

Understanding the brain's natural foil for over-excited neurons

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Glutamate is to the brain like coffee is to our bodies. A cup of Joe in the morning can wake us, but overloading on caffeine causes the stimulant to work against us.

Glutamate is the major excitatory neurotransmitter in a mammal's central nervous system. It is an important component for neuroplasticity, the synaptic communication between neurons. It's also key to learning and memory. But in high concentrations, glutamate becomes toxic-- over-exciting the <u>neurons</u>. Glutamate-induced excitotoxicity is known to exacerbate damage caused by brain injury, stroke and other neurodegenerative diseases.

In order to understand possible ways to reduce the damage of excessive glutamate, researchers at Georgetown University Medical Center have shown how, when high concentrations of glutamate activate the metabotropic glutamate receptor 1 (mGlu1 receptors), they become protective. This concentration of glutamate is normally toxic.

The study, presented at the 39th annual meeting of the Society for Neuroscience, suggests that this glutamate-induced protection occurs due to the association of mGlu1 receptors with the intracellular protein β -arrestin, which causes a sustained phosphorylation of mitogen-activated protein kinases, and protects cells from apoptotic death.

"Studies about the signal transduction involved in mGlu1-mediated neuroprotection may enhance our understanding of the role that this



glutamate receptor plays in <u>brain injury</u>," explains Andrew Emery, a PhD candidate in the Interdisciplinary Program in Neuroscience at GUMC. "Such studies may contribute to rational drug design for potential therapeutic approaches to protect against excitotoxic brain damage following injury, stroke and <u>neurodegenerative diseases</u>.

Source: Georgetown University Medical Center (<u>news</u>: <u>web</u>)

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