

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**



Hyun Kook Lim¹, Chang Uk Lee², Dong Woo Kang³, Yoo Hyun Um⁴, Changtae Hahn⁵, Soo hyun Joo¹, ¹The Catholic University of Korea, Seoul, Republic of South Korea; ²Seoul St. Mary's Hospital, Catholic Medical College, The Catholic University of Korea, Seoul, Republic of South Korea; ³Seoul Saint Mary's Hospital, Seoul, Republic of South Korea; ⁴St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Suwon, Republic of South Korea; ⁵College of Medicine, The Catholic University of Korea, Seoul, Republic of South Korea. Contact e-mail: drblues@catholic.ac.kr

Background: Although previous cross sectional studies demonstrated intrinsic functional connectivity (FC) changes in the pre-clinical 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 stage 2/3 (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**



Sulantha S. Mathotaarachchi^{1,2,3}, Tharick A. Pascoal^{1,2,3,4,5}, Monica Shin^{2,3,6}, Andrea Lessa Benedet³, Min-Su Kang⁷, Hanne Struyfs^{3,8,9,10}, Kok Pin Ng^{2,3,11}, Joseph Therriault³, Vladimir S. Fonov¹², Serge Gauthier^{2,4}, Mistic Bratislav¹³, Pedro Rosa-Neto^{1,2,3,5,14,15}, ¹McGill University, Montreal, QC, Canada; ²McGill University Research Centre for Studies in Aging, Verdun, QC, Canada; ³Translational Neuroimaging Laboratory-McGill University, Verdun, QC, Canada; ⁴Douglas Hospital Research Centre, Verdun, QC, Canada; ⁵Centre for Studies on Prevention of Alzheimer's Disease (StoP-AD Centre), Douglas Mental Health Institute, Verdun, QC, Canada; ⁶Cerebral Imaging Centre - Douglas Research Centre, Verdun, QC, Canada; ⁷Translational Neuroimaging Laboratory, McGill University Research Centre for Studies in Aging- McGill University, Montreal,

QC, Canada; ⁸University of Antwerp, Antwerp, Belgium; ⁹icomatrix, Leuven, Belgium; ¹⁰Reference Center for Biological Markers of Dementia (BIODEM), Laboratory of Neurochemistry and Behavior, Institute Born-Bunge, University of Antwerp, Antwerp, Belgium; ¹¹National Neuroscience Institute, Singapore, Singapore; ¹²Montreal Neurological Institute, McGill University, Montreal, QC, Canada; ¹³Montreal Neurological Institute, Montreal, QC, Canada; ¹⁴Douglas Mental Health University Institute, Montreal, QC, Canada; ¹⁵McCormell Brain Imaging Centre - McGill University, Montréal, QC, Canada. Contact e-mail: sulantha.s@gmail.com

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 [¹⁸F]AV1451 Tau positron emission tomography (PET) images. **Methods:** [¹⁸F]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 (SUV_r) 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 $r \geq 0.5$. 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

