

# Financial Capacity and Regional Cerebral Tau in Cognitively Normal Older Adults, Mild Cognitive Impairment, and Alzheimer's Disease Dementia

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## Abstract.

**Background:** Financial capacity is often one of the first instrumental activities of daily living to be affected in cognitively normal (CN) older adults who later progress to amnesic mild cognitive impairment (MCI) and Alzheimer's disease (AD) dementia.

**Objective:** The objective of this study was to investigate the association between financial capacity and regional cerebral tau.

**Methods:** Cross-sectional financial capacity was assessed using the Financial Capacity Instrument – Short Form (FCI-SF) in 410 CN, 199 MCI, and 61 AD dementia participants who underwent florataucipir tau positron emission tomography from the Alzheimer's Disease Neuroimaging Initiative (ADNI). Linear regression models with backward elimination were used with FCI-SF total score as the dependent variable and regional tau and tau-amyloid interaction as predictors of interest in separate analyses. Education, age, sex, Rey Auditory Verbal Learning Test Total Learning, and Trail Making Test B were used as covariates.

**Results:** Significant associations were found between FCI-SF and tau regions (entorhinal:  $p < 0.001$ ; inferior temporal:  $p < 0.001$ ; dorsolateral prefrontal:  $p = 0.01$ ; posterior cingulate:  $p = 0.03$ ; precuneus:  $p < 0.001$ ; and supramarginal gyrus:  $p = 0.005$ ) across all participants. For the tau-amyloid interaction, significant associations were found in four regions (amyloid and dorsolateral prefrontal tau interaction:  $p = 0.005$ ; amyloid and posterior cingulate tau interaction:  $p = 0.005$ ; amyloid and precuneus tau interaction:  $p < 0.001$ ; and amyloid and supramarginal tau interaction:  $p = 0.002$ ).

**Conclusion:** Greater regional tau burden was modestly associated with financial capacity impairment in early-stage AD. Extending this work with longitudinal analyses will further illustrate the utility of such assessments in detecting clinically meaningful decline, which may aid clinical trials of early-stage AD.

**Keywords:** Alzheimer's disease, amyloid, financial capacity, instrumental activities of daily living, mild cognitive impairment, positron emission tomography, tau

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(<http://adni.loni.usc.edu>). As such, the investigators within the ADNI contributed to the design and implementation of ADNI and/or provided data but did not participate in analysis or writing of this report. A complete listing of ADNI investigators can be found at: [http://adni.loni.usc.edu/wp-content/uploads/how\\_to\\_apply/ADNI\\_Acknowledgement\\_List.pdf](http://adni.loni.usc.edu/wp-content/uploads/how_to_apply/ADNI_Acknowledgement_List.pdf)

## INTRODUCTION

Alzheimer's disease (AD) dementia is a neurodegenerative disorder consisting of cognitive, behavioral, and functional impairment that is estimated to affect about 14 million Americans over the age of 65 by the year 2050 [1]. Progression from a diagnosis of cognitively normal (CN) to amnesic mild cognitive impairment (MCI), followed by further decline and transition to AD dementia, is often accompanied by impairment in the ability to perform instrumental activities of daily living (IADL) [2–4]. In addition to negatively impacting the individual, IADL impairment increases caregiver mental and financial burden [5–7].

Financial capacity [8], performing household chores/repairs, and medication management are some of the many tasks that consist of IADL. Financial capacity has been defined as a complex IADL ability to manage one's own finances in a consistent manner (e.g., making change, balancing the checkbook, and paying bills on time) [9]. Previous studies have shown that financial capacity often manifests as the first aspect of IADL decline in individuals who later progress to MCI and AD dementia [8, 10, 11] giving reason to further investigate them.

The association between a variety of neuroimaging modalities and IADL impairment across older adults has been an increasing topic of interest. Several studies have previously found that inferior temporal cortical thinning, frontal and parietal hypometabolism, frontal white matter hyperintensities, and bilateral prefrontal, striate, and anterior cingulate hypoperfusion have been associated with greater IADL impairment across the AD spectrum [12–15]. More recently, Halawa and colleagues found that greater *in vivo* medial and inferior temporal tau burden and amyloid burden are associated with greater IADL impairment across the AD spectrum [16], while Marshall and colleagues found an association in MCI and AD dementia between IADL impairment and medial temporal and frontal tau burden in individuals with elevated amyloid [17]. These findings are in line with postmortem and cerebrospinal fluid (CSF) studies that have found that IADL impairment are associated with neurofibrillary tangles (tau burden) and neuritic plaques (amyloid burden) in medial temporal, frontal, and occipital regions in individuals with moderate to severe AD, as well as CSF lower amyloid- $\beta$  1–42 and greater total tau across the AD spectrum [18, 19].

