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A Decrease in the Number of GABAergic Somata Occurs in Pre-seizing Monkeys With Alumina Gel Granuloma: A Causal Role of GABA in Focal Epilepsy. Charles E. Ribak, Camil Joubran, and Roy A. E. Bakay (University of California, Irvine, CA, and Emory University, Atlanta, GA, U.S.A.).

Previous studies have shown that a loss of GABAergic neu-ronal somata is associated with a loss of GABAergic terminals at experimental cortical epileptic foci in monkeys. The present study was undertaken to determine whether GABAergic neu-ronal loss precedes the onset of clinical seizures in a way similar to that shown recently for GABAergic terminals. Adolescent monkeys received alumina gel implants into the left pre- and postcentral gyri. Three pre-seizing monkeys were killed 2½ months after the alumina gel implant but prior to clinically active seizures. Three other monkeys that were chronically seizing were killed 3–6 months after the implant. Sections were pro-cessed for immunocytochemistry and examined with a light mi-croscope. The pre-seizing monkeys showed a significant loss of GABAergic somata (23–44%) at the focus. The loss of these cells was greater in the chronic animals that showed losses at both the focus and parafocus, 42–61% and 15–26%, respec-tively. Sham-operated animals showed no seizure activity and no significant losses of GABAergic cells. Therefore, a loss of GABAergic somata at epileptic foci occurs in pre-seizing monkeys, and these results provide additional support for the hypothesis that GABA loss plays a causal role in focal epilepsy.