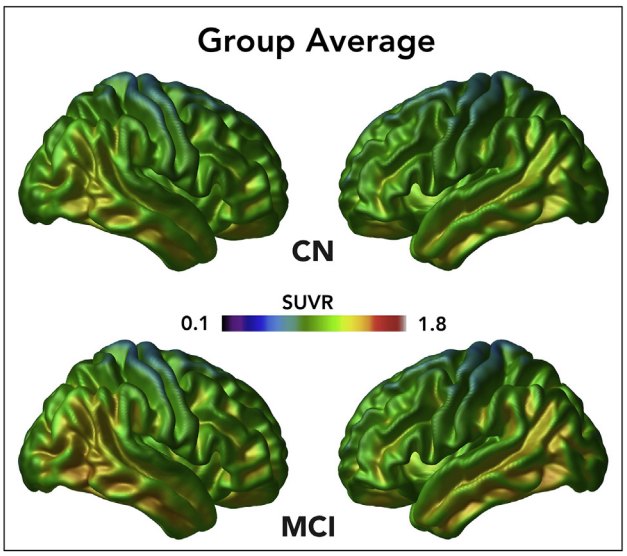
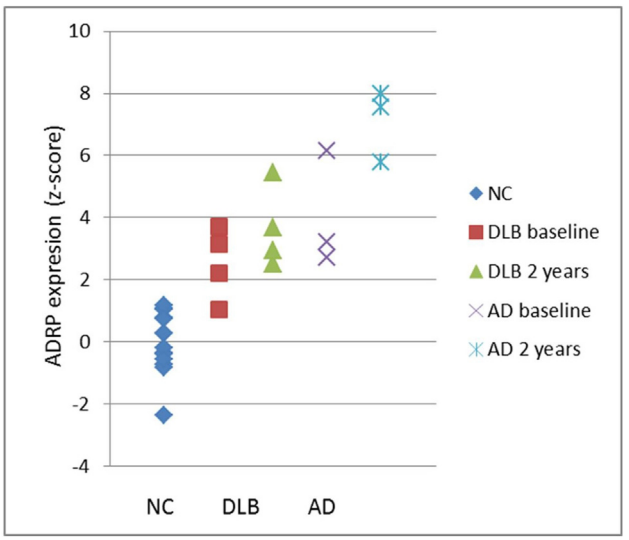
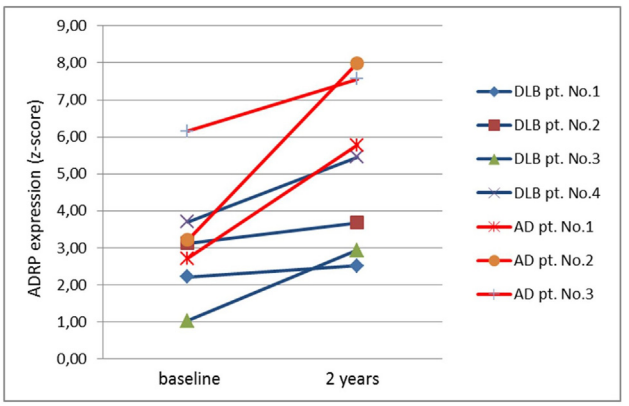




length was significantly lower in MCI when compared to the CN network, resulting in a significantly higher global efficiency. The clustering coefficient of the MCI network was significantly higher than the CN network. Lastly, the small worldness parameter for the MCI network was significantly lower compared to the CN [Figure 2]. **Conclusions:** Deposition of Tau proteins in the MCI [Figure 3] stage involves an increased number of long range and short range network connections (redundancy) [Figure 1] indicated by the increased density, clustering coefficient, and decreased average path length. Nonetheless, the small world property decreases mainly due to the increase in long range connections in the MCI group. These results indicate that in the CN stage, local Tau deposition patterns are confined within the local structural boundaries and as the disease progresses, deposition patterns expand beyond their structural boundaries.



