+ individuals (B= -5.79, SE=2.67, p=.04). No main or interaction effects were observed on the visual working memory task or episodic memory task.
Conclusions: Our finding that Aβ- individuals demonstrate less variability over time on a measure of processing speed is consistent with prior work. No associations were found between IIV in other cognitive domains and PET status. As noted by Allaire and Marsiske (2005), IIV is not a consistent phenomenon across different cognitive domains. Therefore, identifying which tests are the most sensitive to early change is crucial. Additional studies in larger, more diverse samples are needed prior to widespread clinical use for early detection of AD.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: positron emission tomography
Keyword 2: dementia - Alzheimer's disease
Keyword 3: computerized neuropsychological testing
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6 Exercise Induced Growth Factor Increases Directly and Indirectly Reduce Systemic Vascular Risk Parameters: Translational Project Amongst Midlife Human and Animal Models of Preclinical Alzheimer's disease and Vascular Dementia

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Objective: Using a humanized APOE3/4 (Alzheimer's disease genetic risk allele) mouse model we investigated the potential modulating effects of exercise on systemic risk factors and the ability of this mouse model to translate to active or sedentary, midlife, human participants. We present preliminary results of an ongoing, translational pilot study.

Participants and Methods: 26 Midlife individuals, ages 40-65, were recruited from the community and dichotomized into active or sedentary groups following health screening and cognitive assessment. Blood samples were drawn from human participants for lipid assessment and other general health measures as well as peripheral growth factors concentrations (VEGF, BDNF and FGF21). Traditional, transgenic mouse models have helped the scientific community to understand biological mechanisms of Alzheimer's disease (AD), but they do not develop significant neuronal loss, a hallmark of AD pathology. The MODEL-AD consortium has created a new "humanized" APOE4 model that has the human APOE4 allelic sequence in place of the mouse APOE gene; the model has shown known human phenotypes including deficits in cholesterol trafficking, amyloid clearance and BBB integrity. Of upmost importance, this model does not develop a full AD phenotype indicating that additional genetics and/or environmental factors are required as would be seen in human populations. We used males and females of this model to complete identical sedentary and active measures of each APOE genotype.
Results: Lipid and general health marker assessment between mouse and human were similar and reproduced published literature. In both humans and mice we saw increased total cholesterol and HDL in active females and decreased total cholesterol and HDL in active males. We also saw similar relationships between APOE genotype, sex, and activity with regards to triglycerides. Although total cholesterol, HDL and LDL measures are the primary lipids needed to confirm or deny translation, other lipid measurements were not equivalent between the two models.
Growth factor assessment in both species are also similar and reproduce published literature with regards to VEGF and BDNF as we see trending elevated levels in the active group. Less published on is the finding seen between active females and these elevated growth factors levels; our results indicates that although elevated as a result of exercise, this increase may be more prominent in females.
Conclusions: Based on the results found here we conclude that The Jackson Laboratory's humanized APOE3/4 mouse model is a translatable model of vascular dysfunction, dementia and Alzheimer's disease. We also conclude that exercise modulates these aspects by growth factor activation and increases

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resulting in downstream effects that reduce peripheral vascular risk factors and therefore reduce the risk of Alzheimer's disease as a result of neuroinflammation. Complete, APOE genotype results from human participants are still ongoing. Descriptive analysis is limited by human samples size.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: dementia - Alzheimer's disease
Keyword 2: cerebrovascular disease
Keyword 3: genetics
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7 The Role of Depressive Symptomatology in Predicting Cognitive and Functional Decline in Memory Clinic Patients

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Objective: Depressive symptomatology has long been shown to be associated with the onset of dementia, though the exact form and directionality of this association remains unclear. While much research has gone into confirming this link, there has been little investigation into the effects of depression on dementia progression after diagnosis. The aim of this study is to investigate the relationship between depressive symptomatology and cognitive and behavioural decline over the following year.
Participants and Methods: In a Rural and Remote Memory Clinic, 375 patients consecutively diagnosed with mild cognitive impairment (MCI), Alzheimer's Disease (AD), or non-AD dementia completed the Center for Epidemiological Studies Depression Scale (CES-D) at first visit and one-year follow-up to assess depressive symptomatology. The same cohort were evaluated for cognitive and behavioural decline through the completion of five clinical tests performed at the first visit and at one-year follow-up. Cognitive decline was assessed using the Mini Mental Status Exam (MMSE) and the Clinical Dementia Rating Scale (CDR). Neuropsychiatric symptoms were assessed using two subsets of data from the Neuropsychiatric Inventory (NPI severity and distress), both of which are completed by the patients' caregivers. Functional decline was assessed using the Functional Activities Questionnaire (FAQ). In both cognitive and functional decline, data were analyzed with linear regression analysis in the population subgroups of All Type Dementia (ATD, which includes MCI for this study) (N=375), Alzheimer's type dementia (N=187), and Mild Cognitive Impairment (N=74).
Results: In this study, we observed no correlation between CES-D scores at baseline and cognitive or functional decline over one year. However, we observed a significant positive correlation between changes in CES-D scores and NPI-severity scores over one year in patients with ATD (likely the most reliable observation from this study due to larger statistical power) and in the MCI subgroup, but not in the AD subgroup. This relationship may be attributable to a relationship between depression and neuropsychiatric symptoms in general, or to the fact that a person with dementia who exhibits more depressive symptomatology appears more impaired and causes greater distress in their caregivers, despite stability in the objective measures of their cognitive and functional status. This finding may indicate that intervention for depression is needed to alleviated caregiver burden when managing dementia patients.
Conclusions: Increasingly severe depressive symptomatology may exacerbate neuropsychiatric symptomatology but did not correlate with cognitive and functional decline in patients with dementia. More studies are needed to help delineate the relationship between depression and dementia progression.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: dementia - Alzheimer's disease
Keyword 2: dementia - other cortical
Keyword 3: depression
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8 Perspectives of Self, Stigma, and the Future Following Alzheimer's Disease

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