right hemisphere is more likely to be selectively affected. Furthermore, the frequency of significant hemispheric metabolic asymmetry is unknown. **Methods:** We evaluated 95 baseline FDG-PET scans in patients with clinically diagnosed mild AD (MMSE 20-27) enrolled in the Alzheimer's Disease Neuroimaging Initiative (ADNI). Each scan was analyzed with Neurostat, providing 3D-stereotactic surface projection (3D-SSP) maps of glucose metabolism relative to pons and corresponding statistical maps of Z-scores computed in comparison to 27 cognitively normal elderly control subjects. We visually evaluated the pattern of glucose hypometabolism, considering 5 regions in each hemisphere typically affected in dementia. Hemispheric asymmetry was considered significant when hypometabolism was 2 or more standard deviations greater in one hemisphere. We also calculated 3D-SSP maps of mean rates of glucose metabolism and Z-score maps. **Results:** Ten patients had an FTD-like pattern of hypometabolism and were excluded from further analyses. In the remaining 85 AD subjects, FDG-PET images were symmetric in 70 (82.4%). Hypometabolism predominantly involved left hemisphere regions in 8 (9.4%) and predominantly involved right hemisphere regions in 7 (8.2%). These groups did not differ significantly in age or dementia severity as measured by MMSE. Impairment was greater in naming and word fluency for patients with predominant left hemisphere hypometabolism and clock drawing scores were lowest for those with predominant right hemisphere hypometabolism. **Conclusions:** Although AD patients as a group have completely symmetric glucose hypometabolism, individual patterns vary reflecting cognitive differences. In this sample, roughly similar proportions of left and right predominant hypometabolism was seen in a minority of patients. The frequency of metabolic asymmetry may differ in community samples. The mechanisms accounting for selective involvement of one hemisphere are unexplained and need to be explored further. Supported in part by the Center for Alzheimer's Care, Imaging and Research and NIH grant AG024904.

P1-245 MAPPING LOCAL STRUCTURAL HIPPOCAMPAL CHANGES IN ALZHEIMER'S DISEASE AND NORMAL AGING ON MR IMAGING AT 3T

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Background: Histological studies suggest that hippocampal subfields are differently affected by aging and Alzheimer's disease. We aimed to study local hippocampal changes in aging and moderate to severe Alzheimer's patients based on high resolution magnetic resonance images at 3 Tesla. **Methods:** 3D high resolution T1-weighted magnetic resonance (MR) were acquired on a 3.0 T scanner from 14 AD (age 75±5 years, 3 males, Mini Mental State Examination 13±4) and 14 controls (age 71±5 years 9 males, Mini-Mental State Examination 29±1). The hippocampal formation was isolated by manual tracing. Radial Atrophy Mapping was used to assess group differences and correlations by averaging hippocampal shapes across subjects using 3-dimensional parametric surface mesh models. Percentage difference, Pearson's *r*, and significance 3-dimensional maps were produced. **Results:** Hippocampal volumes were inversely correlated with age in older healthy controls (r= .53 and .56 to the right and left, p< 0,05, corresponding to 17% lower volume for every 10 years of older age from age 65). Aging-associated atrophy mapped to dorsal and lateral areas of the tail and body corresponding to the CA1 subfield and to ventral areas of the head corresponding to the subiculum. Significantly increased volume with older age mainly mapped to restricted dorsal areas of the head bilaterally corresponding to the CA1 subfield. Volumes were 37% and 30% smaller in AD patients to the right and left (p<.0005). AD-associated atrophy mapped to areas of the body and tail corresponding to those also associated with age, and dorsal areas of the head corresponding to the CA1 subfield unaffected by age. Regions corresponding to the CA2-3 fields were relatively spared in both aging and AD. **Conclusions:** Hippocampal atrophy in AD maps to areas in the body and tail partly overlapping to those affected by normal aging. Specific areas in the anterior and dorsal CA1 subfield involved in AD were not in normal aging. Such differences might relate to specific systems being involved in AD and ageing.

P1-246 WHITE MATTER HYPERINTENSITIES IN ALZHEIMER PATIENTS AND NON-DEMENTED ELDERLY: POSTMORTEM QUANTITATIVE MRI AND NEUROPATHOLOGICAL CHARACTERISTICS

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Background: The association between white matter hyperintensities (WMH) on conventional MRI and cognitive decline in Alzheimer's disease (AD) and in healthy elderly is only weak and inconsistent. A possible explanation for this would be heterogeneity in the neuropathological substrate underlying WMH. In vivo quantitative MRI techniques may improve the clinico-radiological association. We assessed whether postmortem quantitative MRI reflects differences in neuropathological correlates of WMH in AD and controls. **Methods:** Thirty-three formalin-fixed, coronal brain slices from 11 AD patients (mean age: 83 ± 10 yrs, 8 females) and 15 slices from 7 non-demented controls (mean age: 78 ± 10 yrs, 4 females) with WMH were scanned with qualitative (FLAIR) and quantitative MRI (diffusion tensor imaging [DTI] with b = 750 s/mm2, and T1-relaxation time mapping based on flip-angle array) using a 1.5T Siemens Vision scanner. 104 Regions of interest (ROIs) were defined on FLAIR images in WMH and normal appearing white matter (NAWM; 45 WMH and 30 NAWM ROIs in AD; 16 WMH and 13 NAWM ROIs in controls). Histopathological examination included (semi)-quantitative assessment of axonal density (Bodian), myelin density (LFB), astrogliosis (GFAP), microglial activation (LN3). **Results:** Overall, AD patients had a lower fractional anisotropy (FA) and a higher T1 than controls. WMH had lower FA and higher T1 values than NAWM in both groups. More specifically, WMH of AD patients differed from WMH of controls as it had lower FA and higher T1 values (unpaired T-test: mean FA = 0.44 ± 0.10 vs 0.57 ± 0.07, p= 0.001 and mean T1[ms] = 464 ± 83 vs 398 ± 98, p= 0.01; for AD vs controls). Within the group of AD patients, lower FA and higher T1 correlated with axonal and myelin loss and more microglial activation (Spearman's r: FA= -0.60, -0.41, -0.27 and T1= 0.61, 0.49, 0.38 for axonal density, myelin density and microglial activation, respectively; all p<0.001, except for the correlation between FA and microglial activation: p<0.05). **Conclusions:** Postmortem quantitative MRI (DTI, T1-mapping) reveals differences between WMH in AD and in non-demented elderly. Moreover, postmortem quantitative MRI reflects the severity of axonal and myelin loss and microglial activation.

P1-247 CLINICAL SEVER… IS ASS…

WITHDRAWN

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