



# **Neuroinflammatory Mechanisms in Alzheimer's Disease: Basic and Clinical Research (Progress in Inflammation Research)**

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Research into inflammatory mechanisms that may cause damage to the Alzheimer's disease (AD) brain has now been ongoing for nearly two decades. Some two dozen clinical studies have strongly suggested that conventional anti-inflammatory drugs may be useful to delay the onset or slow the progression of the disorder. Moreover, virtually all the major systems of the innate immune response appear to be present, and most are upregulated, in pathologically-vulnerable regions of the AD brain. These new findings are described in this volume - first in overview form, followed by chapters on topics of special interest. In many ways, to understand AD brain inflammation, one need only review a text on peripheral inflammation biology, leaving out the chapters on humoral mediators and substituting microglia for macrophages. In several other key respects, however, AD brain inflammation is unique, due primarily to idiosyncratic interactions of inflammatory mediators and mechanisms with classical AD pathology: amyloid  $\beta$  peptide ( $A\beta$ ) deposits and neurofibrillary tangles (NFTs). For this reason, some key concepts about the inflammation that occurs in AD may warrant discussion in preparation for the more detailed chapters that follow.

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