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DO FEBRILE SEIZURES CAUSE TEMPORAL LOBE EPI-LEPSY? AN UPDATE.

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Febrile seizures (FS) are the most prevalent seizures during development. Prospective epidemiologic studies indicate that FS do not progress to temporal lobe epilepsy (TLE). Conversely, in retrospective analyses of adults with TLE, a high prevalence (30–50%) of complex (> 15 min., focal or recurrent) FS during early childhood is found, suggesting an etiologic role in TLE. FS-induced neuronal damage as the primary mechanism of mesial temporal sclerosis, the pathologic hallmark of TLE, has been postulated. However, an alternative mechanism for the correlation of FS and TLE involves pre-existing neuronal injury that triggers both FS and the subsequent TLE. This workshop will discuss electrophysiologic and molecular/structural consequences of FS, using experimental models. First, however, Dr. Jackson will present human imaging data in sets of identical twins, demonstrating that a genetic component is not required for FS and the development of mesial temporal sclerosis. These findings justify using genetically homogeneous animal models to probe potential causal relationships between FS and TLE. Dr. Baram will introduce an appropriate-age model for complex FS that permits prospective controlled studies. She will demonstrate significant, transient injury to hippocampal and amygdala neurons and longterm changes in seizure susceptibility. Dr. Soltesz will provide in vitro electrophysiology evidence for profound and lasting effects of experimental FS on the hippocampal circuit. Lastly, Dr. Sperber, inducing FS in a cortical dysplasia model, will show that in abnormal or injured CNS FS consequences may differ markedly from the case in 'normal' CNS. Together with Baram & Soltesz's data of FS-induced longterm limbic circuit vulnerability to further excitation, Dr. Sperber's data are consistent with a '2 hit', multicomponent mechanism for the correlation of FS and TLE.