RUHR-UNIVERSITÄT BOCHUM

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Young Scientist IGSN-Award 2010
for an Outstanding Publication of an Undergraduate

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Enhanced synaptic connectivity and epilepsy in C1q knockout mice

Excessive CNS synapses are eliminated during development to establish mature patterns of neuronal connectivity. A complement cascade protein, C1q, is involved in this process. Mice deficient in C1q fail to refine retinogeniculate connections resulting in excessive retinal innervation of lateral geniculate neurons. We hypothesized that C1q knockout (KO) mice would exhibit defects in neocortical synapse elimination resulting in enhanced excitatory synaptic connectivity and epileptiform activity. We recorded spontaneous and evoked field potential activity in neocortical slices and obtained video-EEG recordings from implanted C1q KO and wild-type (WT) mice. We also used laser scanning photostimulation of caged glutamate and whole cell recordings to map excitatory and inhibitory synaptic connectivity. Spontaneous and evoked epileptiform field potentials occurred at multiple sites in neocortical slices from C1q KO, but not WT mice. Laser mapping experiments in C1q KO slices showed that the proportion of glutamate uncaging sites from which excitatory postsynaptic currents (EPSCs) could be evoked ("hotspot ratio") increased significantly in layer IV and layer V, although EPSC amplitudes were unaltered. Density of axonal boutons was significantly increased in layer V pyramidal neurons of C1q KO mice. Implanted KO mice had frequent behavioral seizures consisting of behavioral arrest associated with bihemispheric spikes and slow wave activity lasting from 5 to 30 s. Results indicate that epileptogenesis in C1q KO mice is related to a genetically determined failure to prune excessive excitatory synapses during development.

Host:

Denise Manahan-Vaughan
Director / Dean International Graduate School of Neuroscience -
Department of Neurophysiology, Ruhr-University Bochum

Guests are welcome !

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