# LOSS OF γ-AMINOBUTYRIC ACID (GABA)-IMMUNOREACTIVITY IN A KINDLING INDUCED FOCUS OF EPILEPTIC ACTIVITY.

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Rats were implanted with electrodes to allow daily tetanization (kindling) of Schaffer-collateral/commissural fibers of the dorsal hippocampus and to record the evoked afterdischarges. After reaching a total number of at least 10 generalized (class V) convulsions, the stimulation was terminated. The rats were fixed 24 hours after the last convulsion for immunocytochemical localization of GABA in the $CA_1$-region of the hippocampus.
GABA immunoreactivity was located within perikarya of some cells in the pyramidal cell layer. Also immunoreactivity was found in the terminals ending on the perikarya of nonstaining pyramidal neurons, corresponding with the suggested site of GABA mediated inhibition. In control animals the pattern of immunoreactivity nearby the electrode tracks was only slightly diminished. On the contrary kindled animals showed a clear reduction in reactivity of the somata as well as a reduction of terminal reactivity at the perikarya of pyramidal cells.

This loss of GABA immunoreactivity indicates that kindling is accompanied by a reduction of GABA mediated inhibition. Such a reduction could play an important role in the observed decreased stability of the neuronal network in the kindling model of epilepsy.