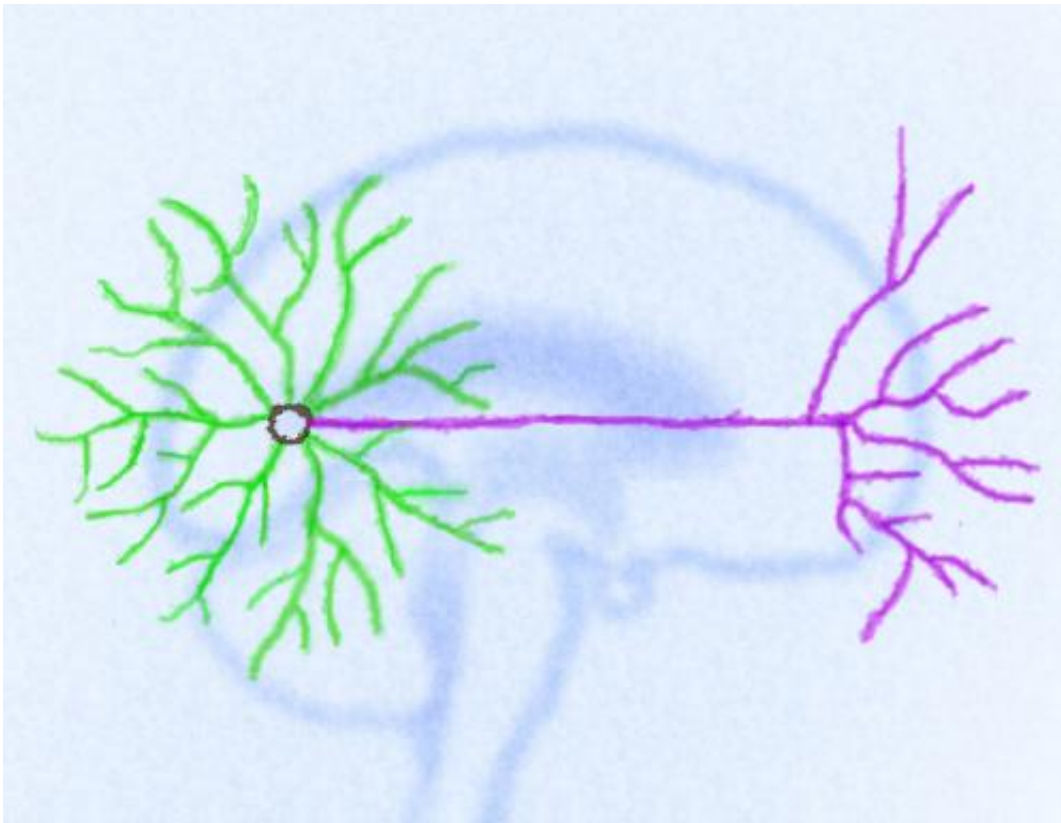


Neuronal regeneration and the two-part design of nerves

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A neuron contains two sets of protrusions of different functions: dendrites (shown in green) receive signals from other neurons or sensory stimuli, whereas the axons (shown in purple) pass signals to other neurons or muscles. Such a two-part design serves as a basis for the functioning neural networks inside of our brains, in a way that is similar to diodes in electric circuits. Credit: Xin Wang

Researchers at the University of Michigan have evidence that a single

gene controls both halves of nerve cells, and their research demonstrates the need to consider that design in the development of new treatments for regeneration of nerve cells.

A paper published online in *PLOS Biology* by U-M Life Sciences Institute faculty member Bing Ye and colleagues shows that manipulating genes of the fruit fly *Drosophila* to promote the growth of one part of the neuron simultaneously stunts the growth of the other part.

Understanding this bimodal nature of [neurons](#) is important for researchers developing therapies for [spinal cord](#) injury, neurodegeneration and other nervous system diseases, Ye said.

Nerve cells look strikingly like trees, with a crown of "branches" converging at a "trunk." The branches, called dendrites, input information from other neurons into the nerve cell. The trunk, or axon, transmits the signal to the next cell.

"If you want to regenerate an axon to repair an injury, you have to take care of the other end, too," said Ye, assistant professor in the Department of Cell and Developmental Biology at the U-M Medical School.

The separation of the nerve cell into these two parts is so fundamental to neuroscience that it's known as the "neuron doctrine," but how exactly neurons create, maintain and regulate these two separate parts and functions is still largely unknown.

While the body is growing, the [neuronal network](#) grows rapidly. But nerve cells don't divide and replicate like other cells in the body (instead, a specific type of stem cell creates them). Adult nerve cells appear to no longer have the drive to grow, so the loss of neurons due to injury or [neurodegeneration](#) can be permanent.

Ye's paper highlights the bimodal nature of neurons by explaining how a [kinase](#) that promotes axon growth surprisingly has the opposite effect of impeding dendrite growth of the same cell.

In the quest to understand the fundamentals of nerve cell growth in order to stimulate regrowth after injury, scientists have identified the genes responsible for axon growth and were able to induce dramatic growth of the long "trunk" of the cell, but less attention has been given to dendrites.

There are technical reasons that studying axons is easier than studying dendrites: The bundle of axons in a nerve is easier to track under the microscope, but to get an image of dendrites would require labeling single neurons.

Ye's lab circumvented that obstacle by using *Drosophila* as a model. Using this simple model of the nervous system, the scientists were able to reliably label both axons and dendrites of single neurons and see what happened to nerve cells with various mutations of genes that are shared between the flies and humans.

One of the genes shared by *Drosophila* and people is the one that makes a protein called Dual Lucine Zipper Kinase, or DLK. As described previously by other groups, DLK is a product of the gene responsible for axon growth. Cells with more of the protein had very long axons, and those without the gene or protein had no regeneration after nerve injury. The DLK kinase seemed a promising target for therapies to regenerate [nerve cells](#).

However, Ye's lab found that the kinase had the opposite effect on the dendrites: Lots of DLK leads to diminished dendrites.

"This in vivo evidence of bimodal control of neuronal growth calls attention to the need to look at the other side of a neuron in terms of

developing new therapies," Ye said. "If we use this kinase, DLK, as a drug target for [axon growth](#), we'll have to figure out a way to block its effect on dendrites."

Provided by University of Michigan

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