NYU STUDY REVEALS HOW BRAIN’S IMMUNE SYSTEM FIGHTS VIRAL ENCEPHALITIS

New York University biologists have uncovered how the innate immune system in mice’s brains fights viral infection of neurons. The findings, published as the cover study in the latest issue of Virology, show that proteins in neurons fight the virus at multiple stages—by preventing the formation of viral RNA and proteins, and blocking the virus’ release, which could infect other cells in the brain.

“There is no magic bullet in fighting viral infections in neurons,” said NYU Biology Professor Carol Shoshkes Reiss, the study’s senior author. “However, these findings show the redundancy of the immune system—when one response fails to fight infection, others step in.”

The study was also conducted at NYU, by a post-doctoral fellow, Mark Trottier, Jr., PhD, now at Michigan State, and Beth Palian, currently a doctoral student at the University of Southern California.

Recently, the West Nile virus has been responsible for a viral encephalitis outbreak in the northeast. The NYU researchers set out to determine how the body can fight viral encephalitis. Specifically, they examined how type I interferons—proteins made by the body that are released in response to stimuli, notably infection—work in neurons and to determine if nerve cells’ response to interferons is similar to that of other cells.

Examining the effect of the virus in mice and in cell culture, the researchers found that neurons are sensitive to the protective effect of interferons, inducing pathways to fight the virus’ spread. However, their findings showed that interferons fight the virus at different stages of the virus’ life cycle. First, they inhibit viral RNA and protein synthesis. If this fails, interferons block the virus from forming particles which can be released and infect other neurons. This is critical, since the immune system does not kill infected precious neurons the way it does other cells, which can be replaced.

The researchers attributed the spread of viral encephalitis to the inability of lab mice to produce sufficient amounts of interferons to fight the virus.