

Thursday 5<sup>th</sup> March, 4pm  
Wallace Wurth, LGO2

Note the time change - Thursday

## Dr Stefanie Robel

(Fralin Biomedical Research Institute, Virginia Tech School of Neuroscience, Virginia Polytechnic Institute and State University, Roanoke, Virginia)

### *The role of astrocytes in post-traumatic epilepsy*



Dr Stefanie Robel is Assistant Professor at the Glial Biology in Health, Disease, & Cancer Center, Fralin Biomedical Research Institute at Virginia Tech Carilion.

Stefanie's major research interest is how astrocytes shape the development and progression of central nervous system trauma and associated neurological diseases including post-traumatic epilepsy. Dr Robel has a strong expertise in cell biological and functional consequences of astrogliosis, the process that astrocytes undergo in response to injury of the CNS. Astrogliosis has beneficial and detrimental aspects and so far the field has not been able to independently modulate these two aspects. Advanced technology now enables her to aim for a comprehensive understanding of the molecular mechanisms underlying astrocyte physiology and function in the healthy and injured brain. Her research group is actively interrogating the molecular signature of astrogliosis and associated functional changes of astrocytes in different disease contexts by combining genomics and genetic approaches with state-of the art imaging, electrophysiology and clinically relevant models of traumatic brain injury. The long-term goal is to molecularly

dissect beneficial from detrimental aspects of astrogliosis and to identify pathways that allow therapeutic modulation of this process.

**Synopsis:** Focal traumatic brain injury (TBI) is a main cause of acquired epilepsies and induces astroglial scar formation, during which astrocytes lose their homeostatic functions responsible for proper neuronal health and function. Yet, the vast majority of human TBIs also presents with diffuse injury caused by acceleration-deceleration forces on the brain tissue. To determine if astrocyte scar formation is initiated and required for epileptogenesis after diffuse TBI, we modeled this injury type in mice using weight drop injury paradigm. We demonstrated that spontaneous recurrent seizures developed after a latency period. While astrocyte scar formation was absent, we identified an atypical response of astrocytes characterized by the rapid and sustained loss of homeostatic proteins and lack of astrocyte coupling. Areas with atypical astrocytes were larger in animals that later developed TBI seizures suggesting that this astrocytic response may be one root cause of epileptogenesis after diffuse TBI.

All welcome. Drinks and nibbles from 3:30pm, seminar starts at 4pm.

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