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**Altered Mitochondrial Acetylation Profiles in a Kainic Acid Model of  
Temporal Lobe Epilepsy**

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**Abstract**

Impaired bioenergetics and oxidative damage in the mitochondria are implicated in the etiology of temporal lobe epilepsy, and hyperacetylation of mitochondrial proteins has recently emerged as a critical negative regulator of mitochondrial functions. However, the roles of mitochondrial acetylation and activity of the primary mitochondrial deacetylase, SIRT3, have not been explored in acquired epilepsy. We investigated changes in mitochondrial acetylation and SIRT3 activity in the development of chronic epilepsy in the kainic acid rat model of TLE.

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