Alzheimer disease (AD) is a devastating neurological disorder, the most common form of dementia. To date, the cause of AD has eluded us but a number of hypotheses have been put forward to try to understand the mechanism(s) involved. Amongst them, the genetics hypothesis, cholinergic hypothesis, amyloid hypothesis, and tau hypothesis are the most common. Above all, the principal risk factor for AD is age. The incidence of the disease doubles every 5 years after 65 years of age. Recent insights have shown that AD causes biochemical and physiological changes in the brain years before the onset of dementia symptoms. For this reason, developing drugs that can be used in that early stage to prevent the disease is of great importance.

According to one of the most famous AD foundations, the Alzheimer’s Foundation of America (AFA), a simple definition of the disease is ‘a progressive, degenerative disorder that attacks the brain’s nerve cells or neurons, resulting in loss of memory, thinking and language skills, and behavioral changes’. Understanding the cause(s) of AD would clearly facilitate the search for a cure. Advances in molecular medicine/genetics have resulted in a better understanding of the genesis of AD. Many molecular mechanisms have been suggested in AD, but the overarching theme emerging from the data is that an accumulation of misfolded proteins in the aging brain results in oxidative and inflammatory damage, leading finally to energy failure and synaptic dysfunction. Researchers inserted a mutant form of the human tau gene into a mouse model of AD and were able to turn the gene off and on using a drug. After the mice had developed neurofibrillary tangles and brain cell degeneration (like that seen in AD), the gene was turned off and the tangles disappeared and neurodegeneration was partially reversed. This finding shows us that drugs targeting abnormal tau protein may deserve the greatest attention for clarifying the pathology and symptoms of the disease. It is important for clinicians and health professionals to understand the mechanisms involved in memory functioning in order to be able to recognise and treat deficits presenting as a consequence of dementia. Upon advancement in technological devices such as PET and SPET scans, MRI, and fMRI (used in conjunction with neuropsychological tests administered at key time points including follow-ups), clinicians are better placed to foresee a more reliable diagnosis and prognosis than in the past. Treatment with acetylcholinesterase inhibitors can temporarily alleviate some symptoms but does not modify disease progression. Therefore, new therapy regimens and therapeutic modalities are urgently needed to cope with the disease and its symptoms.

In this issue, the readers will find 29 original and review articles, which are very important and attempt to provide a better understanding of AD in many aspects. All of them address both basic and clinical science issues associated with AD and its potential impact on the general population. This issue also assembles a variety of findings relevant to the mechanism of AD and ties them together using a current understanding of the basis of the loss of cognition: the accumulation of misfolded proteins, which cause oxidative and inflammatory damage to the brain and, ultimately, synaptic dysfunction. The proposed concept is timely due to the increased aging population. The readers’ interest will be drawn to several clinically relevant markers/parameters from the patients or experimental setup. The articles encompass a wide spectrum of topics ranging from forensic medicine, neurology, and radiology to molecular genetics, anesthesiology, and clinical biochemistry. In general, they offer a good challenge as regards the pathophysiology of AD and new curative approaches.

Collectively, these papers give a broad insight into certain issues related to AD. We hope that readers will also find them inspiring. We also hope that readers will find this special issue exciting and useful for speculating and conjecturing about future trends in the field. This is an appropriate time and place to thank all the referees for their guiding influence. Without their help, for sure, this special issue would have not been possible.
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Guest Editors

Mark S Kindy, PhD, Professor,
Departments of Neurosciences, Regenerative Medicine and Cell Biology,
Medical University of South Carolina, Senior Research Career Scientist,
Ralph H Johnson VA Medical Center, Charleston, SC 29425, USA

Ömer Akyol, MD, PhD, Professor
Department of Biochemistry,
Faculty of Medicine, Hacettepe University,
Ankara, Turkey

Kumar Sambamurti, PhD, Professor,
Department of Neurosciences,
Medical University of South Carolina,
Charleston, SC 29425, USA