Annals of General Hospital Psychiatry

BioMed Central

Oral presentation

Open Access

Recent advances in the pharmacotherapy of Alzheimer's disease

G Mentenopoulos*

Address: Professor of Neurology, 2nd University Department of Neurology, Aristotle University of Thessaloniki, Greece
* Corresponding author

from International Society on Brain and Behaviour: 1st International Congress on Brain and Behaviour
Hyatt Regency Hotel, Thessaloniki, Greece, 20–23 November, 2003

Published: 23 December 2003 Received: 1 November 2003

Annals of General Hospital Psychiatry 2003, **2**(Suppl 1):S22

This article is available from: http://www.general-hospital-psychiatry.com/content/2/S1/S22

Most drug research in AD has been directed at providing symptomatic cognitive enhancement through a variety of neurotransmitter manipulations. Cholinergic neurotransmission has been most frequently targeted in drug development for a variety of reasons. There is a well characterized degeneration of the acetylcholine (Ach) synthesizing neurons in the basal forebrain region and their cortical synaptic connections in AD. The severity of AD has been correlated to the degree of decline of choline acetyltransferase (CAT) the key synthetic enzyme of Ach. Impairment of number and function of muscarinic and nicotinic receptors have been implicated in the cognitive impairment of AD. It has been the interventions directed at the inhibition of cholinesterases (ChE) that have been the most successful to date at producing symptomatic benefit, measurably enhancing cognitive function. During recent years, treatment of cognitive impairment in AD patients has been made possible by the introduction of ChE inhibitors, such as rivastigmine, donepezil and galanthamine. The drugs improve memory, cognitive and global function in AD, probably augmenting cholinergic function. ChE inhibitors differ substantially in their pharmacological properties which may translate into differences in clinical efficacy, safety and tolerability. Galanthamine show a dual mode of action by the allosteric modulation, with clinically meaningful improvement on cognition in AD, Lewy body dementia, Parkinson's disease dementia and vascular dementia. Rivastigmine has demonstrated a strong action as a dual AChE and BuChE inhibitor. Recent reports focus on the role of Butirylcholinesterase (BuChE) in AD. The activity of this enzyme increases during the progression of the disease while AChE decreases. Inhibition of both enzymes may have more prolonged clinical results. Recent findings indicate that both AChE and BuChE in addition to their role in neurotransmission may play a role in the formation of amyloid plaques. It is found that the activity of BuChE substantially increases in the affected areas of the brain. The reason for this increase is under research. It is believed that inhibition of AChE and BuChE can delay deterioration in AD patients. In addition to the cognitive and functional symptoms treatment with ChE inhibition may also improve the behavioural and psychiatric symptoms of AD and other dementias. Agitation, irritability, anxiety and depression may improve. Dual ChE inhibitors of AchE and BuChE can also improve delusions and hallucinations. This is very important especially in Levy body where antipsychotics can cause serious adverse events.