their key role as regulators of synaptic transmission and of the abnormal glutamate overexcitation implicated in both acute and chronic brain diseases. We have previously showed that activation of astrocytic A2AR reduce astrocytic glutamate uptake under physiological and pathological conditions,\(^1\)\(^{-4}\) and that A2AR are aberrantly up-regulated upon multiple brain insults.\(^4\)\(^{-6}\)

**Methods:** We incorporated EGFP reporter either alone or combined with either a small hairpin to down-regulate A2AR (shA2AR) or a control sequence (shCTR) into Mokola Lyssavirus (Mok-G) and Vescicular Stomatitis Virus (VSV-G) lentivectors and tested whether Mok-G-coated lentivirus selectively and efficiently transduced astrocytes in primary culture or in mouse brain through stereotoxic administration of lentivectors into striatum [STR], hippocampus [HIPP] and prefrontal cortex [PFC] (compared to neurotropic VSV-G-coated lentivirus as controls). Herein, we evaluated viral spreading and cell-type transduction through immunofluorescent colocalization of EGFP with glial (GFAP and vimentin) and neuronal (NeuN) markers.

**Results:** After 25 days post-infection, Mok-G,EGFP transduced 68% of cultured astrocytes (EGFP- and DAPI-positive, \(n = 1\)); 100% of GFAP-positive cells colocalized with EGFP as well as 86% cells expressing Vimentin only and 47% expressing both Vimentin and GFAP. Mok-G shA2AR lentiviruses robustly reduced A2AR immunoreactivity compared to Mok-G shCTR in cultured astrocytes. At 4 weeks post-brain administration, Mok-G,EGFP was expressed mainly in astrocytes (GFAP-positive cells) in both STR and HIPP, and to a lower extent in the PFC, whereas VSV-G-coated lentivirus colocalized with NeuN marker and not with GFAP in any tested brain areas.

**Conclusion:** These data supports the ability of Mok-G lentivectors to efficiently transduce astrocytes to control A2AR density, paving the way for their application to control pathophysiological processes involving astrocytes.


**References**


**PS077**

**Adenosine A1 receptor antagonist prevents DSI in hippocampal CA1 pyramidal cells**

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**Aim:** How adenosine interfere with a short-term form of neuronal plasticity dependent on endocannabinoid, the depolarization-induced suppression of inhibition (DSI).

**Conclusion:** These data supports the ability of Mok-G lentivectors to efficiently transduce astrocytes to control A2AR density, paving the way for their application to control pathophysiological processes involving astrocytes.


**References**


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**PS087**

**High-sucrose diet effects on the dendritic trees of developing neurons of the adolescent rat**

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**Aim:** In the present study, we aimed to explore the effect of high-sucrose diets on the dendritic trees of immature granule cells of the adolescent male rats.

**Introduction:** Adolescence is a period of high susceptibility to exogenous factors as the rat brain is still developing. Evidence shows that high-sucrose diets may be more detrimental to adolescent rats, therefore we intended to study immature granule cells in the hippocampal formation of these animals. For that, we used...