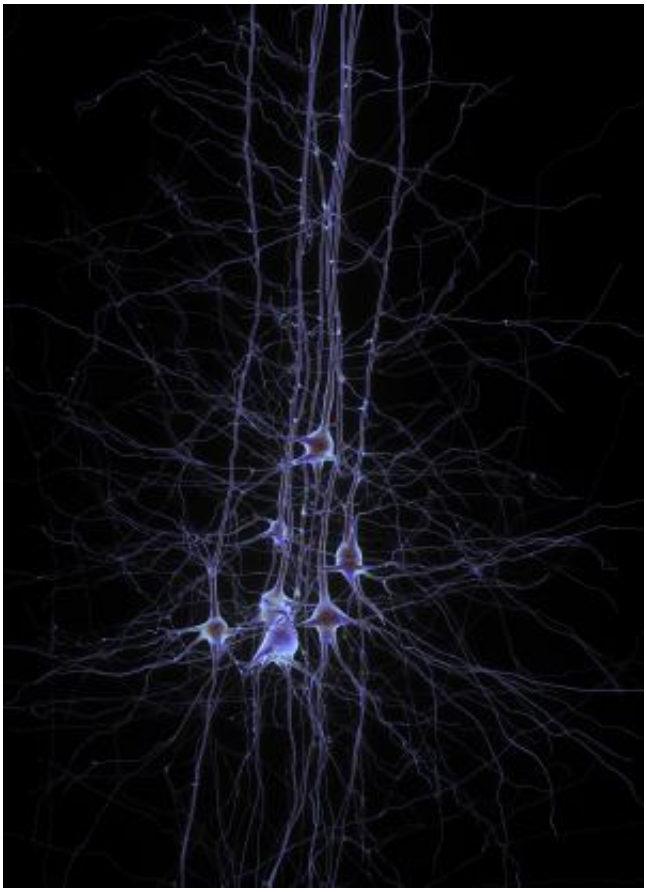
Changing clocks and changing seasons: Scientists find role for neuronal plasticity

July 30 2015



This is a group of neurons. Credit: EPFL/Human Brain Project

A team of scientists has linked changes in the structure of a handful of central brain neurons to understanding how animals adjust to changing seasons. Its findings enhance our understanding of the mechanisms vital

to the regulation of our circadian system, or internal clock.

The work, which appears in the journal *Cell*, focuses on the regulation of "neuronal plasticity"—changes in neuronal structure—and its function in the brain.

"Neuronal plasticity underpins learning and memory, but it is very challenging to tie changes in specific neurons to alterations in animal behavior," explains Justin Blau, the paper's senior author and a professor in NYU's Department of Biology and at NYU Abu Dhabi. "In our research, we've discovered how plasticity of a very small number of neurons helps run the biological clock and aids transitions to different seasons."

The paper's other authors were Afroditi Petsakou, a recent PhD graduate from NYU's Department of Biology, and Themistoklis Sapsis, an assistant professor at MIT.

In their study, the researchers focused on the principal s-LNv clock neurons in the fruit fly Drosophila, which is commonly used for research on circadian rhythms—earlier studies of "clock genes" in fruit flies lead to the identification of similarly functioning genes in humans.

Specifically, their work centered on the ends/tips of the axons of these neurons, where they release their signals. Previous research had established that these termini change their structure with a 24-hour rhythm, but it was unclear what function these alterations served.

In the *Cell* study, the scientists quantified the daily changes in s-LNv axon termini and found that they grow and retract every 24 hours. They also identified the protein that drives these rhythms in neuronal plasticity: Rho1. Moreover, they found that plasticity of the s-LNvs is required both for maintaining circadian rhythms (the biological clock)

and for allowing seasonal adaptation of these rhythms. Specifically, if s-LNvs are unable to retract then flies behave normally in winter but fail to predict the early dawn of long summer days. Conversely, if s-LNvs remain in a retracted state, then flies behave as if they are in summer on both short and long days.

They also found rhythms in the proteins at the ends of the s-LNv axons. At dawn, s-LNvs have high levels of proteins involved in sending signals and low levels of the proteins that allow them to receive signals. The opposite is true at dusk. This unusual type of neuronal plasticity suggests that the function of s-LNvs changes dramatically over the day: from mainly sending signals at dawn to mainly receiving signals at dusk.

The findings may also shed new light on a human affliction, spinocerebellar ataxia—a neurodegenerative disease that affects coordination and movement. Blau's group found that the daily changes in Rho1 activity are controlled by rhythms in transcription of a gene very similar to human Puratrophin-1.

"Since some forms of spinocerebellar ataxia are associated with mutations in human Puratrophin-1, our data support the idea that defective neuronal plasticity underlies loss of motor control and leads to neurodegeneration," notes Blau.

Provided by New York University