

identify early presymptomatic change and could also be a measure of disease progression. This study therefore investigates retinal changes in both presymptomatic and symptomatic subjects across all three genetic mutations (GRN, MAPT, C9orf72). **Methods:** We have recruited nine cases so far including two affected participants (2 C9orf72 mutations) and 7 presymptomatic participants: 4 mutation positive gene carriers and 3 mutation negative controls. We measured automated peripapillary retinal nerve fibre layer (RNFL) thickness acquired using spectral-domain optical coherence tomography (Optos OCT SLO) and averaged the RNFL thickness of all interpretable scans. **Results:** Average RNFL thickness for affected cases was 88.5 (standard deviation (sd) = 2.1)  $\mu\text{m}$ . For the presymptomatic cases average RNFL thickness was 103.8 (8.0)  $\mu\text{m}$  for mutation positive carriers and 106.0 (10.2)  $\mu\text{m}$  for mutation negative controls. **Conclusions:** Preliminary analysis suggests that RNFL thinning may be seen in symptomatic genetic FTD. There is also a trend for decreased thickness in mutation positive carriers although this was not significantly different from controls. Further analysis in a larger group is needed to see at what point abnormalities may be seen during the presymptomatic phase and whether there are differences between the different genetic groups.

**P2-085**      **TASK-SWITCHING ERRORS SHOW SENSITIVITY TO PRECLINICAL ALZHEIMER'S DISEASE BIOMARKERS**

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**Background:** Patterns of cerebrospinal fluid (CSF) and PIB imaging biomarkers characteristic of Alzheimer Disease (AD) pathology have been detected in individuals deemed to be cognitively normal. Recently, it has been shown that CSF markers, particularly those related to amyloid deposition, are related to preclinical cognitive deficits on an attention-demanding semantic discrimination task. It is unclear, however, whether this relationship is also found using an attention-demanding switch task in which individuals must actively switch between two task sets. Task-switching errors have been shown to be particularly sensitive to very early symptomatic AD, due to disruptions in the ability to reconfigure to a new task set when task demands change. We examined task-switching errors as a sensitive risk factor for preclinical AD as assessed by CSF and PIB imaging biomarkers in cognitively normal adults. **Methods:** 239 cognitively normal (Clinical Dementia Rating of 0) participants (age range = 45-84) with CSF measurements and PIB imaging at the Knight Alzheimer's Disease Research Center at Washington University completed 62 trials of a computerized consonant-vowel/odd-even (CVOE) switch task. Participants were presented with a letter/number stimulus (e.g., A-14) and were cued to classify the letter as a consonant/vowel or the number as odd/even. The letter/number cue switched every two trials. The primary dependent measures were the proportion of errors made during the classification task and the response latency with which the stimuli were correctly classified. **Results:** Correlational analyses indicated PIB imaging and CSF

A $\beta$ 42 were related to CVOE switch errors after controlling for participant age and education. No relationship was found between switch errors and CSF Tau and p-Tau181. Regression analyses revealed that both PIB and A $\beta$ 42 accounted for significant unique variance in CVOE errors. Biomarkers were not related to response latencies however, highlighting the sensitivity of switch errors in amyloid pathology. **Conclusions:** Results indicate that switch errors are a sensitive measure of preclinical cognitive deficits associated with accumulating amyloid pathology in cognitively normal adults. These results suggest that CSF markers, particularly those related to cortical amyloid deposition such as PIB and A $\beta$ 42, are associated with subtle attentional control deficits in cognitively normal adults.

**P2-086**      **THE EFFECT OF DEPRESSION ON THE SERUM LEVELS OF VASCULAR FACTORS IN ALZHEIMER'S DISEASE**

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**Background:** Growing evidence suggests that angiogenesis might represent a new pathogenic mechanism involved in the progression of Alzheimer's disease (AD). Among angiogenic cytokines, vascular endothelial growth factor (VEGF) levels in AD patients have been evaluated, but the results are controversial among studies. We investigated serum levels of VEGF in AD patients with depression, AD patients without depression, and the controls, respectively. The aim of this study is to elucidate relationship between VEGF, depression and cognitive impairment in AD. **Methods:** The CDR (Clinical Dementia Rating), MMSE-KC (the Mini Mental Status Examination-Korean version) and SGDS-K (the Korean version of the Geriatric Depression Scale-Short form) were measured in the subjects. Serum VEGF levels were measured in 24 AD patients with depression, 25 AD patients without depression, and 26 controls, using an enzyme-linked immunosorbent assay kit. **Results:** Serum VEGF levels in AD patients with depression were significantly higher than AD patients without depression or the control. A correlation was observed between VEGF and scores on SGDS-K, but no correlation was detected between VEGF and MMSE-KC scores. **Conclusions:** Serum VEGF levels in AD patients with depression were higher than those without depression. Depression might be associated with changes in serum levels of VEGF in AD patients. Our study suggests that presence of depression should be suspected in AD patients if altered vascular factor levels are detected.

**P2-087**      **A SYSTEMATIC REVIEW AND META-ANALYSIS FOR THE PREVALENCE OF DEPRESSION IN MILD COGNITIVE IMPAIRMENT**

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**Background:** Depressive symptoms commonly occur in association with mild cognitive impairment (MCI). Studies examining