As described above, previous studies have displayed an association between IADL and certain brain regions, but few studies have assessed the relationship between financial capacity and AD pathology. One such recent study found that higher cortical amyloid burden was significantly associated with higher impairment in financial capacity in participants with early-stage AD [20]. However, to our knowledge, no previous study has investigated the relationship between tau pathology and financial capacity.

Tau and amyloid accumulation are the hallmarks of AD pathology and correspond to cognitive decline [21–23]. Cohorts of older adults followed longitudinally with multi-modal imaging such as the Alzheimer's Disease Neuroimaging Initiative (ADNI-3), utilize positron emission tomography (PET) tracers such as flortaucipir (FTP) (a.k.a. AV-1451) and florbetapir (FBP) to measure tau and amyloid burden respectively [24–26], [27]. Cortical amyloid PET has also been used in clinical settings to assess biomarker burden in MCI and dementia patients [22]. Increases in amyloid and tau burden are associated with the progression of cognitive decline in older adults at risk for AD [28].

Change in financial capacity, a complex, high-level aspect of IADL, may play an important role in the early clinical manifestation of AD. Therefore, if assessed with a sensitive enough test, deficits in financial capacity could be detected as early as the preclinical stage of AD. The objective of this study was to investigate the association between financial capacity and regional cerebral tau across CN, MCI, and AD dementia participants. The current study looked at six bilateral subcortical and cortical regions (entorhinal cortex, inferior temporal cortex, dorso-lateral prefrontal cortex, posterior cingulate cortex, precuneus, and supramarginal gyrus), since these are regions that have been examined in the most recent study [16]. To assess financial capacity, we used the Financial Capacity Instrument – Short form (FCI-SF) [9], a performance-based IADL measure that evaluates one's financial knowledge. We hypothesized that worse performance on the FCI-SF will be associated with greater tau burden primarily in temporal and frontal regions across CN and cognitively symptomatic participants. We further hypothesized that an interaction between greater tau and greater amyloid will be associated with worse financial capacity, given that Halawa and colleagues reported such results using a 10-item IADL questionnaire. Studying the cross-sectional relationship between regional tau

deposition and financial capacity can serve as a basis for future longitudinal studies assessing the utility of IADL impairment in tracking and predicting disease progression in early-stage AD.

## MATERIALS AND METHODS

### *Participants*

The study consisted of 410 CN, 199 amnesic MCI, and 61 AD dementia older adults ranging in age from 56–95 years old who were participants in the Alzheimer's Disease Neuroimaging Initiative 3 (ADNI-3) study [29]. ADNI was launched in 2003 as a public-private partnership, led by Principal Investigator Michael W. Weiner, MD and involves 50 North American sites. Its primary goal has been to test whether serial magnetic resonance imaging (MRI), PET, other biological markers, and clinical and neuropsychological assessments can measure the progression from CN to MCI and mild AD dementia. Only participants who underwent AV-1451 tau PET imaging within a year of completing the FCI-SF were included in the sample. Participants were 48.1% female. Diagnoses were determined by the principal investigator or co-investigators at each site utilizing assessments of cognition, neuropsychiatric symptoms, and IADL (not including the FCI-SF). Participants with scores higher than 6 on the 15-item Geriatric Depression Scale (GDS) were excluded from the study. The study was approved by the local Institutional Review Board of each participating ADNI site. Participants and study partners reviewed and signed forms prior to completion of any study procedures.

### *Clinical assessments*

Financial capacity were assessed using the FCI-SF [9] that evaluates participants financial skills in the domain of monetary calculation, financial concepts, register usage, and bank statement usage. The FCI-SF consists of 37 items and five domain scores (Mental Calculation, Financial Conceptual Knowledge, Single Checkbook Register, Complex Checkbook Register, Bank Statement Management) that are summed into a Total Score (range of 0–74), with higher scores indicating better financial capacity. The total score is a summary of individual domains: Mental Calculation (0–4), Financial Conceptual Knowledge (0–8), Single Checkbook Register (0–20), Complex Checkbook Register

(0–28), and Bank Statement Management (0–14).

