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**The Role of Cypin in Neuronal Development**

Synaptic transmission requires spatial assembly of neurotransmitter receptors and associated signal transduction machinery at synaptic sites and the precise patterning of dendritic processes. Targeting of proteins to the synapse is a dynamic process, in which there is a balance of assembly and disassembly of proteins at synaptic homeostasis. In fact, when learning occurs, recruitment of existing and newly synthesized proteins to the synapse is increased. In contrast, when disassembly of synaptic signaling molecules occurs faster than assembly, homeostasis is lost and disease states such as Alzheimer's disease occur in which synaptic transmission is compromised. An important long-term goal our work is to understand how synaptic targeting of proteins is regulated during development and homeostasis and how this trafficking is perturbed in pathophysiological states.

Our most recent studies focus on dendrite branching. The amount of branches that a dendrite, or input center of a neuron, contains is thought to be directly related to learning and memory. In fact, in a number of learning disorders, such as autism, Rett Syndrome, Down syndrome, and Alzheimer's disease, patients show a reduced number of dendrite branches. These patients also often show alterations in the metabolism, or breakdown, of a class of compounds called purines. My laboratory has recently discovered that cypin regulates the number of dendrite branches in areas of the brain related to learning and memory (Akum et al., 2004). We have found that cypin promotes microtubule assembly and that branching in vivo correlates with cypin's activity as an enzyme involved in purine metabolism. The ultimate goal would be to be able to develop pharmaceutical agents to help patients with autism, Rett Syndrome, Down syndrome, Alzheimer's disease and other disorders to increase memory