The role of glutamate transporters in neurodegenerative diseases and potential opportunities for intervention [HTML] nih.gov

AL Sheldon, MB Robinson - Neurochemistry international, 2007 - Elsevier

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G Sanacora, CA Zarate, JH Krystal... - Nature reviews Drug ..., 2008 - nature.com

... US Food and Drug Administration-approved drug riluzole (2-amino-6-(trifluoromethoxy) benzothiazole), which is used for the treatment of ALS, has also been shown to inhibit glutamate release. However, riluzole exerts a range of effects on the glutamatergic system, including ...

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K Hashimoto - Brain research reviews, 2009 - Elsevier

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NJ Maragakis, JD Rothstein - Neurobiology of disease, 2004 - Elsevier

... expect. Furthermore, cerebellar anatomy was not grossly altered and electrophysiologic study in the cerebellum did not implicate a major role for GLAST in the synaptic clearance of glutamate from Purkinje cells. However ...

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Glutamate transporters, EAAT1 and EAAT2, are potentially important in the pathophysiology and treatment of schizophrenia and affective disorders

Georgia M Parkin, Madhara Udawela, Andrew Gibbons, Brian Dean

Abstract

Glutamate is the predominant excitatory neurotransmitter in the human brain and it has been shown that prolonged activation of the glutamatergic system leads to nerve damage and cell death. Following release from the pre-synaptic neuron and synaptic transmission, glutamate is either taken up into the pre-synaptic neuron or neighbouring glia by transmembrane glutamate transporters. Excitatory Amino Acid Transporter (EAAT)1 and EAAT2 are Na^+-dependant glutamate transporters expressed predominantly in glia cells of the central nervous system. As the most abundant glutamate transporters, their primary role is to modulate levels of glutamatergic excitability and prevent spill over of glutamate beyond the synapse. This role is facilitated through the binding and transportation of glutamate into astrocytes and microglia. The function of EAAT1 and EAAT2 is heavily regulated at the levels of gene expression, post-transcriptional splicing, glycosylation states and cell-surface trafficking of the protein. Both glutamatergic dysfunction and glial dysfunction have been proposed to be involved in psychiatric disorder. This review will present an overview of the roles that EAAT1 and EAAT2 play in modulating glutamatergic activity in the human brain,

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