



## FENS Forum 2008

For posters, morning sessions: 9:30-13:30; afternoon sessions 13:30-17:30.  
Authors are expected to be in attendance at their posters at the time indicated.  
For other sessions, time indicates the beginning and end of the sessions.

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**First author** Martin, Mairena (poster)

Poster board B50 - Mon 14/07/2008, 16:00 - Hall 1

Session 115 - GPCR 2

Abstract n° 115.15

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**Authors** Martin M., Castillo C. A., León D., Iglesias I. & Albasanz J.

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**Title** Glutamate differently modulates adenosine receptors natively expressed in neuronal and glial cells.

**Text** Adenosine is a neuromodulatory substance which inhibits glutamate release acting mainly through Adenosine A1 receptors (A1R). Primary cortical neurons culture and C6 glioma cells were exposed to L-glutamate, and transduction pathways mediated by A1R or A2AR were analyzed. A1R and A2AR were increased and decreased respectively in C6 cells. Levels of mRNA expression were decreased (A2AR) or unaltered (A1R) after L-glutamate treatment. Accordingly, CHA-mediated AC inhibition and CGS21680-mediated AC stimulation were higher or lower, respectively, than in control C6 cells. L-glutamate caused excitotoxic damage only in neurons, C6 cells being preserved. Loss of cell viability in neurons was L-glutamate concentration- and time-dependent, being maximal at 100 microM after 24 hours of treatment. In neurons there was an increase in both A1R and A2AR without changes in gene expression levels. Moreover, AC responsiveness to A1R or A2AR agonist was potentiated in these cortical neurons. Results show that adenosine receptors are regulated by L-glutamate exposure, this regulation being dependent on the cell type, and suggesting the relationship between adenosine receptors and cellular death elicited by excitotoxicity.

**Theme** B - Neural excitability, synapses and glia: cellular mechanisms  
G-protein linked receptors - Purine receptors