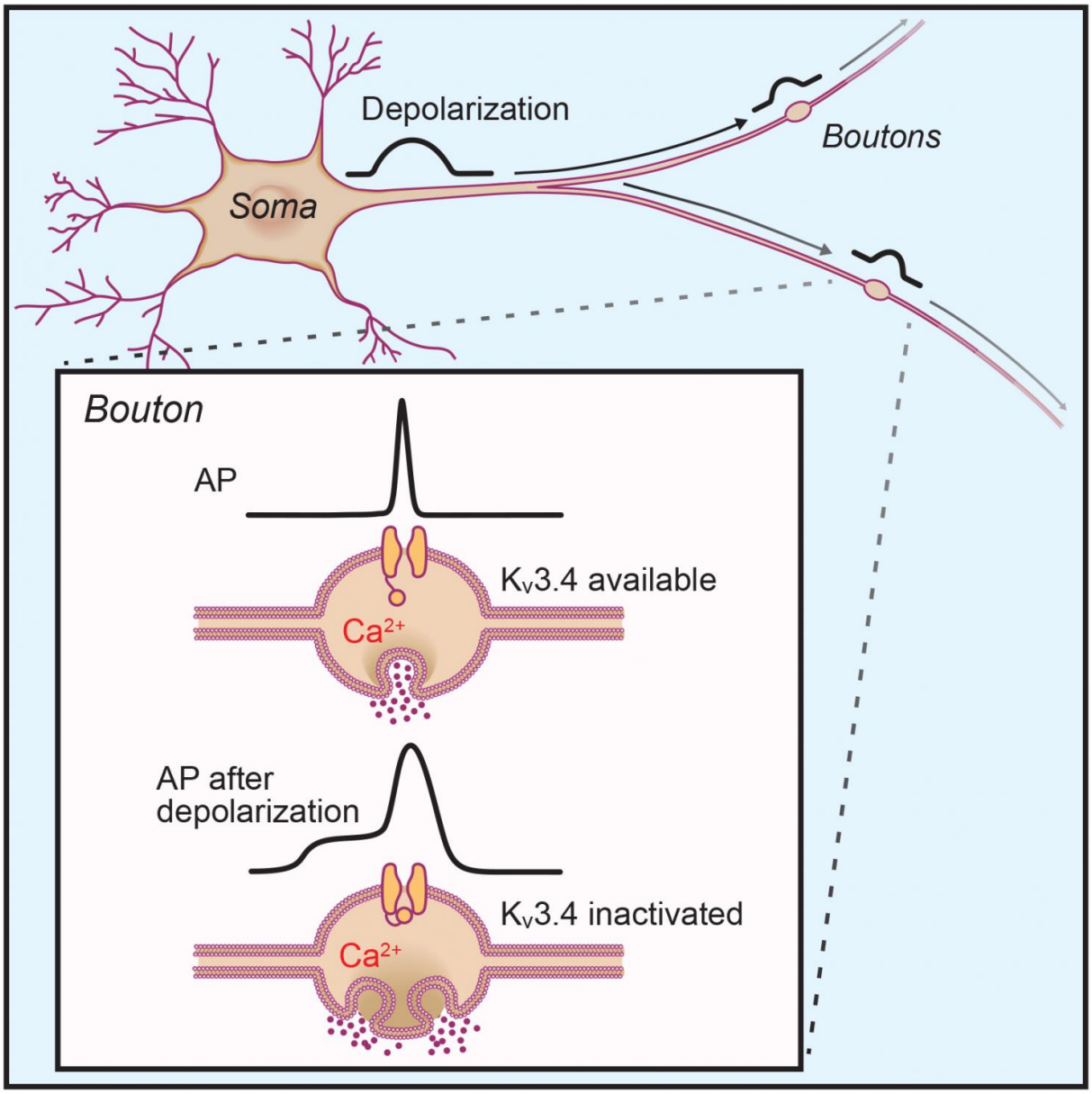
New insights into the information processing of motor neurons

February 21 2017



A form of short-term neuronal plasticity known as analog-to-digital facilitation can result from brief somatic depolarizations lasting as little as 100 milliseconds. Such activity transiently makes Kv3.4 subunit containing channels unavailable thereby increasing the duration of the presynaptic spike and enhancing neurotransmitter release. Credit: Max Planck Florida Institute for Neuroscience

In a study published in *Cell Reports* in February 2017, Matt Rowan, Ph.D., a Post-doctoral researcher in the lab of Dr. Jason Christie, sought to understand the molecular mechanisms behind a type of short-term neuronal plasticity that may have importance for motor control. The team showed that this type of plasticity can impact neurotransmission in as little as 100 milliseconds and depends upon inactivation of Kv3 channels. Interestingly, the team also found that this type of plasticity occurs more readily in juvenile brains than in mature ones.

Neuronal communication is frequently described simply as an all-or-nothing event. If a neuron is depolarized enough, it will fire and release neurotransmitters to communicate with another neuron; if it doesn't reach the threshold to fire, it doesn't send a signal at all. However, depolarizations that don't reach the threshold to make the neuron fire can still impact neurotransmission. The depolarization spreads throughout the neuron, and when the neuron does eventually reach the threshold to fire, it releases a stronger signal with more neurotransmitters. This is known as analog-to-digital facilitation, a type of short-term plasticity.

"This has been seen before, and we're adding a molecular mechanism showing exactly the molecule you need to get this sort of facilitation," explained Rowan.

Researchers were already aware that this type of short-term plasticity exists, but had struggled to view it directly because the axons that utilize

this type of plasticity are difficult for scientists to access. This means that some of the molecular mechanisms behind the phenomenon remain mysterious. For the current study, the team used novel techniques for voltage imaging and patch clamp recordings that allowed them to visualize and record from these tiny sections of individual neurons.

The researchers observed analog-to-digital facilitation as it occurred in experimental models. They showed that subthreshold depolarization spreads from the body of the neuron down its axon, the long extension through which action potentials travel before causing the neurons to release neurotransmitters into a synapse. Here, subthreshold depolarizations impacted neurotransmission in the juvenile models by briefly making Kv3.4 channel unavailable thereby increasing the duration of the presynaptic spike. The fact that the group observed less of this same plasticity in mature models suggests that learning and experience may temper this type of plasticity as an animal matures.

The team chose to study inhibitory interneurons in the cerebellum because they play an especially important role in the function of circuits throughout the cerebellum as well as the rest of the brain. Understanding this type of neuronal plasticity may have important implications for understanding motor disorders such as cerebellar ataxia, a disorder that can cause a variety of motor problems in humans ranging from increased falling to difficulty with speech and swallowing.

More information: Rapid state-dependent alteration in Kv3 channel availability drives flexible synaptic signaling dependent on somatic subthreshold depolarization, *Cell Reports*, February 21 2017, DOI: 10.1016/j.celrep.2017.01.068

Provided by Max Planck Florida Institute for Neuroscience