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Editorial

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Dendrites are complicated, branched processes that typically comprise the vast majority of neuronal surface area. Here thousands of synapses coverage from varied sources. However, rather than simply channeling synaptic information toward the soma and axon where action potentials (APs) are initiated, dendrites combine and transform synaptic inputs in ways that are still not fully understood, despite decades of directed research. Pressed with the job of receiving, integrating, and transforming excitatory and inhibitory synaptic information into meaningful AP output, dendritic integration and plasticity are likely commonly affected in neurological disorders of diverse molecular origins.

In this Special Issue focusing on *Dendrites and Disease*, Lucy Palmer [4] provides a historical review of dendritic integration and how various aspects of its dysfunction may lead to neurological diseases. Brager and Johnston [1] review recent research identifying several channelopathies associated with a mouse model of Fragile X syndrome. These findings expand the current predominant theory of a synaptic locus of Fragile X syndrome, to include role of dendritic voltage-gated ion channels in its pathophysiology. Many dendritic processes have been implicated in the pathogenesis of Alzheimer's disease. Cochran et al. [3] provide a systematic review of these with a focus on those with support from human tissue. From these studies they propose a "dendritic hypothesis" for Alzheimer's disease that may serve as a framework for therapeutic strategies.

Dendritic spines are complicated structures tasked with transforming chemical neurotransmitters signals back into electric signals. Pathological studies have found changes in spine size and number associated with numerous disorders of the central nervous system. Remmers et al. [5] present mounting evidence of an important role for the guanine-nucleotide exchange factor, Kalirin as a regulator of structural and functional plasticity in dendritic spines. They propose that problems with dendritic spine plasticity associated with altered Kalirin signaling may contribute to the pathogenesis of several psychiatric and neurological disorders.

Finally, Casanova et al. [2] review studies showing a common effect of seizures on dendritic branch and spine morphology that may contribute to intellectual developmental disabilities associated with childhood epilepsy. They review relevant molecular mechanisms potentially involved with growth suppression, which may lead to treatments aimed toward improving the intellectual development of children with epilepsy syndromes.

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