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2 DIFFERENT ABNORMAL GABAERGIC CIRCUITS IN EXPERIMENTAL-MODELS OF EPILEPSY - LOSS OF INHIBITION AND DISINHIBITION

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Two Different Abnormal GABAergic Circuits in Experimental Models of Epilepsy: Loss of Inhibition and Disinhibition. Charles E. Ribak (Department of Anatomy, University of California, Irvine, CA, U.S.A.).

Previous immunocytochemical studies of epileptic foci have indicated that the GABAergic axon terminals of both cortical basket and chandelier cells are dramatically reduced by over 80% (see Ribak, *Brain Res* 326:251-60, 1985). To determine if this loss was associated with a loss of GABAergic somata, an anti-GAD serum was used that detects somal GAD without colchicine. The number of GAD-positive neurons was shown to be reduced at chronic epileptic foci. These results add further support for the GABA hypothesis of epilepsy that suggests a loss of GABA-mediated inhibition may cause focal epilepsy. To test this hypothesis in genetic models of epilepsy, similar methods were utilized to study the GABAergic neurons in the seizure-sensitive gerbil and genetically epilepsy-prone rat. The gerbils displayed an increase in the number of GABAergic neurons and terminals in the dentate gyrus of the hippocampus, whereas the audiogenic rats had a similar increase in the central nucleus of the inferior colliculus. Electron microscopic results indicate that an increased number of GABA-ergic axosomatic contacts are formed with these GABAergic neurons. If the GABAergic neurons are inhibited more in these genetically epileptic models, it would cause a release of tonic inhibition of projection neurons and thereby disinhibit them to cause seizure activity. Thus, epilepsy may result from two different abnormalities in GABAergic circuits, loss of inhibition or disinhibition.