Fruit fly neuron can reprogram itself after injury

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Studies with fruit flies have shown that the specialized nerve cells called neurons can rebuild themselves after injury.

These results, potentially relevant to research efforts to improve the treatment of patients with traumatic nerve damage or neurodegenerative disease, were presented at the American Society for Cell Biology 49th Annual Meeting, Dec. 5-9, 2009 in San Diego.

An injured neuron's remarkable ability to reprogram itself was reported by Michelle Stone, Melissa Rolls, Ph.D., and colleagues at Penn State University (PSU).

The scientists said that the reprogramming was particularly surprising because once formed, neurons normally are relatively staid and stable, and because the injured part of the neuron was "replaced" by a very structurally and functionally different component of the same cell.

It's in the nature of the neuron's job to maintain and keep the parts that were present when it was created.

The PSU scientists experimentally induced the injury to the neuron in the Drosophila melanogaster's brain by using a laser to cut off the entire axon, the long, slender part that sends the neuron's signals to the brain's other cells.

In response to the injury, the dendrites, the parts of the neuron that receives signals from other cells in the brain, reshuffled their internal support structures, or cytoskeletons. Eventually one of the dendrites switched its polarity and grew into a permanent replacement for the damaged axon.

Polarity refers to the varying degrees of structural and functional asymmetry that characterize the neuron's parts.

The polarity of the microtubules in axons and dendrites differ, reflecting the different roles of these cellular structures.

Microtubules, the basic units of the cytoskeleton, are polymerized proteins that stack up into a strong, stable and highly polarized cellular backbone that also acts as an internal trackway for long-range intracellular transport.

When the researchers used a laser to cut off the entire axon of the Drosophila neuron, the supposedly stable dendritic microtubules burst into action, essentially deconstructing and rebuilding the entire dendritic microtubule cytoskeleton.

Growing microtubules dramatically increased in number, and their polarity became fluid. This dynamic microtubule response, the researchers said, was specific to axon, not dendrite, injury.

Only two to three days after the injury, the one dendrite finally took on its new axonal microtubule polarity and began forming an axon.

Microtubule dynamics settled down in the remaining dendrites, assuming the normal overall layout that characterized the neuron before the injury. The repaired neuron had one axon and several dendrites with opposite microtubule orientation.

Stone and colleagues said that they believe that exploring microtubule control is critical to understanding how axons regenerate from dendrites.

By manipulating levels of intracellular proteins, the researchers discovered that they could speed up the dendrite-to-axon transformation by slowing down microtubule dynamics.

This antagonism could be important to allow the neuron to correctly repolarize its microtubules

before growing the new axon, they said. Finding the exact mechanisms that control dendrite regeneration is the work ahead.

Source: American Society for Cell Biology