Cognitive assessments were used as covariates in analyses. The Rey Auditory Verbal Learning Test (RAVLT) [30] total learning score assesses performance of episodic memory. In this assessment, participants are presented a list of 15 words over 5 trials and are asked to repeat them after a distractor list and a delay. The total learning score ranges between 0–75 and lower scores indicates poorer performance. The Trail Making Test B (TMT-B) [31] score was used to assess executive function (divided attention and task switching performance). It is recorded by the time it takes an individual to complete the task, where higher scores indicate greater impairment.

### *Tau and amyloid PET data*

FTP and FBP PET imaging acquisition and processing was described in prior publications extensively [32, 33]. Briefly, FTP scans were co-registered to corresponding MRI scans that were segmented and parcellated with Freesurfer (version 5.3.0) in order to calculate the mean FTP uptake within each Freesurfer-defined region with cerebellar cortex as the reference region yielding a standardized uptake value ratio (SUVR) (UC Berkeley—AV1451 Analysis Methods, ADNI). Six bilateral subcortical and cortical regions of interest (ROI) were chosen based on prior studies assessing cerebral regional changes and IADL: entorhinal cortex, inferior temporal cortex, dorsolateral prefrontal cortex, posterior cingulate cortex, precuneus, and supramarginal gyrus [12, 19, 34, 35]. FBP scans were co-registered to corresponding MRI scans that were segmented and parcellated with Freesurfer (version 5.3.0) to define the amyloid cortical gray matter ROI (frontal, anterior cingulate, posterior cingulate, lateral parietal, and lateral temporal) that make up an aggregate cortical ROI with whole cerebellum as the reference region yielding an SUVR (UC Berkeley—AV45 Analysis Methods, ADNI). Amyloid status (positive or negative) was defined by a cutoff of FBP SUVR of 1.10 as previously described—amyloid-positive >1.10 and amyloid-negative  $\leq$ 1.10.

### *Statistical analysis*

All analyses for this study were carried out using SPSS version 26.0 (IBM). First, Pearson correlations between the FCI-SF total score and each tau region (entorhinal cortex, inferior temporal cortex, dorsolateral prefrontal cortex, posterior cingulate cortex,

Table 1  
Participant demographics and characteristics

Diagnosis	All	CN	MCI	AD dementia
<b>n</b>	<b>670</b>	<b>410</b>	<b>199</b>	<b>61</b>
Age (y)	74.5 ± 7.8 <sup>††</sup>	73.6 ± 7.3	75.4 ± 8.4	77.3 ± 8.4
Sex (% female)	48.1	41.2	58.3	60.7
Education (y)	16.6 ± 2.5 <sup>††</sup>	16.8 ± 2.3	16.3 ± 2.6	15.8 ± 2.7
Race (% White)	90.7	88.8	93.5	95.1
MMSE	28.1 ± 2.6*	29.1 ± 1.2	27.8 ± 2.0	22.3 ± 3.5
CDR sum of boxes	0.9 ± 1.9*	0.1 ± 0.2	1.5 ± 0.9	5.6 ± 2.5
TMT-B	91.7 ± 58.4*	72.9 ± 33.9	104.2 ± 56.7	192.9 ± 92.9
RAVLT Total Learning	41.3 ± 12.8*	47.1 ± 10.4	34.8 ± 10.0	23.3 ± 7.5
Entorhinal FTP SUVR	1.17 ± 0.23*	1.10 ± 0.13	1.22 ± 0.26	1.49 ± 0.31
Inferior temporal cortex FTP SUVR	1.25 ± 0.28*	1.18 ± 0.15	1.27 ± 0.27	1.69 ± 0.49
Dorsolateral prefrontal cortex FTP SUVR	1.04 ± 0.18**	1.01 ± 0.09	1.04 ± 0.16	1.26 ± 0.38
Posterior cingulate FTP SUVR	1.11 ± 0.18**	1.07 ± 0.10	1.12 ± 0.16	1.33 ± 0.37
Precuneus FTP SUVR	1.13 ± 0.22**	1.08 ± 0.09	1.13 ± 0.16	1.47 ± 0.51
Supramarginal FTP SUVR	1.09 ± 0.20 <sup>‡</sup>	1.06 ± 0.10	1.09 ± 0.17	1.37 ± 0.46
Cortical florbetapir (amyloid) SUVR	1.15 ± 0.23*	1.10 ± 0.15	1.21 ± 0.28	1.44 ± 0.22
Amyloid status (% amyloid-positive)	40.3	30.0	49.5	89.7