IC-P-035

MONITORING THE PROGRESSION OF DEMENTIA USING FDG-PET BRAIN IMAGING AND NETWORK ANALYSIS: ROLE OF THE ALZHEIMER'S DISEASE-RELATED PATTERN

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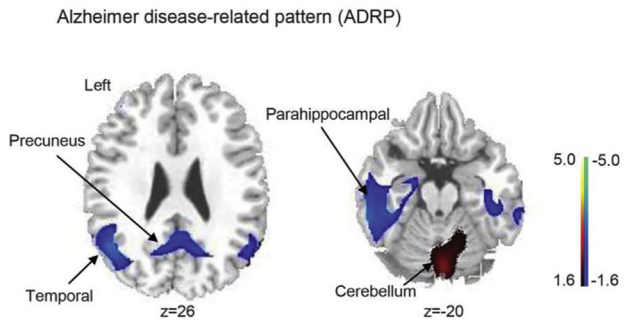
Background: Alzheimer's dementia (AD) and dementia with Lewy bodies (DLB) are most common dementias in elderly and share some pathological characteristics. Clinical differentiation between them is often challenging in early stages. Characteristic patterns of brain glucose metabolism may help differentiating between neurodegenerative dementias (Marcus C, 2014) and provide a tool to follow up disease progression. A characteristic metabolic brain pattern of AD has been identified using FDG-PET brain imaging and specific network analysis - the so called *Alzheimer's disease related pattern* (ADRP, Figure 1). It has been shown to be a reproducible and stable biomarker of AD (Mattis PJ, 2016). The aim of this pilot study was to analyse the expression of ADRP in AD and DLB patients at baseline and after the follow up period of two years. **Methods:** Seven demented patients (three AD and four DLB, Table 1) and 14 age-matched healthy controls (HC) underwent FDG-PET scan, patients were scanned twice in two years. Four patients underwent CSF amyloid analysis. NEUROSTAT/3D-SSP was performed to confirm the clinical diagnosis of AD and DLB and observe progression. ADRP expression was calculated in these 14 images of demented patients and in HC scans using ScanVP software (www.feinsteinneuroscience.org). **Results:** Mean age of DLB and AD patients was 80.2 and 72.2 years, respectively (p=0.12). Time between two scans was 2.1±0.7 years. We confirmed the diagnosis and disease progression using NEUROSTAT/3D-SSP



	Age (years)	Gender	Clinical diagnosis	Diagnosis based on NEUROSTAT analysis	Disease duration at first scan (years)	Time between the scans (months)	CSF ß-amiloid (pg/ml)
DLB pt. No.1	76	F	DLB	DLB	4	38	492
DLB pt. No.2	74	M	DLB	DLB	4	44	819
DLB pt. No.3	85	F	DLB	DLB	1	12	/
DLB pt. No.4	86	F	DLB	DLB	2	44	401
AD pt. No.1	75	F	AD	AD	1	35	/
AD pt. No.2	74	M	AD	AD	1	65	470
AD pt. No.3	67	M	AD	AD	1	33	/

analysis in all patients. ADRP expression was higher in patients compared to HC at baseline (AD vs. HC p=0.057, DLB vs. HC p=0.015, Figure 2). Expression values significantly increased in all individual AD and DLB patients between baseline and two years (p=0.0149, paired t-test; Figure 3), reaching relatively higher levels in AD than DLB patients at two years (p=0.015). Additionally, more pronounced progression was observed in AD patients compared to DLB patients over two years of follow up. **Conclusions:** ADRP is a specific metabolic brain pattern characteristic for AD. It may be used as an imaging biomarker of disease progression for demented patients with AD. ADRP expression may also be useful to help differentiate demented patients from HC as well as AD from DLB patients.

