Neuroscientist says a critical protein prevents secondary damage after stroke

November 2 2011, By Robin Lally

(Medical Xpress) -- One of two proteins that regulate nerve cells and assist in overall brain function may be the key to preventing long-term damage as a result of a stroke, the leading cause of disability and third leading cause of death in the United States.

In a recent study published in the Journal of Neuroscience, Bonnie Firestein, professor of cell biology and neuroscience, in the School of Arts and Sciences, says the new research indicates that increased production of two proteins – cypin and PSD-95 – results in very different outcomes.

While cypin – a protein that regulates nerve cell and neuron branching critical to normal brain functioning -- prevents nerve cells not damaged during the initial stroke from losing the ability to communicate with other cells and halts any secondary brain or neurological damage, PSD-95 accelerates cell destruction and inhibits recovery. Secondary injury from a stroke can occur days or even weeks after the injury and often includes a lack of blood flow, insufficient oxygen, and swelling of the brain.

“We don’t know how or why cypin acts during this process, but what we do know is that cypin helps nerve cells survive,” said Firestein, who first isolated and identified cypin more than a decade ago. Since then, she has been researching how it works in the brain and could be used to treat traumatic brain injury and other serious neurological disorders.

Firestein and her former graduate student Chia-Yi Tseng conducted the laboratory research by putting nerve cells in a dish and creating an "experimental stroke" – mimicking a massive amount of glutamate released, resulting in nerve cells destroyed.

They wanted to determine if anything could be done to stop the secondary damage that occurs after a stroke and discovered that while a greater number of neurons that survived the stroke were spared secondary destruction with increased amounts of cypin, too much PSD-95 resulted in the death of nerve cells not damaged inititally.

"I would hope that this research aids in the development of an effective therapeutic intervention, saving neurons and reducing the long-term effects of stroke and other traumatic brain injuries," said Firestein.

Provided by Rutgers University