

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,<sup>1–3</sup> and that A2AR are aberrantly up-regulated upon multiple brain insults.<sup>4–6</sup>

**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 Vesicular 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 stereotaxic administration of lentivectors into striatum [STR], hippocampus [HIPPP] 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 HIPPP, 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.

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## PS077

### Adenosine A1 receptor antagonism prevents DSI in hippocampal CA1 pyramidal cells



J. Freire<sup>1,2,\*</sup>, D.M. Rombo<sup>1,2</sup>, A.M. Sebastião<sup>1,2</sup>

<sup>1</sup> Instituto de Farmacologia e Neurociências, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal

<sup>2</sup> Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal  
E-mail address: [joanamorimf@gmail.com](mailto:joanamorimf@gmail.com)

(J. Freire).

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

**Introduction:** The widely consumed psychoactive drug cannabis, containing cannabinoid compounds, and/or caffeine, with adenosinergic antagonizing proprieties, exert their central actions by affecting cognitive operations such as learning and memory. Indeed, endogenous adenosine and endocannabinoids (eCB) are known to interfere with physiological synaptic plasticity phenomena that represent the neuronal substrate of memory formation.

**Methods:** Whole-cell voltage-clamp recordings ( $V_h = -70$  mV) were performed on hippocampal CA1 pyramidal cells of 3 to 5 weeks-old C57BL/6 mice. Slices (350  $\mu$ m thick) were perfused with artificial cerebrospinal fluid (aCSF) supplemented with glutamate receptor antagonists (CNQX, 25  $\mu$ M and DL-APV, 50  $\mu$ M) to block glutamatergic transmission and isolate GABA-mediated responses. Inhibitory postsynaptic currents (IPSCs) were evoked every 3 s through a stimulation electrode placed in stratum radiatum. The recording electrode was filled with a CsCl-based intracellular solution and DSI was evoked through a 5 s voltage step of +80 mV. The magnitude of DSI was measured 9 s after the depolarizing step and DSI recovery was evaluated between 30 and 60 s after depolarization.

**Results:** When recording eCB-mediated DSI we observed a decrease in electrical-evoked IPSC amplitudes to  $81.0 \pm 5.4\%$  of baseline ( $p < 0.01$ ,  $n = 14$ ) that fully recovered to  $90.2 \pm 5.4\%$  after 30–60 s. The adenosine A1 receptor antagonist, DPCPX (100 nM), prevented DSI, recordings showing a non-significant change in IPSCs amplitude to  $95.1 \pm 12.0\%$  of baseline ( $p = 0.3473$ ,  $n = 10$ ) that was maintained throughout the recovery period ( $87.1 \pm 12.0\%$ ).

**Conclusion:** These results suggest that tonic adenosine A1 receptor activation is necessary for the occurrence of DSI. The mechanisms involved in this process remain unclear and need further investigation.<sup>1–4</sup>

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## PS087

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



R. Rodrigues<sup>1,2,\*</sup>, F. Barreto<sup>1,2</sup>, A. Cardoso<sup>1,2</sup>, J.P. Andrade<sup>1,2</sup>

<sup>1</sup> Department of Biomedicine – Unit of Anatomy, Faculty of Medicine, University of Porto, Alameda Prof. Hernâni Monteiro, 4200-319 Porto, Portugal  
<sup>2</sup> Center of Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Rua Dr. Plácido da Costa, 4200-450 Porto, Portugal  
E-mail address:

[patriciarafaelarodrigues@gmail.com](mailto:patriciarafaelarodrigues@gmail.com)

(R. Rodrigues).

**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