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Amyloid-beta regulation in Alzheimer's disease

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Abstract

The amyloid-beta (Aβ) peptide remains an intensely studied factor in Alzheimer's disease (AD) as genetic studies continue to implicate this peptide in disease pathogenesis. Aβ is produced by the proteolytic processing of the amyloid-β precursor protein (APP) by beta secretase and presenilin associated gamma secretase activities. All known familial-AD causing mutations are in APP as well as the presenilins and increase the production, accumulation, or aggregation of Aβ. In the more common sporadic form of AD, clinical evidence implicates reduced clearance of Aβ rather than increased production in disease pathogenesis. Neprilysin (NEP) has been shown to be a critical Aβ-degrading enzyme; however, evidence suggests that other "NEP-like" enzymes are also contributing to clearance of Aβ. We have shown that the NEP homolog NEP2 can degrade Aβ and is important for the control of Aβ levels in vivo. We have also found that NEP2 expression and activity may be dysregulated early in AD pathogenesis. This work has implications for the development of molecular markers of AD and for the development of NEP2 augmentation for AD therapy. Also, for therapy, the induction of reduced Aβ production is an obvious therapeutic objective. Recently we have shown that stimulation of the Reelin pathway through F-spondin gene transfer can reduce Aβ levels and ameliorate AD-like pathology in mice.

Biography

Dr. Marr completed his Ph.D. at the age of 29 years from McMaster University and went on to do his postdoctoral studies at the Salk Institute for Biological Studies. He has been an Assistant Professor of Neuroscience at Rosalind Franklin University of Medicine and Science since 2005. He has published 30 papers in reputed journals and has received funding from the Alzheimer's Association, Alzheimer's Drug Discovery Foundation, and the Schweppe Foundation.