brain changes, deficits prompted by mTBI, and the extent to which mTBI may increase AD risk.

IC-P-033 LONGITUDINAL INTRINSIC FUNCTIONAL CONNECTIVITY CHANGES IN PRECLINICAL ALZHEIMER'S DISEASE: A TWO-YEAR FOLLOW UP STUDY

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Background: Although previous cross sectional studies demonstrated intrinsic functional connectivity (FC) changes in the preclinical Alzheimer's disease (AD), effects of amyloid burden in FC changes are still not yet clear on the longitudinal basis **Methods:** Sixty four florbetaben (FBB) amyloid PET defined NIA-AA preclinical AD subjects (stage 1 (N= 31) and stage2/3 (N=33)) and 32 healthy control subjects (stage 0) completed resting state functional magnetic resonance imaging (fMRI) scans. FC changes were examined for three networks of interest (default mode network (DMN), salience network (SN), and central executive network (CEN)) using independent component analysis during a 2-year follow-up. We also analyzed the group by amyloid retention with FC changes in the three networks. **Results:** Longitudinal analysis showed that the DMN of the stage 1 subjects showed significantly increased FC compared to the stage 0 and 2/3 subjects. On the other hand, stage 2/3 subjects showed decreased FC compared to stage 0 and 1 subjects. The FC of the CEN showed significantly decreased in the stage 1 and increased in stage 2/3 during the follow up periods. There were no longitudinal FC changes in SN in all subjects. In addition, there were significant group x FBB amyloid retention level interaction with FC changes in the posterior DMN. **Conclusions:** Our results of aberrant DMN FC changes and distinctive interaction patterns might reflect a biphasic trajectory of changes in FC in preclinical AD subjects. These changes might have clinical implications as surrogate markers of efficacy in clinical trials of the disease modifying agents.

