DEVELOPING TOPICS: P4
POSTERS

P4-251 DISPHAGIA IN INDIVIDUALS WITH ALZHEIMER’S DISEASE: A SYSTEMATIC REVIEW OF THE EVIDENCE
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Background: Dementia is a syndrome that can be caused by a number of neurodegenerative disorders that affect memory, thinking, behavior, and the ability to perform activities of daily living. Alzheimer’s disease is the most common type of dementia and currently affects approximately 250,000 Canadians. Individuals with dementia of the Alzheimer’s type may experience swallowing impairment, or dysphagia, during the progression of the disease. Swallowing function is controlled in part by the parasympathetic branch of the autonomic nervous system. The primary objective of this study was to review studies of dysphagia in individuals with Alzheimer’s disease. Secondary objectives included identification and review of the literature concerning autonomic dysfunction in individuals with Alzheimer’s disease. Methods: Systematic searches of the PubMed, EBSCOHost, PsychINFO, Cochrane, EMBASE, and Scopus databases were completed. Search terms included dementia, Alzheimer’s disease, swallow, deglutition disorders, autonomic nervous system, and parasympathetic nervous system. Published studies and grey literature describing dysphagia or autonomic dysfunction in the context of Alzheimer’s disease were identified. Studies were reviewed and organized into categories according to type. These categories included clinical reports, physiologic studies, and brain imaging studies. Results: The literature contains evidence that dementia of the Alzheimer’s type results in distinct dysphagia or dysautonomia profiles, even in the early stages of the disease process. Conclusions: Although the prevalence and incidence of dysphagia or dysautonomia in the Alzheimer’s population is unknown, there is preliminary evidence to suggest that dysphagia or autonomic dysfunction can occur in dementia of the Alzheimer’s type.

P4-252 BLOOD ASSAY TO DETECT OLIGOMERIC BETA-AMYLOID WITH HIGH SENSITIVITY AND SPECIFICITY
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Background: The number of Alzheimer’s Diseases (AD) patients is exponentially increasing without ultimate cure or accurate diagnosis. Recently, imaging methods and CSF assay with triple markers could help diagnose AD and monitor the progression of the disease. However, the cost of these assays, confined availability of the materials, and invasiveness of CSF tap could limit a wide use for the early diagnosis and monitoring test. Originally, multimer detection system (MDS) was designed to detect specific oligomer form of pathologic prion protein in blood; in the mixture of pathologic and normal prion protein. After having developed the scapie blood test, MDS was applied to AD diagnostics. Here, we present the preliminary data of MDS for detecting oligomers of beta amyloid in blood to diagnose AD, as MDS-AD-Blood. The objective of this study was to improve the sensitivity and specificity of the MDS-AD as a possible option for new AD blood diagnostics. Methods: We collected blood samples from 30 patients with probable AD, 15 with SMI, 20 of normal healthy control. MDS-AD-Blood was repeatedly done for these samples with modification of many fundamental experimental elements, as like antibodies, anticoagulant, incubation time, volume of samples, and buffers. Results: MDS-AD distinguished AD blood samples from normal samples with approximately 90% sensitivity and specificity. Furthermore, MDS-AD detected the amyloid-beta oligomer from some patients’ samples with subjective memory impairment. Conclusions: The results from MDS-AD Blood test suggested the possibility to be used for the early diagnostic test. Also, the development of the sensitive detection methods for Aβ aggregates or oligomers in blood could be important for the therapeutic strategy and for the evaluation of the drugs, especially with upcoming releases of disease-modifying-drug candidates.

P4-253 HILAR GABAERGIC INTERNEURON ACTIVITY CONTROLS SPATIAL LEARNING AND MEMORY RETRIEVAL
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Background: Although extensive research has demonstrated the importance of excitatory granule neurons in the dentate gyrus of the hippocampus in normal learning and memory and in the pathogenesis of amnesia in Alzheimer’s disease (AD), the role of hilar GABAergic inhibitory interneurons, which control the granule neuron activity, remains unclear. Methods: Using transgenic mice specifically expressing Cre recombinase in GABAergic inhibitory neurons, we explored the function of hilar GABAergic interneurons in spatial learning and memory. We inhibited their activity by Cre-dependent viral expression of enhanced halorhodopsin (eNpHR3.0)Za light-driven chloride pumpZd during spatial learning and memory tests. Results: We found that, in GABAergic specific Cre mice injected with eNpHR3.0 virus in the hilus of the dentate gyrus, in vivo and in vitro illumination with yellow laser elicited inhibition of hilar GABAergic interneurons and consequent activation of dentate granule neurons, without affecting pyramidal neurons in CA3 and CA1 regions of the hippocampus. Optogenetic inhibition of hilar GABAergic interneuron activity impaired spatial learning and memory retrieval, without affecting memory retention, as determined in the Morris water maze test. Importantly, optogenetic inhibition of hilar GABAergic interneuron activity did not alter short-term working memory, motor coordination, or exploratory activity. Conclusions: Our findings establish a critical role for hilar GABAergic interneuron activity in controlling spatial learning and memory retrieval and provide evidence for the potential contribution of GABAergic interneuron impairment to the pathogenesis of amnesia in AD.

P4-254 PREDICTING BRAIN AMYLOIDOSIS IN MCI USING CLINICAL, COGNITIVE, IMAGING AND PERIPHERAL BLOOD PROTEIN MEASURES
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Background: Randomized, double-blind clinical trials for the latent stages of Alzheimer’s disease are now being planned. To identify enough amyloid positive cognitively normal subjects, these trials would need to screen tens of thousands of asymptomatic individuals using amyloid positron emission tomography (PET) imaging. Identifying people highly likely to be amyloid-positive without relying on amyloid PET could save time and resources. We investigated the accuracy of a novel unsupervised multimodal biomarker classifier for predicting brain amyloidosis in MCI subjects. By combining the predictive information of cognitive, imaging and peripheral blood protein biomarkers, we expected to achieve high accuracy in differentiating Pittsburgh compound B (PIB) positive (SUVR>1.6) from PIB negative (SUVR<1.6) subjects. Methods: Using the baseline cognitive, imaging