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Cognitive Function and Alzheimer's Disease

Guest Editors:

Dr. Priya Madhavan
School of Medicine, Faculty of Health & Medical Sciences, Taylor's University, Subang Jaya, Malaysia

Dr. Mohamed Saleem Abdul Shukkoor
College of Pharmacy, Riyadh Elm University, Riyadh, Saudi Arabia

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Message from the Guest Editors

Dear Colleagues,

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 Aβ-amyloid hypothesis, Aβ-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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Editor-in-Chief

Prof. Dr. Stephen D. Meriney
Department of Neuroscience,
University of Pittsburgh,
Pittsburgh, PA 15260, USA

Message from the Editor-in-Chief

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Contact Us

Brain Sciences Editorial Office
MDPI, St. Alban-Anlage 66
4052 Basel, Switzerland

Tel: +41 61 683 77 34
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