

EFFECTS OF GUANOSINE ON THE HIPPOCAMPAL VOLUME IN RATS
SUBMITTED TO NEONATAL HYPOXIC- ISCHEMIC INJURY.

Viola, G. G.¹, Oses, J. P.¹, Moretto, M. B.², Xavier, L. L.³, Lavinsky, D.¹, Netto, C. A.¹, Achaval, M.⁴, Souza, D. O.¹

¹Departamento de Bioquímica, ICBS, UFRGS, ² Departamento de Análises Clínicas e Toxicológicas, CCS,UFSM, ³ PUCRS, ⁴Departamento de Ciências Morfológicas, ICBS, UFRGS

Perinatal brain hypoxic-ischemic (HI) injury is relevant to morbidity and mortality in humans, often leading to seizures and mental impairment. The vulnerability of the developing brain to HI damage is different from that seen in adult brain and is thought to be due partly to the release of excitatory amino acids. Recent evidence suggests that glutamate excitotoxicity is the major mechanism for neuronal death after neonatal injury. Guanosine effect could contribute to the maintenance of extracellular glutamate in physiological conditions, and so avoid excitotoxicity. To investigate the effect of guanosine on the HI neonatal injury, specifically on the protection of hippocampal hemispheres volumetric. The volume of injured hemisphere in HI rats ($1,44 \pm 0,53$) decreased in comparison to controls ($2,28 \pm 0,12$). Three times of guanosine administration (0h, 24h, 48h) after HI injury did not prevent the damage ($1,34 \pm 0,26$). Moreover, the animals exposed to HI injury and received guanosine treatment presented a decrease in the hippocampal volume ($1,61 \pm 0,22$) in the contra lateral hemisphere. Guanosine increase the glutamate uptake, this fact preventing glutamatergic excitotoxicity, but it does not influence the variance of the hippocampal volumetric in the HI injury.