jects with and without the use of spatial normalization and template-based ROI definition. **Methods:** MTL regions of 11 healthy subjects were scanned using BOLD fMRI in a Siemens Magnetom Trio 3T with a GE-EPI sequence optimized for the MTL structures (TR=2000ms, TE=30ms, 64x64 matrix) [8]. The activation paradigm [6] involved blocks of trials during which the subjects' task was either to encode objects (OE) or positions of objects (PE). BOLD contrast between OE and PE conditions was computed using SPM2 (http://www.fil.ion.ucl.ac.uk/spm). PrC boundaries were determined using Insausti [9] criteria. **Conclusions:** As reported in [6] significant activation occurred in PrC (using $p<0.05$, FDRcorr). Here we show a) that individuals exhibited different patterns of PrC activity; and b) spatial normalization resulted in the displacement of some Prc activations into other regions in the normalized space. The group-derived activation peak was distant from activation sites observed in many individuals. Spatial variability of individual activations is displayed using corner-cube visualization [10]. Use of current spatial normalization techniques to define specific MTL regions for evaluation with fMRI probes may lead to significant loss of sensitivity.

P-103 IMAGING NEUROINFLAMMATION IN ALZHEIMER DISEASE WITH [1-11C]ARACHIDONIC ACID AND POSITRON EMISSION TOMOGRAPHY

Stanley I. Rapoport[1], Richard E. Carson[2], Abesh Bhattacharjee[1], Mark B. Schapiro[3], Peter Herscovitch[2], William Eckelman[2], Giuseppe Esposito[4]; *[1]NIA, NIH, Bethesda, MD, USA; [2]CC, NIH, Bethesda, MD, USA; [3]School of Medicine, University of Cincinnati, Cincinnati, OH, USA; [4]Washington Hospital Center, Washington, DC, USA*

Background: Arachidonic acid (AA), a component of membrane phospholipids, is released by phospholipase A2 (PLA2) activation. Neuroinflammation, which is considered to contribute to AD, can increase PLA2 activity and AA turnover in brain phospholipids; this process has been imaged in unanesthetized rats (Lee, JNeurochem, 91, 936, 2004). **Objectives:** To image neuroinflammation in AD patients. **Methods:** Regional brain AA incorporation coefficients (K*) were measured with [1-11C]AA and positron emission tomography (PET) (Giovacchini, JCBFMetab, 22, 1453, 2002) in 8 moderately-severely demented AD patients and 7 age-matched controls; regional cerebral blood flow (rCBF) was measured with 15O-water. PET images were corrected for partial voluming from MRI scans. Data were normalized to a global mean, which was independently determined. SPM2 was used to test for group differences. Results are at $p < 0.001$. **Results:** Compared to controls, rCBF reductions of ~21% were in temporal (BA 37, 20 and 22) and 20-40% in parietal (BA 39 and 7) association cortex bilaterally of AD patients. AD patients had increased normalized K* for AA of ~15% in temporal association cortex bilaterally (BA 21 and 22), ~12% in the right parietal lobule (BA 39), and 10-14% bilaterally in orbitofrontal cortex (BA 11 and 47). Between group differences were greater with absolute K* and rCBF values. Compared to controls, AD patients had 35% decreased mean global CBF and 23% increased global K*. **Conclusions:** AA incorporation is increased in AD compared with control brainn, globally and regional in brain areas demonstrating inflammatory neuropathology on postmortem; rCBF redudctions in these areas are consistent with prior evidence of reduced neuronal activity. As animals studies indicate that K* increases represent neuroinflammation involving increased PLA2 activity and AA turnover (Rosenberger, JNeurochem, 88, 1168, 2004), our findings suggest that neuroinflammation can be imaged in vivo in AD patients. PET imaging of AA incorporation might be used as a surrogate marker of AD neuroinflammation, for early diagnosis and evaluation of disease progression.

