## Expression of Nucleoside Triphosphate Diphosphohydrolase CD39 Family Members by Glioma Cell Line U87

<u>Kipper, F.C.<sup>1</sup></u>, Tamajusuku, A.S.K.<sup>2</sup>, Lenz, G.<sup>2</sup>, Battastini, A.M.O.<sup>3</sup>, Robson, S.C.<sup>4</sup>, Wink, M.R.<sup>1,2</sup>

<sup>1</sup>Departamento de Ciências Básicas da Saúde, UFCSPA, Rio Grande do Sul, Brazil, <sup>2</sup>Departamento de Biofísica e <sup>3</sup>Departamento de Bioquímica, UFRGS, Porto Alegre, Brazil, <sup>4</sup>Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical Schol, Boston, MA, USA.

Introduction: Glioblastoma multiforme (GBM) is the most common, malignant brain tumor and has a poor prognosis. It has been shown that ATP can act as a mitogen for glial cells and that glioma cell lines present very low nucleoside triphosphate diphosphohydrolase (NTPDase) activity when compared to primary astrocytes. Aim: Analyze the patterns of expression of NTPDases in malignant glioma cell line U87 and verify the importance of these enzymes in glioma growth. Methods and Results: The expression of NTPDases was investigated by RT-PCR, immunocytochemistry and Western blotting. The expression of NTPDase 6 (CD39L2), NTPDase 3 (CD39L3) and NTPDase 5 (CD39L4) was detected in the glioma cell line U87, while NTPDase 2 (CD39L1) was absent in the transformed cells. In order to understand the importance of these enzymes, U87 gliomas were transduced with lentivirus containing NTPDase2-EGFP. These cells present 16 times more ecto-ATPase activity, confirming the functionality of the transgene. Conclusions: The low ectonucleotide hydrolysis observed in gliomas may have and implication in tumor growth and alterations in the ecto-nucleotidases may represent an important mechanism associated with malignant transformation of glioma cell lines. The importance of NTPDase2 in in vitro growth will be evaluated with the stable cell line over-expressing NTPDase2-EGFP. **Keywords:** Ecto-nucleotidases, Glioblastoma Multiforme, NTPDase 2. Financial support: FAPERGS, CNPq, NIH.