therefore, in the present study, we investigated if neonatal hyperglycemia was able to alter the expression of proteins associated with survival/death signaling. Seven-day-old Wistar rats were subjected to a single administration of streptozotocin (100 mg/Kg body weight) while controls received saline. Glycemia was verified every day (>200 mg/dL are considered hyperglycemic). Five days after injection of streptozotocin, animals were killed and the brain homogenates were utilized for analyses. The expression of proteins JNK, GSK3 β , Bax, Bcl2, p38, Total AKT and phosphosforylated AKT (p-AKT) was quantified by "western blot". No significant changes were detected in JNK, GSK3 β , Bax and Total AKT protein expression. On the other hand, brain of neonatal hyperglycemic group presented increased p38 and Bcl2 protein expression. Furthermore, brain of neonatal hyperglycemic rats showed decreased p-AKT protein expression as compared to control group. Finally, these results suggest that neonatal hyperglycemia induces death signaling activation, indicating the potential harmful effect of hyperglycemia on the central nervous system.

Biography

Andrea Pereira Rosa received her MSc in Biochemistry from Federal University of Rio Grande do Sul. She is currently a PhD student in Biochemistry. She has published 15 papers in prestigious scientific journals.

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