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West Nile Virus (WNV) is the most common cause of epidemic viral encephalitis in the United States. More than half of patients who survive West Nile neuroinvasive Disease (WNND) exhibit chronic cognitive impairments, such as spatial learning deficits, that resulted from the disease. Little is known about the cellular and molecular mechanisms that underlie the neurocognitive sequela that follows the infection. Previously, the lab discovered that some infected wild type mice exhibited spatial learning deficits while some did not, these mice were labeled "good learners" and "poor learners". Something is naturally protecting the good learners from the spatial learning defects. The lab identified more than thirty target genes that could play a role in this protective mechanism. Because interferon gamma receptor (IFNgR) knockout mice are also protected from spatial learning defects, we used IFNgR knockout mice as a model to study the natural mechanism present in the good learners. We hypothesized that alterations in gene expression that are conserved between "good learners" and IFNgR knockout animals following WNV infection would point to a common protective mechanism. qPCR was done to compare gene expression in good versus poor learners and infected IFNgR versus wild type mice. The qPCR reactions revealed a significant decrease in the expression of Crry, a complement regulatory gene, in the infected IFNgR knockout mice hippocampi compared to wild type mice. Decreased expression of Crry was also seen in the hippocampi of WNV good learners when compared to the poor learners. Our next step is to look at gene expression at earlier time points to see if the protective mechanism works acutely or if it is a continuous process, long after the virus has cleared. Determining the details of this protective mechanism could lead to therapies for people experiencing cognitive deficits following WNV infection.