Editorial

Since their discovery about ten years ago, Cdk5 and its specific activators, p25, p35 and p39 have emerged as key players in nervous systems. The kinase has been implicated in a wide variety of brain functions ranging from drug addiction to learning and memory. Deregulation of Cdk5 has been suggested to contribute to the pathology of several common neurodegenerative diseases including Alzheimer's disease and amyotrophic lateral sclerosis. The literature on Cdk5 is expanding rapidly, and the contributions have come from neuroscientists of diverse disciplines. Cdk5 and its activators have been demonstrated to associate with and to phosphorylate a wide variety of cellular proteins implicating the kinase in diverse neuronal processes, such as neurocytoskeleton dynamics, neuro-signaling, axonal guidance, membrane recycling, synaptic function etc. Crystal structure of Cdk5-p25 has been elucidated providing the molecular basis for the understanding of the unique regulatory properties of the enzyme that has long fascinated researchers in the Cdk and neuroscience fields. This special issue of *Neurosignals* contains up-to-date and critical reviews on various aspects of Cdk5. We are very grateful that many of the leading scientists in the field have made timely contributions to this issue.

Li-Huei Tsai, Boston, Mass.
Jerry H.C. Wang, Hong Kong