Axonal density in the right uncinate fasciculus negatively correlated with both CCI-12 and CCI-TOT (20 item) ($p<0.001$, Figure 2). Other ROIs had no significant correlations. **Conclusions:** These results suggest that lower axonal density and fiber coherence are risk factors for self-perceived memory decline. The two most vulnerable white matter tracts - the right stria terminalis and uncinate fasciculus, connect between the amygdala and hippocampus - two of the areas that show the earliest disease-associated changes. References: 1. Villemagne, V.L., S. Burnham, P. Bourgeat, B. Brown, K.A. Ellis, O. Salvado, C. Szoeke, S.L. Macaulay, R. Martins, P. Maruff, D. Ames, C.C. Rowe, C.L. Masters, B. Australian Imaging, and G. Lifestyle Research, Amyloid beta deposition, neurodegeneration, and cognitive decline in sporadic Alzheimer's disease: a prospective cohort study. Lancet Neurol, 2013. 12(4): p. 357-67. 2. Jack, C.R., Jr., V.J. Lowe, S.D. Weigand, HJ. Wiste, M.L. Senjem, D.S. Knopman, M.M. Shiung, J.L. Gunter, B.F. Boeve, B.J. Kemp, M. Weiner, R.C. Petersen, and I. Alzheimer's Disease Neuroimaging, Serial PIB and MRI in normal, mild cognitive impairment and Alzheimer's disease: implications for sequence of pathological events in Alzheimer's disease. Brain, 2009. 132(Pt 5): p. 1355-65. 3. Sperling, R.A., P.S. Aisen, L.A. Beckett, D.A. Bennett, S. Craft, A.M. Fagan, T. Iwatsubo, C.R. Jack, Jr., J. Kaye, T.J. Montine, D.C. Park, E.M. Reiman, C.C. Rowe, E. Siemers, Y. Stern, K. Yaffe, M.C. Carrillo, B. Thies, M. Morrison-Bogorad, M.V. Wagster, and C.H. Phelps, Toward defining the preclinical stages of Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimers Dement, 2011. 7(3): p. 280-92. 4. Saykin, A.J., H.A. Wishart, L.A. Rabin, R.B. Santulli, L.A. Flashman, J.D. West, T.L. McHugh, and A.C. Mamourian, Older adults with cognitive complaints show brain atrophy similar to that of amnestic MCI. Neurology, 2006. 67(5): p. 834-42. 5. Rattanabannakit, C., S.L. Risacher, S. Gao, K.A. Lane, S.A. Brown, B.C. McDonald, F.W. Unverzagt, L.G. Apostolova, A.J. Saykin, and M.R. Farlow, The Cognitive Change Index as a Measure of Self and Informant Perception of Cognitive Decline: Relation to Neuropsychological Tests. J Alzheimers Dis, 2016. 51(4): p. 1145-55. 6. Zhang, H., T. Schneider, C.A. Wheeler-Kingshott, and D.C. Alexander, NODDI: practical in vivo neurite orientation dispersion and density imaging of the human brain. Neuroimage, 2012. 61(4): p. 1000-16. 7. Wu, Y.C. and A.L. Alexander, Hybrid diffusion imaging. Neuroimage, 2007. 36(3): p. 617-29. 8. Kodiweera, C., A.L. Alexander, J. Harezlak, T.W. McAllister, and Y.C. Wu, Age effects and sex differences in human brain white matter of young to middle-aged adults: A DTI, NODDI, and q-space study. Neuroimage, 2016. 128: p. 180-92. 9. Yamada, H., O. Abe, T. Shizukuishi, J. Kikuta, T. Shinozaki, K. Dezawa, A. Nagano, M. Matsuda, H. Haradome, and Y. Imamura, Efficacy of distortion correction on diffusion imaging: comparison of FSL eddy and eddy_correct using 30 and 60 directions diffusion encoding. PLoS One, 2014. 9(11): p. e112411. 10. Avants, B.B., N.J. Tustison, G. Song, P.A. Cook, A. Klein, and J.C. Gee, A reproducible evaluation of ANTs similarity metric performance in brain image registration. Neuroimage, 2011. 54(3): p. 2033-44. 11. Oishi, K., K. Zilles, K. Amunts, A. Faria, H. Jiang, X. Li, K. Akhter, K. Hua, R. Woods, A.W. Toga, G.B. Pike, P. Rosa-Neto, A. Evans, J. Zhang, H. Huang, M.I. Miller, P.C. van Zijl, J. Mazziotta, and S. Mori, Human brain white matter atlas: identification and assignment of common anatomical structures in superficial white matter. Neuroimage, 2008. 43(3): p. 447-57.

## IC-02-05 ABNORMAL STRUCTURAL BRAIN CONNECTOME IN INDIVIDUALS WITH PRECLINICAL ALZHEIMER'S DISEASE

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**Background:** Alzheimer's disease has a long preclinical phase during which amyloid pathology and neurodegeneration accumulate in the brain without producing cognitive deficits. It is currently unclear whether these early disease stages are associated with a progressive disruption in the communication between brain regions that might lead to clinical decline and dementia. **Methods:** In this study we assessed the organization of the structural networks in cognitively normal (CN) individuals harbouring amyloid pathology (A+N-), neurodegeneration (A-N+) or both (A+N+). We combined graph theory with diffusion tensor imaging to investigate integration, segregation and centrality measures in the brain connectome of the previous groups. **Results:** At baseline, our findings revealed a disrupted network topology characterized by larger paths, lower efficiency, increased clustering and modularity in CN A-N+ and CN A+N+. After two years, CN A+N+ showed a progressive increase in the clustering, whereas no changes were observed in the other groups. Network topology correlated with cognitive speed in all groups and with memory performance specifically in CN A+N+. **Conclusions:** Altogether, our findings suggest that amyloid pathology is not sufficient to disrupt structural network topology, whereas neurodegeneration is. In contrast to CN A-N+, network organization in CN A+N+ individuals continued to decline over time and was associated with memory functions.

## SATURDAY, JULY 15, 2017
## ALZHEIMER'S IMAGING CONSORTIUM
## IC-03
## NON-ALZHEIMER'S DISEASE PATHOPHYSIOLOGY

## IC-03-01 WHAT HAPPENS TO THE HIPPOCAMPUS 12 MONTHS AFTER TRAINING? LONGITUDINAL LINEAR MIXED-EFFECTS MODEL ANALYSIS OF MILD COGNITIVE IMPAIRMENT IN THE SMART TRIAL

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