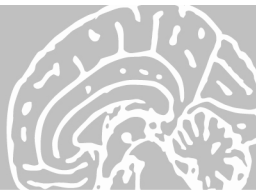
Alzheimer's Disease: an Update on Pathophysiology and Outlook for Treatment



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An unwanted result of the aging population worldwide is the rise in prevalence of age-associated neurological diseases, such as Alzheimer's disease (AD) in particular. Despite decades of intense research, therapeutics for AD remain inadequate and limited to symptomatic treatment with limited effectiveness. However, data from many laboratories around the world support the concept that accumulation of amyloid β-protein (Aβ) in brain due to increased production or reduced clearance represents an early and possibly seminal role in AD pathogenesis. Like many diseases with both sporadic and genetic forms, studies on the pathophysiology of AD have benefited tremendously from the rare inherited forms of AD that point to the enzyme (presenilin) that generates Aβ or the substrate for presenilin (amyloid precursor protein, APP) as key molecules involved in AD. Although many other cellular perturbations contribute to AD pathogenesis, misregulation of Aβ homeostasis likely plays an obligatory role in AD pathogenesis. In this talk, I will summarize recent advances in AD research to update the audience on the pathophysiology of AD and secondarily on diagnosis and treatment opportunities.