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Authors
Dube, C
Brunson, K
Eghbal-Ahmadi, M
et al.

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FEBRILE SEIZURES LEAD TO INCREASED SUSCEPTIBIL-ITY TO LIMBIC SEIZURES DURING ADULTHOOD.

Celine Dube, UCI & INSERM 398, IRVINE Strasbou, CA; kristen brunson, Mariam Eghbal-Ahmadi, Wayna Cariaga, Tallie Z Baram, UCI, Irvine, CA

RATIONALE: Febrile seizures are the most prevalent seizure type in infants and young children. Whether febrile seizures cause long-lasting alterations in limbic excitability is a critical question pertaining to the later development of temporal lobe epilepsy. Recent neuroanatomic and electrophysiologic data indicate that, in the immature rat model, hyperthermic seizures on postnatal day 10 (P10) lead to significant alterations of physicochemical properties (Toth, J Neurosci 1998) and excitability (Chen, Nature Medicine, 1999) of hippocampal neurons. OBJECTIVE: To test the hypothesis that hyperthermia-induced seizures result in persistent vulnerability to generation of limbic seizures. METHODS: Hyperthermic seizures were induced on P10 (Baram, Brain Res 1997). Mean seizure duration was 21.5±0.9 min and threshold temp. averaged 40.9±0.3 C. As adults (P78-80) rats experiencing febrile seizures and litter-mate controls were implanted with hippocampal bipolar electrodes and monitored for spontaneous behavioral and electrographic seizures over 7–10 days. To determine susceptibility to limbic seizures, a sub-threshold dose of the prototypical limbic convulsant, kainate (5 mg/kg) was given i.p., and EEG and behavioral seizures were evaluated. RESULTS: Neither experimental nor control rats (n = 11/group) developed spontaneous seizures, and EEGs were normal. Following kainate administration, 2/ 11 controls had 2 short (<60 sec) seizures each. In contrast, all animals with early-life febrile seizures developed hippocampal seizures, and most (8/11, 73%) progressed to status epilepticus. CONCLUSIONS: These data indicate that febrile seizures early in life result in striking reduction of limbic seizure threshold, suggesting lasting alterations in excitability of the hippocampal network. Supported by NIH NS35439 (TZB) and EFA (CD).