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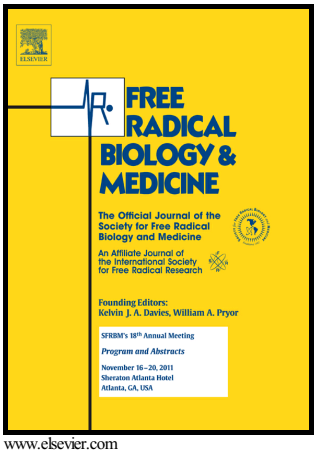

www.elsevier.com

PII: S0891-5849(18)30887-6
DOI: https://doi.org/10.1016/j.freeradbiomed.2018.05.063
Reference: FRB13765

To appear in: *Free Radical Biology and Medicine*

Received date: 15 March 2018
Accepted date: 15 May 2018

Cite this article as: Lindsey B. Gano, Li-Ping Liang, Kristen Ryan, Cole R. Michel, Joe Gomez, Athanassios Vassilopoulos, Nichole Reisdorph, Kristofer S. Fritz and Manisha Patel, Altered Mitochondrial Acetylation Profiles in a Kainic Acid Model of Temporal Lobe Epilepsy, *Free Radical Biology and Medicine,* https://doi.org/10.1016/j.freeradbiomed.2018.05.063

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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.