The Department of Pharmacology

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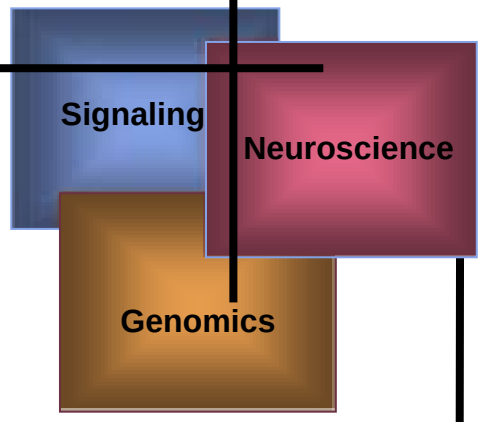

Frontiers in Pharmacology

Regulation of sodium-activated potassium channels and the Fragile X Mental Retardation Protein signalling pathway

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The *Slack* and *Slick* genes encode Na^+-activated K^+ channels (K_{Na} channels), which regulate the rate at which neurons adapt to maintained synaptic stimulation and the accuracy of timing of neuronal action potentials. We have found that the C-terminal domain of Slack interacts with Fragile-X Mental Retardation protein (FMRP), an RNA-binding protein that regulates trafficking and translation of a subset of subset of neuronal mRNAs. In humans, absence of FMRP results in Fragile X syndrome (FXS), the most common inherited form of intellectual disability. The direct interaction of Slack with FMRP produces a potent stimulation of channel activity, and, in the absence of FMRP, the characteristics of neuronal K_{Na} channels are altered. We have used neurons in the auditory brainstem to investigate the biological consequences of Slack-FRMP interactions. Our findings raise the hypothesis that Slack activity may link changes in neuronal firing to changes in protein translation, and suggest that some abnormalities in FXS result from altered Slack channel function.

Friday, February 26th
10:00 am
GBSF Auditorium
(Rm. # 1005)