

Role of purinergic signal in pathophysiology of epileptic seizures

Prashant Srivastava¹, Saurabh Shukla¹, Sanjay Kumar Choubey¹ and Gomase V.S.²

¹Yeshwant College of Information Technology (Bioinformatics & Biotechnology),
Parbhani, MS

²School of Technology, S.R.T.M. University, Sub-Centre, Latur, 413512, India

Purinergic Signaling contribute a lot in the pathophysiology of epileptic seizures. Adenosine Tri Phosphate (ATP) together with gap junction transmits the glial calcium waves. It is an intraneuronal signaling pathway of central Nervous System (CNS). Intracellular waves are reported to contribute epileptic seizures; the calcium waves are debilitated in response to contribute antiepileptic drugs. Interrupting the ATP – Associated Gliotransmission would be a breakthrough for using antiepileptic drug against epileptic seizures. Enzymatic hydrolysis of accumulated ATP releases one of the purine compound called adenosine. Adenosine releases by astrocytes is supposed to inhibit remote synapse by interacting with adenosine receptor. Failure of the above mechanism would result in epileptic seizures. Purinergic signals assist in spread of epileptic form discharge and may contribute to proper co – ordination of synaptic plasticity. ATP is reported to extra cellularly on pre and post junctional membranes at synapse. Purinergic signaling contributes to lots of physiological activity. Nerve cells releases ATP as co - transmitter and this triggers response in astrocytes and oligodendrocytes which express P2 receptors. Astrocyte responds by strengthening the synapse. There is rise in intracellular Ca⁺⁺ level due to action potential generating while neuromuscular junction respond to axonal ATP. Increased extracellular ATP level in epileptic seizures strengthening the fact that Purinergic Signaling is involved in epilepsies. There is increased in extracellular ATP level due to fall in ATPase activity in the nervous system. Releases of Adenosine by the hydrolysis of ATP bring about activation of A1 receptor, a major cause of epileptic seizures. A1 receptor is actively involved in the development of epileptic seizures, presynaptic modulation and excitability of neurons. Inhibition of Adenosine Kinase activity may cause increased level of extracellular adenosine causing epileptic seizures. ATP analogs may be contributes to epileptic seizures. Other than adenosine receptor P2x7 receptors have also been found to induce epileptic seizures.