One of the leading causes of epilepsy is cerebral ischemia, also known as ischemic stroke. The brain is particularly vulnerable to ischemia due to the high rate of oxidative metabolism, which requires a continuous supply of oxygen and glucose. By initiating an excitotoxic glutamate injury, it is hypothesized to induce ischemic epileptic discharges in the rat neural stem cells in-vitro. Using the cell-type specific miRNA biochemical marker of normal brain function, it is possible to identify the pathology of stroke specific miRNAs. Signaling pathways, such as the MAPK pathways, have demonstrated participation in the pathogenesis of ischemic stroke. By observing the dysregulation of the target gene hsa-miR-106b-5p due to the glutamate injury, the MAPK pathway will be analyzed by targeting the MAPKK protein markers. It is hypothesized that the upregulation of hsa-miR-106b-5p will affect the tightly regulated signaling network of the MAPKK activity in the event of a stroke.