Kainic acid

Kainic acid (KA) is an excitotoxic agent commonly used to induce epilepsy in rodents. The relationship between KA-induced neuronal damage and Golgi complex fragmentation has not been investigated, leaving a major gap in our understanding of the molecular mechanism underlying the development of pathophysiology in epilepsy.

Kaneko et al cultured primary rat cortical neurons either in ambient condition (control) or treated with a range of KA doses to reveal whether Golgi complex fragmentation impaired neuronal function. The half-life maximal inhibitory concentration (IC 50) value of KA was detected to be approximately 5 µM, whereby at these concentrations, KA impaired neuronal viability, which was closely associated with initial Golgi complex fragmentation and subsequent reduction in both the expression and glycosylation patterns of Reelin. These findings implicate that Golgi complex fragmentation and Reelin dysfunction are key contributors to neuronal cell death in the early stage of epilepsy pathophysiology, thereby representing as novel disease biomarkers, as well as potent therapeutic targets for epilepsy [1)].

1)
Kaneko Y, Sullivan R, Dailey T, Vale FL, Tajiri N, Borlongan CV. Kainic Acid-Induced Golgi Complex Fragmentation/Dispersal Shifts the Proteolysis of Reelin in Primary Rat Neuronal Cells: An In Vitro Model of Early Stage Epilepsy. Mol Neurobiol. 2016 Apr;53(3):1874-83. doi: 10.1007/s12035-015-9126-1. Epub 2015 Mar 21. PubMed PMID: 25790952; PubMed Central PMCID: PMC4577368.