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Presentation Abstract

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Presentation Title: Early changes in synaptic inputs to dentate molecular layer neurons following concussive brain injury.

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Abstract: Closed head injury results in an early increase in excitability of the dentate gyrus one week after trauma. Alterations in both excitatory and inhibitory networks contribute to early post-injury dentate hyperexcitability. Since loss of hilar neurons is a typical feature of brain injury, studies have focused on granule cells and hilar neurons to determine if post-traumatic changes in intrinsic and synaptic physiology underlie the changes network excitability. Recently, we reported that semilunar granule cells (SGCs), a class of excitatory neurons in the molecular layer, demonstrate post-traumatic increase in intrinsic excitability not observed in other dentate glutamatergic neurons. We showed that granule cells and SGCs show marked differences in the effect of injury on synaptic and tonic inhibition. Since SGCs and molecular layer interneurons (MLI) contribute to feed-back and feed-forward inhibition in the dentate, we examined SGCs show post-traumatic changes in glutamatergic synaptic inputs and investigated whether early post-injury decreases synaptic inhibitory inputs is unique to SGCs or is common to other neurons in the molecular layer.
Whole-cell patch clamp recordings were obtained from SGCs and dentate MLI in hippocampal slices from juvenile male rats one week after lateral fluid percussion injury (FPI) and sham-injured controls (Gupta et al., 2012). Morphological reconstruction of biocytin-labeled neurons and intrinsic physiological characteristics were used to distinguish MLI from SGCs. Interneurons with somata in the molecular layer were classified as MLI and included morphologically identified MOPP, axo-axonic, and neurogliaform cells. We found that the frequency of excitatory

postsynaptic currents (sEPSCs) in SGCs was enhanced one week after FPI. Recordings in slices from control rats revealed that, compared to SGCs and granule cells, the frequency spontaneous inhibitory postsynaptic currents (sIPSCs) in MLI was significantly lower. There was a post-traumatic increase in sIPSC frequency in MLI, which parallels the early increase in granule cell sIPSC frequency, but contrasts with the decrease in sIPSC frequency in SGCs one week after FPI. Curiously, sIPSC frequency in MLI and SGCs was not different one week after FPI.
The early post-traumatic increase in SGC synaptic excitatory inputs observed here, could actively recruit hyperexcitable SGCs during network activity and contribute to post-traumatic dentate hyperexcitability. The distinctive early decrease in SGC synaptic inhibition after FPI suggests that interneuronal populations that innervate SGCs may be different from those synapsing with granule cells and MLI.

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DENTATE GYRUS

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