IC-P-034 GRAPH-THEORY ANALYSIS SHOWS A HIGHLY EFFICIENT BUT REDUNDANT NETWORK IN MCI TAU PROPAGATION

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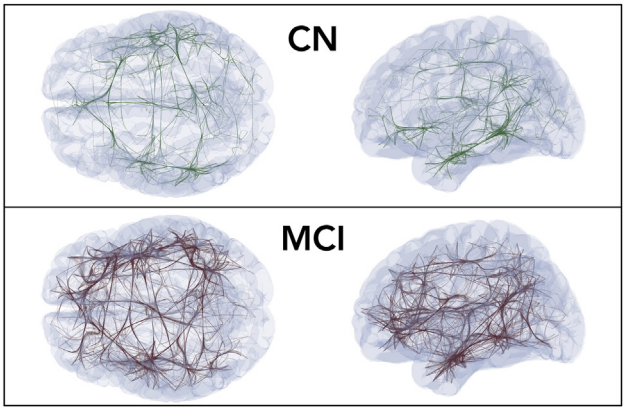
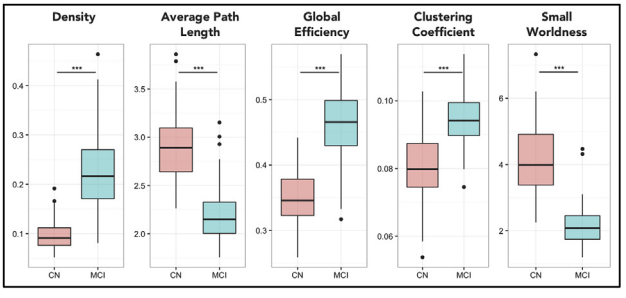
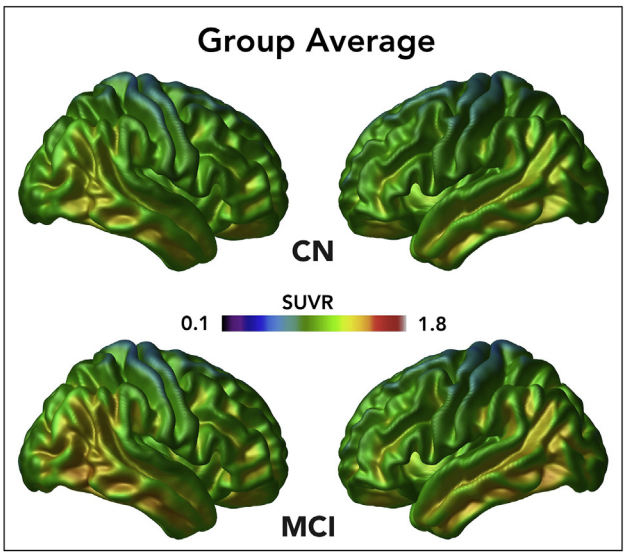
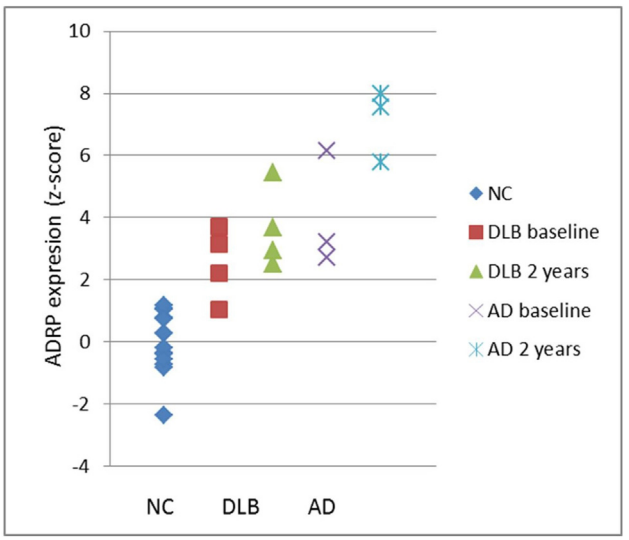
Background: The stages of neurofibrillary tangles (NFT) during the course of Alzheimer's disease is well understood through the Braak stages; however, the underlying mechanism behind the NFT propagation remains unclear. Here, we propose a graph theory based method to identify the patterns of NFT deposit propagation using [^{18}F]AV1451 Tau positron emission tomography (PET) images. **Methods:** [^{18}F]AV1451 images of 38 cognitively normal (CN) and 34 mild cognitive impairment (MCI) individuals were acquired from the ADNI cohort and the standardized uptake value ratio (SUVr) maps were generated using the cerebellar grey matter as the reference region. Group-based Tau networks of CN and MCI were then constructed from 201 nodes distributed across the cerebral cortex and using Pearson correlation coefficients based on 100 bootstrap samples. The networks were corrected for multiple comparisons using False Discovery Rate (FDR) and thresholded at r >= 0.5. Network measures such as density, average path length, global efficiency, clustering coefficient, and small worldness were calculated for each of the bootstrap sample. Welch two sample t-test was used to compare each measure across both subject groups. **Results:** The density of the MCI Tau network was significantly higher compared to CN [Figure 1]. Furthermore, the average path

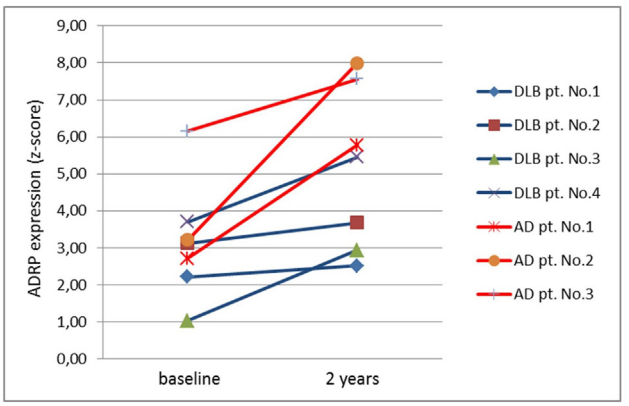








length was significantly lower in MCI when compared to the CN network, resulting in a significantly higher global efficiency. The clustering coefficient of the MCI network was significantly higher than the CN network. Lastly, the small worldness parameter for the MCI network was significantly lower compared 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