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Virally mediated functional suppression of GABA-A receptors in the rodent thalamus

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GABAergic inhibition is essential for the normal function of thalamocortical networks. In addition beside the phasic inhibition mediated by synaptic GABA-A receptors, extrasynaptic GABA-A receptors also exert powerful tonic inhibition on thalamocortical cells. The nucleus specific role of synaptic inhibition is difficult to assess using the traditional pharmacological or KO methods due to the limited spatial and temporal control of their effects. Thus, in this study, we used stereotaxic injection of viral particles to focally knock down synaptic GABA-A receptors. All synaptic GABA-A receptors in the thalamus contain g2 subunit. Injection of AAV-Cre viral particles into the thalamus of g2 floxed mice results in the focal removal of the g2 gene. Successful removal of the g2 gene was identified by immunocytochemistry for g2 protein. Immunoreactivity of the other subunits of synaptic GABA-A receptors (a1, b2) as well as extrasynaptic receptors (alpha4) remained unchanged. Preliminary juxtacellular recording in anaesthetized mice demonstrated that it is possible to record, label and identify virally infected thalamocortical cells, which lack functional synaptic GABA-A receptors and to differentiate them from non-infected cells with intact inhibition. Our data demonstrate that the present method is an excellent tool to study the nucleus specific role of synaptic GABA-A receptor in cellular and network activity.