

Neuroscience Colloquium 2007/08

Location: Virchow-Lecture-Room, Anatomy, CCM **Date:** Tuesday, 6:15 p.m.

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SUMOylation regulates kainate receptor endocytosis and synaptic transmission.

Kainate receptors (KARs) play crucial roles in the regulation of both excitatory and inhibitory neurotransmission. Their dysfunction has been proposed to underlie or contribute to many conditions that have a major impact on the quality and length of human life associated with profound economic and social consequences for society as a whole. Protein SUMOylation has important roles in the regulation of nuclear function. Far less well investigated are the targets and roles of SUMO modification outside the nucleus. We have shown recently that SUMOylation of the GluR6a KAR subunit following agonist activation evokes kainate receptor endocytosis and modulation of synaptic transmission. The specific implications of our observations are that regulation of kainate receptors by SUMOylation will be involved in synaptic plasticity and neuronal excitability. In addition, SUMO has been associated with a variety of neuropathological conditions and I will present data showing that neuronal protein SUMOylation is dramatically upregulated following ischemia. Finally, I will discuss strategies we are currently employing to identify which proteins are modified by SUMO. In a wider context, our findings raise the prospect that protein SUMOylation, similar to other posttranslational modifications such as phosphorylation and ubiquitination, may be of fundamental importance in controlling the interactions and functions of synaptic target proteins.

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