α7 NICOTINIC RECEPTOR PRESENT ON MOUSE CORTICAL ASTROCYTES MODULATES ENDOGENEOUS GLUTAMATE RELEASE

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The presence of nicotinic receptors on astrocytes in human and rat brain has been previously demonstrated (1) however their possible functional role is still poorly understood. In this study we investigated on the presence of nicotinic receptors on gliosomes (2), purified from mouse cortex, and on their role in eliciting glutamate release. Epibatidine significantly increased basal release of [^{3}H]D-aspartate and of endogenous glutamate from mouse gliosomes but not from synaptosomes. This effect was prevented by methyllycaconitine, α-bungarotoxin and mecamylamine but not by dihydro-b-erytroidine. Epibatidine provoked also a significant increase of calcium concentration in gliosomes but not in synaptosomes; the $[Ca^{2+}]_I$ enhancement induced by epibatidine and KCl in gliosomes were very similar each other. The present results indicate that α7 nicotinic receptors exist on mouse cortical glial particles and stimulate glutamate release.

(1) Xiu J, Nordberg A., Zhang JT., Guan ZZ., (2005) Neurochem.Int. 47, 281-290
(2) Stigliani S. et al (2006). J Neurochem. Feb, 96(3):656-68.

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