FOREWORD

Alzheimer's disease (AD) is the sixth leading cause of death in the USA. Globally about 50 million individuals have AD or related dementias. With the increasing average age of humans worldwide, the total number of people with dementia is projected to reach 82 million by 2030 and 152 million by 2050. Despite its prevalence, AD is the only cause of death among the top 10 causes of death globally for which no effective pharmaceutical agents exist to halt or slow down the disease progression. By some estimates, AD and related dementias are the single most expensive medical condition. In 2019, direct costs of AD in the USA will be ~$290 billion, which is expected to rise to ~$1.1 trillion by 2050 if no treatments are developed. Hence, there is a tremendous imperative to gain a better understanding of the pathogenesis of AD and to develop effective treatments. AD is a complex, multifactorial disease, which is unique to humans. AD is defined neuropathologically by the accumulation of amyloid β (Aβ) into extracellular plaques in the brain parenchyma and in the vasculature (known as congophilic amyloid angiopathy [CAA]), and abnormally phosphorylated tau that accumulates intraneuronally forming neurofibrillary tangles (NFTs). Pathological aggregation of phosphorylated tau and Aβ occurs in a sequential process. Monomers first aggregate into oligomers intraneuronally that then further aggregate into the fibrils observed in amyloid plaques and NFTs. This pathology then spreads in a characteristic brain topography that is distinct for NFTs and plaques. This process develops over many years, with a preclinical period of two to three decades, the onset of which is modulated by apolipoprotein E (apoE) genotype, as well as other genetic and environmental risk factors.

This book integrates considerable expertise from a wide range of authors from different disciplines. It includes clinicians through to translational and basic scientists. In aggregate, this book provides a comprehensive and up-to-date overview of AD. It covers the heterogeneous underlying AD pathology, with a review of genetic and proteomic approaches to better understand the disease. In addition, there is an extensive review of various potential contributing factors to the emergence of AD, as well as a discussion of novel biomarkers and potential effective therapeutic approaches. I trust that these reviews will be of value to clinicians and health professionals caring for patients with AD, and will provide a comprehensive and thought-provoking introduction to young investigators interested in translational aspects of the AD and related dementias field.

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