## Review Article

# Glutamate transporters and excitotoxicity in nervous system

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## Abstract

L-glutamate is the major excitatory neurotransmitter in the central nervous system (CNS). It contribute in various physiological conditions such as brain development, synaptic plasticity, memory and learning. However, increasing of the extracellular glutamate concentration and overactivation of glutamate receptors in particular ionotropic subtypes leads to excitotoxicity which is the fundamental pathological pathway of neuronal injury. Due to lack of extracellular enzymatic destruction, the removal of released glutamate is achieved through the excitatory amino acid transporters (EAATs) which are distributed in glia that tightly surround the synaptic clefts, as well as in neurons. EAATs which known as Na+-dependent high-affinity glutamate transporters are the main responsible for maintaining extracellular glutamate concentration below excitotoxic levels. Moreover another membrane transporters regulating the flux of glutamate in different areas of the CNS. This system is cystine-glutamate exchanger (XCG-) that is Na+-independent system. Dysfunction of EAATs has been implicated in both acute insults e.g. stroke, trauma and chronic neurological and neuropsychiatric disorders e.g. amyotrophic lateral sclerosis, epilepsy, schizophrenia and Alzheimer's disease. Therefore, the purpose of this review article is to explain the pathway of glutamate biosynthesis, its release into CNS, discribing and elaborating Glutamate transporters, activites and their role in excitoxcity in CNS.

**Keywords:** Glutamate, Glutamate transporters, Nervous system, Excitotoxicity, Glia cells

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