P-104 MEDIAL TEMPORAL LOBE ATROPHY AND WHITE MATTER CHANGES ARE ASSOCIATED WITH MILD COGNITIVE DEFICITS IN NON-DISABLED ELDERLY

Wiesje M. van der Flier[1], Ilse van Straaten[1], Frederik Barkhof[1], Jose Ferro[2], Leonardo Pantoni[3], Timo Erkinjuntti[4], Lars-Olof Wahlund[5], Egill Rostrup[6], Franz Fazekas[7], Philip Scheltens[1]; *[1]VU Medical Center, Amsterdam, The Netherlands; [2]Hospital de Santa Maria, Lisboa, Portugal; [3]University of Florence, Florence, Italy; [4]Helsinki University, Helsinki, Finland; [5]Huddinge University Hospital, Huddinge, Sweden; [6]Copenhagen University Hospital, Copenhagen, Denmark; [7]Medical University, Graz, Austria*

Background: On MRI, medial temporal lobe atrophy (MTA) presumably reflects Alzheimer-type pathology, whereas white matter changes (WMC) are likely to be of vascular origin. MTA and WMC often coexist in dementia. However, the role of the combination of these MRI abnormalities in the early stages of cognitive decline is not known. **Objective(s):** To assess the associations between MTA and WMC and cognitive function in a large group of independently functioning elderly. **Methods:** Data were drawn from the multicentre, multinational Leukoaraiosis and Disability (LADIS) project that prospectively studies the role of WMC as an independent predictor of the transition to disability in non-disabled elderly. In total, 639 participants were enrolled in the LADIS study. For the present analysis, data of 581 subjects were available. Cognitive function was assessed by the Mini Mental State Examination (MMSE). The MR protocol included coronal T1-weighted MPRAGE, and axial T2 and FLAIR images. Visual ratings of WMC and MTA were performed. Relative risks of mild cognitive deficits (MMSE < 26) depending on MTA and/or WMC were estimated as odds ratios (OR) and their 95% confidence interval. **Results:** The presence of either severe WMC or MTA was associated with a modest, but non-significant increase in frequency of mild cognitive deficits (severe WMC: OR = 1.9; 95% CI: 1.0 - 3.7, MTA present: OR = 1.5; 95% CI: 0.8 - 2.8). However, subjects with the combination of MTA and severe WMC had a more than fourfold increase in frequency of mild cognitive deficits (OR = 4.1; 95% CI: 2.3 - 7.4). ANOVA with post hoc Bonferroni t-tests revealed that subjects with both MTA and severe WMC performed worse on MMSE than subjects with neither MRI abnormality or one single MRI abnormality ($p < 0.05$). **Conclusions:** These results provide further evidence for the combined involvement of both Alzheimer-type pathology and vascular pathology in the earliest stages of cognitive decline and suggest an additive effect of WMC and MTA. These data may provide clues for prevention of cognitive decline in the elderly.

P-105 AMYLOID IMAGING IN ALZHEIMER'S DISEASE AND DEMENTIA WITH LEWY BODIES WITH 11C-PIB-PET

Victor L. Villemagne[1,2], Uwe Ackermann[1], Sylvia J. Gong[1], Kerryn E. Pike[3], Kathy E. Novakovic[1], Julie C. Price[4], Chester A. Mathis[4], William E. Klunk[5], Colin Masters[2], Christopher C. Rowe[1]; *[1]Centre for PET, Austin Hospital, Heidelberg, VIC, Australia; [2]Department of Pathology, University of Melbourne, Melbourne, VIC, Australia; [3]Department of Psychology, Monash University, Melbourne, VIC, Australia; [4]Department of Radiology, University of Pittsburgh, Pittsburgh, PA, USA; [5]Department of Psychiatry and Neurology, University of Pittsburgh, Pittsburgh, PA, USA*

Background: Aβ amyloid plaques are one of the pathological hallmarks of Alzheimer's disease (AD) but are also present in Lewy Body Dementia (LBD). The contribution of amyloid to the development of LBD is unclear. **Objective:** The purpose of the study was to compare cortical amyloid deposition in LBD and AD patients in-vivo using the amyloid ligand Pittsburgh Compound-B (PIB, 2-(4'methylaminophenyl)-6-hydroxybenzothiazole) and PET. **Methods**: AD (n=5 with mild to moderate dementia, MMSE 15-28), LBD (n=4 with moderate to severe progressive dementia, MMSE 10-25, persistent visual hallucinations, Parkinsonism and cognitive fluctuation), and age-matched control subjects (n=5 with MMSE>27) underwent dynamic