This Week in The Journal

A Potassium Channel on Axons Reduces Ectopic Spikes

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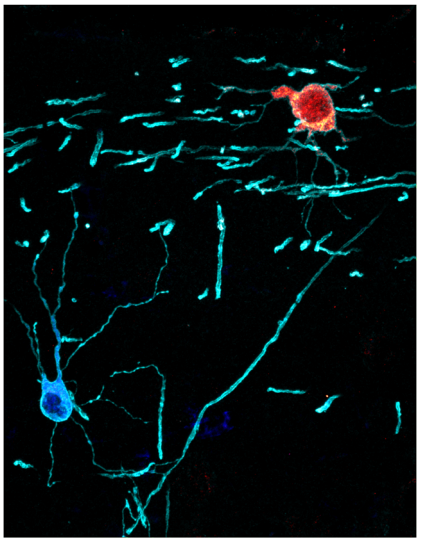
(see article e1889242025)

Action potentials, or spikes, transmit information between neurons over long distances by propagating down the axon. The axon must be excitable enough to propagate spikes reliably, but not so excitable that new spikes are initiated ectopically in the axon. Abdollahi and colleagues explored what channels reduce the risk of ectopic spikes. They evoked spikes in different subcellular compartments of hippocampal pyramidal neurons using targeted optogenetic stimulation. Abdollahi et al. discovered that spike initiation differed between the soma and axon. The soma used a low-pass filter that allows graded depolarization to evoke spikes de novo, whereas the axon used a high-pass filter that allows only rapid depolarization to evoke spikes. This high-pass filter was driven by type 1 voltage-gated potassium channels whose activation dampened slow depolarization. This study advances our understanding of this potassium channel by suggesting it controls ectopic spike initiation.



Pictured are two oligodendrocytes in layer 1 of the mouse cerebral cortex. Both cells are labeled by CNP (cyan), an oligodendrocyte marker. At the left is a mature oligodendrocyte, as evidenced by colabeling with carbonic anhydrase II (blue). At the right is a differentiating oligodendrocyte, as evidenced by a larger soma and labeling with procaspase-3 (red). See Kamen et al. for more information.

Identifying a Regulator of Oligodendrocyte Fate

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(see article e2066242025)

Oligodendrocyte death occurs during aging and characterizes several disorders. While oligodendrocyte regeneration even in neurodegenerative conditions is possible throughout life, the regenerative process has a high failure rate and is not efficient. In this issue, Kamen et al. explored regulators of oligodendrocyte development during stages of generation. A barrier to delineating stages of oligodendrocyte development is the lack of known unique molecular markers at each stage. In their study, the authors identified a marker that is upregulated during oligodendrocyte differentiation in both males and females: the cytoplasmic inactive enzyme precursor procaspase-3. This marker enabled Kamen and colleagues to discover that oligodendrocyte differentiation continues in the cortex and white matter into old age. Inhibiting both the active and inactive form of caspase-3 resulted in less oligodendrocytes, suggesting that procaspase-3 upregulation promotes oligodendrocyte development. These findings shed light on underlying mechanisms of oligodendrocyte fate and point to procaspase-3 as an effective marker for differentiation.

This Week in The Journal was written by Paige McKeon

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