



Cosponsored by Neuroscience

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Reactive species and mitochondrial mechanisms in neuronal disorders

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WHEN: Thursday, September 21, 2017
12:00 pm

WHERE: Room 2226
Veterinary Medicine Building

HOST: Dr. Thippeswamy

PIZZA SERVED!

Epilepsy is a recent addition to the diverse array of acute and chronic neurological disorders in which the role of oxidative stress and mitochondrial dysfunction is rapidly emerging. Work from our laboratory is focused on identifying disease-modifying redox and metabolic pathways in epilepsy. We have demonstrated increased production of reactive oxygen species and persistent depletion of glutathione during epileptogenesis in animal models of epilepsy. We demonstrated an oxidative post-translational modification via carbonylation of the 75kDa subunit of CI in acquired. A functional consequence of oxidative damage to mitochondria in epilepsy is a bioenergetics decline, which can increase neuronal excitability. We recently demonstrated that increased ROS result in deficits of mitochondrial respiration in acquired epilepsy. Finally, we showed that mitochondrial oxidative stress results in mitochondrial dysfunction and neuronal death which contributes to cognitive deficits associated with chronic epilepsy. The seminar will discuss redox and mitochondrial pathways as novel therapeutic approaches for modifying acquired epilepsy and associated cognitive deficits. Neuroinflammation is an important therapeutic target for disease modification in various neurological diseases including epilepsy. Studies in our laboratory suggest a cross-talk exists between seizure-induced oxidative stress and inflammation. The seminar will discuss the therapeutic strategies to control seizure-induced neuroinflammation by altering redox status.