Special Issue

Cognitive Function and Alzheimer's Disease

Message from the Guest Editors

Alzheimer's disease is the leading cause of dementia among the aged population and is recorded as one of the most well-known medical problems today. Late-onset Alzheimer's disease (LOAD) is a complex and heterogeneous disease. As of now, there are neither modification treatments nor a cure found for this compelling disease. As there has recently been an increase in the aging population around this world, the need for designing novel biomarker and therapeutic targets has become more vital toward achieving these aims. However, mechanisms linking Alzheimer's disease and cognitive impairment are also not clearly elucidated. Throughout the decades, many hypotheses have been developed to explain the pathogenesis of Alzheimer's disease, including the A☐-amyloid hypothesis, A☐-amyloid oligomer hypothesis, presenilin hypothesis, Ca2+ dysregulation hypothesis, lysosome hypothesis, infection hypothesis, and tau hypothesis. To achieve these aims, more comprehensive knowledge of the prime molecular mechanisms of Alzheimer's disease determining cognitive impairment is required, which will ultimately lead to therapeutic targets.

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