IC-P-036 CORRELATION OF GREY MATTER NETWORK MEASURES IN COGNITIVELY HEALTHY ELDERLY MONOZYGOTIC TWIN PAIRS

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Background: Grey matter connectivity is disrupted in Alzheimer's disease (AD), and has been associated with amyloid pathology and cognitive decline in non-demented subjects. The contribution of genetic and environmental factors to grey matter connectivity is currently unknown. To estimate the upper limit of genetic contribution to grey matter connectivity, we examined the similarity of this measure in cognitively healthy elderly monozygotic twin pairs. **Methods:** Monozygotic twin pairs were selected from the EMIF-AD PreclinAD study. Inclusion criteria were age $\geq$ 60 years and having a delayed recall score less than 1.5 SD of normative data. Single-subject grey matter networks were constructed from structural MRI. Small regions of interest were connected when they showed statistical similarity in cortical grey matter structure. We calculated network size, degree, connectivity density, betweenness centrality, normalized clustering coefficient and normalized path length. Monozygotic twin pair correlations were assessed for each connectivity measure after adjusting for age, gender and total grey matter volume. Since monozygotic twins are genetically identical, within pair correlations reflect the upper limit of genetic contribution to a trait. Correlations analyses were repeated for random (non-twin) pairings of subjects. **Results:** We included 96 monozygotic twin pairs (n = 192 subjects) (age = 70.2 ± 7.3 years; 112 (58%) females; MMSE = 29.0 ± 1.1). All grey matter network properties were correlated in monozygotic twin pairs (figure 1). The highest correlation was found for network size (0.82, p<0.001) and lowest for connectivity density (0.45, p<0.001). The other correlations were: degree (0.53, p<0.001), betweenness centrality (0.79, p<0.001), normalized clustering coefficient (0.68, p<0.001), normalized path length (0.76, p<0.001). Estimating the correlation in non-twin pairs did not yield any significant results. **Conclusions:** In cognitively healthy elderly monozygotic twins, we found significant within pair correlations of grey matter connectivity. These data suggest that in addition to a moderate-strong genetic background for grey matter connectivity, also non-genetic factors substantially influence this trait. Future studies will focus on identifying environmental risk factors for grey matter connectivity disruptions and determinants of twin discordance. This work has received support from the EU/EFPIA Innovative Medicines Initiative Joint Undertaking EMIF grant agreement n°115372.

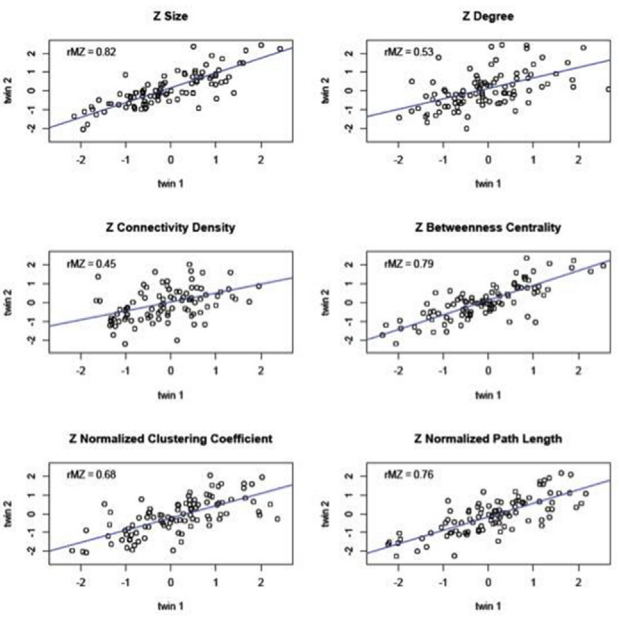

Figure 1. Correlation of grey matter connectivity measures in monozygotic twins. Displayed are standardized residuals after correcting for age, gender, total grey matter volume. Betweenness centrality, normalized clustering coefficient and normalized path length were additionally corrected for connectivity density. rMZ = Pearson correlation within monozygotic twin pairs.