Drug interaction between fibrillar amyloid β and α7 nicotinic receptor in Alzheimer brain

Ruiqing Ni1, Amelia Marutle1, and Agneta Nordberg1,2
1Karolinska Institutet, Alzheimer Neurobiology Center; 2Department of Geriatric Medicine, Karolinska University Hospital Huddinge, Sweden

Introduction
Alzheimer’s disease (AD) is the most common neurodegenerative disease. The accumulation of amyloid β (Aβ) in the brain is one of the hallmarks of AD. Emerging evidence suggested a link between amyloid pathology and nicotinic acetylcholine receptors (nAChRs), which plays an important role in mediating cognitive and neuroprotective function. The neurotoxicity of Aβ has been suggested mediated in part through an interaction between Aβ and α7 nAChRs. The amyloid positron emission tomography tracer PIB that binds selectively to fibrillar Aβ provides opportunity for visualizing amyloid deposition in living patients and for understanding the pathological mechanism in autopsy brain.

Aims
To examine how different cholinergic and anti-amyloid drugs influence 3H-PIB binding to fibrillar Aβ in autopsy AD and control brains.

Methods:
In vitro saturation and competition assays with amyloid tracer 3H-PIB (0.5 - 200nM) on AD autopsy brain.
3H-PIB binding assays in the presence of investigating drugs in frontal cortical AD (n=5, age 69.6 ± 4.2 years) and control brain (n=5, age 67.4 ± 3.9 years).

Results:
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