Participants and Methods: 135 clinically referred older adults (mean age 75.5 years) undergoing neuropsychological evaluation at a comprehensive multidisciplinary memory clinic were included in this study [37% with mild cognitive impairment (MCI) and 51.5% with dementia]. Collateral informants completed the Functional Activities Questionnaire (FAQ; Pfeffer, 1982) as well as 11 items created to parallel the FAQ wording that assessed technology-related iADLs such as digital financial management (i.e. online bill pay), everyday technology skills (i.e. using a smartphone; remembering a password), and other technology mediated activities (i.e. visiting internet sites; online shopping).
Results: Care partners rated tech iADLs items as applicable for the majority of items. For example, technology skill items were applicable to 90.4% of the sample and online financial management questions were applicable for 76.4% of participants. Applicability ratings were similar across patients in their 60's and 70's, and lower in those over age 80. Care partners indicated less overall impairment on technology-related iADLs (M =1.22, SD =.88) than traditional FAQ iADLs (M =1.36, SD = .86), t(129) = 3.529, p =.001). A composite of original FAQ paperwork and bill pay items (M = 1.62, SD = 1.1) was rated as more impaired than digital financial management tasks (M = 1.30, SD = 1.09), t(122) = 4.77, p <.001). In terms of diagnostic accuracy, tech iADL items (AUC= .815, 95% CI [.731, -.890]) appeared to perform comparably to slightly better than the traditional FAQ (AUC =.788, 95% CI [.705, .874]) at separating MCI and dementia, though the difference between the two was not statistically significant in this small pilot sample.
Conclusions: Technology is rapidly changing how older adults and those with ADRD perform a host of iADLs. This pilot study suggests broad applicability of tech iADL to the lives of those with ADRD and highlights how measurement of these skills may help identify trends in iADL habits that may help to mitigate the impact of ADRD on daily functions. Further, this data suggests the need to refine and improve upon existing iADL measures to validly capture the evolving technological landscape of those living with ADRD.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: activities of daily living
Keyword 2: dementia - Alzheimer's disease
Keyword 3: aging disorders
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28 Traumatic Brain Injury and Genetic Risk for Alzheimer's Disease Influence β-Amyloid Levels

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Objective: Traumatic brain injuries (TBIs) are a common occurrence among Veterans and may increase risk for neurodegenerative diseases, such as Alzheimer's disease (AD). Neuropathological correlates of AD, including buildup of β-amyloid (Aβ) plaques, formation of neurofibrillary tangles, and cortical atrophy, begin years before the onset of noticeable clinical and cognitive symptoms, emphasizing the importance of identifying early risk factors that could be targeted to prevent the development of AD. Of note, Aβ ratios (e.g., Aβ 42/40) have been shown to efficiently capture brain amyloid accumulation in prodromal AD, and thus may serve as a useful biological marker of preclinical AD. The present study investigates the mechanism by which TBI is associated with AD by examining the synergistic effects of TBI and genetic risk for AD on Aβ among aging Veterans without dementia.
Participants and Methods: Participants included 88 White, Non-Hispanic/Latino male Vietnam War Veterans (M_{age} = 68.3 years) from the Alzheimer's Disease Neuroimaging Initiative Department of Defense (ADNI DoD) cohort, 49 of whom reported a history of at least one mild, moderate, or severe TBI. Genetic risk for AD was assessed via genome-wide polygenic risk scores. Aβ levels were extracted from cerebrospinal fluid and Aβ 42/40 ratios were calculated as an index of Aβ deposition in the brain. Linear regression models were run to determine if TBI history and polygenic risk influence Aβ 42/40 levels. An ANCOVA was implemented to examine the interaction between TBI severity and polygenic risk. Covariates in all models included age, education, and posttraumatic stress disorder symptoms.
Results: Results demonstrated a significant interaction between TBI and genetic risk on Aβ 42/40 (B = -0.45, $P_{uncorrected}$ = 0.029, $P_{corrected}$ =

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0.0495). Specifically, higher polygenic risk was associated with lower Aβ 42/40 ratio, suggesting greater Aβ burden in the brain, among those with a history of TBI (*pr* = -0.33, *P* = 0.024) compared to individuals without a history of TBI (*pr* = 0.17, *P* = 0.308). This relationship trended towards being stronger as a function of increasing TBI severity ($F(2, 77) = 3.01$, $P = 0.055$).
Conclusions: These results show that, in the context of TBI, higher genetic risk for AD is associated with greater AD-related pathology, particularly with more severe injuries. TBI and polygenic risk may implicate similar biological pathways, notably amyloid precursor protein processing, to increase Aβ burden in the brain and likelihood of progression to AD in future years. These findings could inform early intervention techniques to delay or preclude conversion to AD.

Categories: Dementia (Alzheimer's Disease)
Keyword 1: dementia - Alzheimer's disease
Keyword 2: traumatic brain injury
Keyword 3: genetics
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29 Smoking as a Risk Factor: Altered Brain Activity in Areas Associated with Preclinical Alzheimer's Disease

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Objective: Those at genetic risk for Alzheimer's Disease (AD) because of the ApoE ε4 allele show differences in activation during olfactory information processing and memory in areas such as MTL structures, entorhinal cortex, posterior cingulate, precuneus, and inferior parietal lobule, suggesting preclinical AD neuropathology and olfactory impairment as a biomarker for predicting later AD onset (Murphy, 2019). The effects of smoking on AD have varied, with early studies suggesting either no effect or protective effects, and recent studies suggesting smoking as a risk factor for AD but with the need for further investigation in preclinical stages. Therefore, this study focused on olfaction and smoking as risk factors for preclinical AD neuropathology by studying differences in fMRI BOLD signal changes in smokers and nonsmokers during olfactory tasks.
Participants and Methods: Archival data from 25 non-demented older adults recruited from the UCSD Alzheimer's Disease Research Center who completed an Assessment Scale-Cognitive Subscale (ADAS-Cog) and functional MRI scans at 3T, acquired during performance of an odor identification task. Odor Identification (OI) measured correct (hits) or incorrect (misses) identification of odors presented by an olfactometer to deliver the odor stimuli in short, controlled durations during fMRI scanning.
Results: fMRI data were preprocessed using fMRIprep, smoothed at 4mm, scaled, and first level analyses were conducted using 3dDeconvolve in AFNI with time points corresponding to hits and misses as regressors. Differences between smokers and nonsmokers revealed smokers show a larger difference in BOLD signal change from hits minus misses at five significant clusters ($p = 0.01$ with the minimum cluster size [voxels] at 42). Peak areas of significant clusters included the right precuneus, right calcarine gyrus, left inferior parietal lobule, left superior parietal lobule, and left middle occipital gyrus. Analyses suggested a greater difference in activity between hits and misses in smokers compared to nonsmokers, with more activity during hits.
Conclusions: Differences in activation between smokers and nonsmokers during an olfactory identification task, with greater activity in smokers during hits, suggests greater effort to correctly identify an odor. These findings of hyperactivation in areas (such as the precuneus and inferior parietal lobule) are similar to findings of hyperactivation during odor memory observed in studies of ε4 carriers during preclinical stages. Results provide further insight into smoking as a risk factor for AD. Moreover, results suggest the risk of smoking could potentially be reflected in altered activity in olfactory information processing networks in preclinical stages of AD. The study highlights the need for research to further understand the role smoking plays in the development of AD and the use of olfaction as a biomarker to aid in disease detection, prevention, and stage-associated treatments.

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