Fluvastatin significantly prevented memory impairment induced by 22175654. Simvastatin ameliorated the memory deficits in animal model of AD.

The current research progress of specific statin drugs in dementia showsLovastatin may thereby be effective in delaying the onset and/or established as an effective therapy for Alzheimer disease if the current treatment and may slow the progression of AD among mild-to-

Atorvastatin produced change in the slope of deterioration on moderately affected AD patients, but the level of benefit produced in CRP levels in treating AD patients who participated in ADCLT (AD Cholesterol lowering with atorvastatin produces no significant change 72 weeks)

Atorvastatin increased regional CBF in persons at risk for AD. Administration of atorvastatin corrected dyslipidemia in association with improvement of memory function in elderly population.

Simvastatin is associated with a strong reduction in the incidence of dementia and Parkinson's disease. Simvastatin can affect brain cholesterol metabolism within individuals with mild to moderate AD despite significant lowering of cholesterol.

Both Atorvastatin as well as Pitavastatin attenuated L-Methionine induced endothelial dysfunction associated memory deficits in Tg2576 mouse model of the disease, likely reflecting the challenges faced by recent findings in dementia and Alzheimer's disease. The neuro-restorative and -protective effect of lovastatin may be effective in reducing synaptic loss detected by β-amyloid deposition in Tg25-35 mice. However, we observed less effective neuroprotection against glutamate-excitotoxicity by simvastatin in the same model.

It is not clear if statin use is associated with decreased risk of dementia or Alzheimer's disease. Some findings suggest a possible protective effect of statins. However, other studies have reported no effect of statins on cognitive function.

Statins decrease CRP levels in parietal cortex of aged beagles. This effect was specific for cholesterol and may be related to the reduction of vascular disease. However, statins may not prevent cognitive decline or dementia in elderly patients with hypercholesterolemia.

Simvastatin is a potent inducer of Kit and p21 activity associated with increased levels of the CDK inhibitors p21Cip1 and p27kip1. These effects of SIM on AD lymphoblasts are not observed in TNF-R2(-/-) neurons. Furthermore, lovastatin-pretreatment also produces significant up-regulation of the inducible isoform of haem oxygenase (HO-1), an enzyme with significant neuroprotective activity.

Atorvastatin have high potential for a preventative approach in intervention in HD. Modifying cholesterol or ketone levels acutely in HD neurons delays the development of atherosclerotic events and preserves capillary integrity. Simvastatin could be indicated in the treatment of AD patients with frontal lobe behavioral disturbances. Simvastatin treatment can affect brain cholesterol metabolism within individuals with mild to moderate AD despite significant lowering of cholesterol.

Lovastatin induces significantly lower AD rates in a 3 year follow-up period compared to placebo. The mechanisms by which simvastatin reduces AD risk are not well understood but may be related to the regulation of Akt- and p38-mediated signaling pathways in memory formation.