Special Issue

Mechanism of Cellular Signaling, Dysfunction, and Drug Effects on Alzheimer's Disease

Message from the Guest Editor

The pathogenesis of Alzheimer's Disease (AD) remains not completely understood to this day. This is thought to be one important reason as to why no present therapy is able to cure or stop the progression of this disease. Several different pathways are considered as important players in the initiation and/or progression of the disease; the currently most accepted theory is the cascade of pathological mechanisms initiated by soluble or fibrillar forms of Abeta, including increased inflammation, mitochondrial failure, disruption of Ca++ and protein homeostasis, and alterations or loss of synapses, among others. Considering the complexity of AD and the failure or only very limited clinical effects of most single-targeted therapies, it seems to be essential (1) to elucidate further details of and connections to the intricate cellular and molecular mechanisms involved in the pathophysiology of AD and (2) to strive in advancing future therapeutic approaches that target several pathogenic factors of the disease in a stage-dependent manner as much as possible.

Guest Editor

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