Summary of “Glut1 Deficiency mouse slices” presented by Joseph J. Pancrazio, PhD and Ms. Elysandra Solis, from The University of Texas at Dallas

This presentation provided an overview of electrophysiological measurements that aim to contribute to our understanding of how genetic glucose transporter 1 (Glut1) deficiency manifests seizures and approaches to examine potential therapeutic interventions. Based on human electroencephalography and functional imaging, it is understood that these seizures are associated with aberrant thalamocortical oscillations. Prior work using a genetically modified mouse model revealed that inhibitory neuron failure in thalamus and cortex underlies these abnormalities. To capitalize on the G1D mouse model, we described progress to develop a neural circuit testbed. A full description of our work has been recently published as follows:

Solis EM, Good LB, Granja Vázquez R, Patnaik S, Hernandez-Reynoso AG, Ma Q, Angulo G, Dobariya A, Cogan SF, Pancrazio JJ, Pascual JM, Jakkamsetti V (2023) Isolation of the murine Glut1 deficient thalamocortical circuit: wavelet characterization and reverse glucose dependence of low and gamma frequency oscillations. *Frontiers in Neuroscience*, section *Neuroenergetics and Brain Health*. https://doi.org/10.3389/fnins.2023.1191492

Key for our experimental work involves the use of extracellular recording microelectrode arrays (MEAs). When brain slices are placed in intimate contact with the surface of a MEA, it is possible for individual microelectrode recording sites to resolve local field potentials, which represent the ensemble activity of small and local populations of neurons represented by their extracellular potentials. We showed that mouse G1D thalamocortical slices on MEAs exhibit spontaneous low frequency LFPs and less frequent 30–50 Hz or gamma LFPs under near-physiological bath glucose concentration. Using the cortical recordings from layer IV among other regions recorded, we quantified oscillation epochs via an automated wavelet-based algorithm, a method which is analytically superior to power spectral density, short-time Fourier transform, or amplitude-threshold detection. As expected from human observations, increased bath glucose reduced the lower frequency LFPs while augmenting the gamma LFPs in G1D slices, likely reflecting strengthened inhibitory neuron activity. This approach provides an ex vivo method for the evaluation of mechanisms, fuels, and pharmacological agents in a crucial G1D epileptogenic circuit.