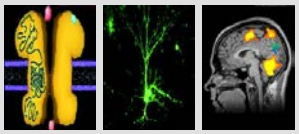
CONFERENCE



Molecular and Neural Correlates of Memory and Cognition

April 9 - 10, 2019 Veranstaltungszentrum, Ruhr University Bochum

Tuesday April 9, 9:25 – 12:35

Session 1 **Molecular substrates of memory and cognition**

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Control of presynaptic function and plasticity by strategic phosphorylation

A hallmark of synapses, the key site of information exchange and storage in the brain, is their ability to regulate and adapt their strength, a process termed synaptic plasticity. Synaptic plasticity can occur on both sites of the synapse. Whereas it is well established that phosphorylation in the postsynaptic compartment plays an important role, less is known about the mechanisms of plasticity of release on the presynaptic site. One protein that has been identified to be essential for basal synaptic transmission and presynaptic plasticity is the presynaptic scaffolding protein is RIM1α, the most abundant isoform of the RIM protein family. While RIM1α is a known phosphoprotein, it is still unresolved how phosphorylation affects RIMα function and what may be the consequences for transmitter release and plasticity. We have identified the SR protein kinase 2 (SRPK2) kinase to trigger phosporylation of several presynaptic active zone proteins and as a novel RIM1αand ELKS interacting protein. We now identified a single key phosphosites in RIM1a regulated by SRPK2 and show that elevated kinase activity selectively increases presynaptic RIM1 levels. Furthermore, SRPK2 induced the formation of additional RIM1 nano-clusters per synapses and potentiated action potential evoked transmitter release. Silencing-induced homeostatic up-scaling of transmitter release was occluded by elevated SRPK2 activity without saturing the vesicle release. Taken together, our data identifies SRPK2 as a novel presynaptic plasticity-relevant kinase that opens up more presynapitc vesicle releas sites by enhancing local RIM1a availability in the active zone via control of a single key phopsho-site.