The downside of the current tendency to prolonged life expectancy in developed countries is the increase in diseases associated with advanced age, especially those involving the central nervous system (CNS). Foremost among these is sporadic Alzheimer’s disease (AD) which leads to dementia (Brookmeyer et al. 2007; Qiu et al. 2009; Reitz et al. 2011; Mayeux and Stern 2012). Nevertheless, today, despite all efforts on numerous fronts, no causal or disease-modifying therapy is available (Doody et al. 2014; Salloway et al. 2014). AD is a neurological disorder of the human CNS. The pathological lesions associated with the AD process require an unusually long period of time to evolve, but, in the final analysis, they result in clinically recognizable impairment of higher brain functions.

This book is written for a readership that is to some extent familiar with the anatomy of the human nervous system and is interested in the changes it undergoes during the AD process. As in the previously published book on sporadic Parkinson’s disease from the same Springer series (Braak and Del Tredici 2009), the present effort approaches and interprets the pathological process in AD chiefly from a neuroanatomical perspective. However, we want to make the text readable for non-experts, inter alia by including throughout it both introductory and more detailed explanations pertaining to important anatomical relationships that facilitate understanding the material but that are not available in standard textbooks or only cursorily explained therein, e.g., the anatomy of the entorhinal region.

Clinically, AD only occurs in humans, and the hallmark lesions underlying the disease process predominantly are found in the human CNS. Thus, there are no truly adequate animal models for AD (Rapoport and Nelson 2011), although the implications of this reality are largely overlooked in much current research. For the past 25 years, an amyloidocentric understanding of AD research has largely ignored opposing data and arguments, thereby leaving aside important questions that still require answers (Maarouf et al. 2010). The authors focus on fundamental aspects of the AD process as a whole with the intention of encouraging alternatives to the Aβ-centered understanding of AD.

As indicated by its title, this book deals mainly with morphologically recognizable deviations from the normal anatomical condition of the human CNS. The
AD-associated pathology is illustrated from its beginnings (sometimes even in childhood) until its final form that is reached late in life. The AD process commences much earlier than the clinically recognizable phase of the disorder and its timeline includes an unusually extended non-symptomatic phase. The further the pendulum swings away from the symptomatic final stages towards the early pathology, the more obvious the lesions become, although from a standpoint of severity they are more unremarkable and, thus, frequently overlooked during routine neuropathological assessment. For this reason, we decided to deal with the hallmark lesions in early phases of the AD process in considerable detail. Clinically manifest cases of AD, on the other hand, display extensive disease-associated lesions that, as a rule, are accompanied by non-AD-related pathologies, including vascular changes and concomitant neurodegenerative disorders.

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