MMSE, Mini-Mental State Examination; CDR, Clinical Dementia Rating; TMT-B, Trail Making Test B; RAVLT, Rey Auditory Verbal Learning Test. All values (except n, sex, and race) represent mean ± standard deviation (range). <sup>††</sup> $p < 0.05$  for CN versus MCI and CN versus AD dementia. \* $p < 0.001$  for CN versus MCI, CN versus AD dementia and MCI versus AD dementia. \*\* $p < 0.05$  for CN versus MCI, and  $p < 0.001$  for CN versus AD and MCI versus AD dementia. <sup>‡</sup> $p < 0.001$  for CN versus AD dementia and MCI versus AD dementia.

precuneus, and supramarginal gyrus) were performed to evaluate the unadjusted associations across all participants and then by diagnoses (CN and cognitively symptomatic consisting of MCI and AD dementia). The tau regions with associations of  $p < 0.05$  were subsequently used in the primary analyses. Linear regression models with backward elimination ( $p < 0.10$  for retention,  $p < 0.05$  for significance) were used in the primary analyses to determine the association between the FCI-SF total score (dependent variable) and the significant tau regions identified from the unadjusted correlations. Each tau region was tested in its own model. We included the following covariates in the models: education, age, sex, RAVLT Total Learning Score, and TMT-B. RAVLT and TMT-B were included as covariates because memory impairment and executive dysfunction are associated with greater IADL impairments in individuals with MCI and AD dementia and have a wider range of performance across early-stage AD compared to a test of global cognition such as the Mini-Mental State Examination (MMSE) [36, 37].

In secondary analyses, models were repeated for the FCI-SF total score but with the addition of an interaction term between cortical regional tau and cortical amyloid. Additional analyses looked at FCI-SF total score and cortical tau burden associations, separately, in cognitively asymptomatic (CN) and symptomatic (combined MCI and AD dementia) participants. Lastly, models were repeated using the

five FCI-SF domain scores to assess regional tau association with each FCI-SF domain (see the Supplementary Material).

Unstandardized coefficients ( $\beta$ ) with 95% confidence intervals (CI), Partial regression correlations (pr), and significance test results (p values) were reported. We did not adjust for multiple comparisons in any of the above analyses.

## RESULTS

Participant demographics and characteristics, including cognitive, tau, and amyloid values by diagnostic group can be found in Table 1. FCI-SF total scores and domain scores (Mental Calculation, Financial Conceptual Knowledge, Single Checkbook Register, Complex Checkbook register, and Bank Statement Management) can be found in Table 2.

Unadjusted correlations across all participants showed significant associations between the FCI-SF total score and all 6 tau regions (entorhinal cortex:  $r = -0.40$ ,  $p < 0.001$ ; inferior temporal cortex:  $r = -0.48$ ,  $p < 0.001$ ; dorsolateral prefrontal cortex:  $r = -0.39$ ,  $p < 0.001$ ; posterior cingulate cortex:  $r = -0.36$ ,  $p < 0.001$ ; precuneus:  $r = -0.44$ ,  $p < 0.001$ ; and supramarginal gyrus:  $r = -0.41$ ,  $p < 0.001$ ). Unadjusted correlations performed within CN participants showed significant associations between the FCI-SF total score and 2 tau regions (entorhinal cortex:

Table 2  
Financial capacity instrument – short form performance scores

Diagnosis	All	CN	MCI	AD dementia
FCI Total Score	64.01 ± 12.30*	68.59 ± 5.77	61.47 ± 10.57	37.81 ± 18.40
FCI Mental Calculation	3.42 ± 1.20**	3.65 ± 0.87	3.39 ± 1.29	1.93 ± 1.71
FCI Financial Conception Knowledge	7.18 ± 1.29‡	7.54 ± 0.84	7.04 ± 1.28	5.05 ± 1.82
FCI Single Checkbook Register	17.56 ± 3.56*	18.71 ± 1.85	17.32 ± 2.98	10.44 ± 5.50
FCI Complex Checkbook Register	23.58 ± 6.32*	25.70 ± 3.50	22.45 ± 6.01	12.21 ± 9.37
FCI Bank Statement Management	12.03 ± 2.68*	12.91 ± 1.74	11.41 ± 2.74	8.03 ± 3.58

\* $p < 0.001$  for CN versus MCI, CN versus AD dementia and MCI versus AD dementia. \*\* $p < 0.05$  for CN versus MCI, and  $p < 0.001$  for CN versus AD dementia and MCI versus AD dementia. †  $p < 0.001$  for CN versus AD dementia and MCI versus AD dementia.

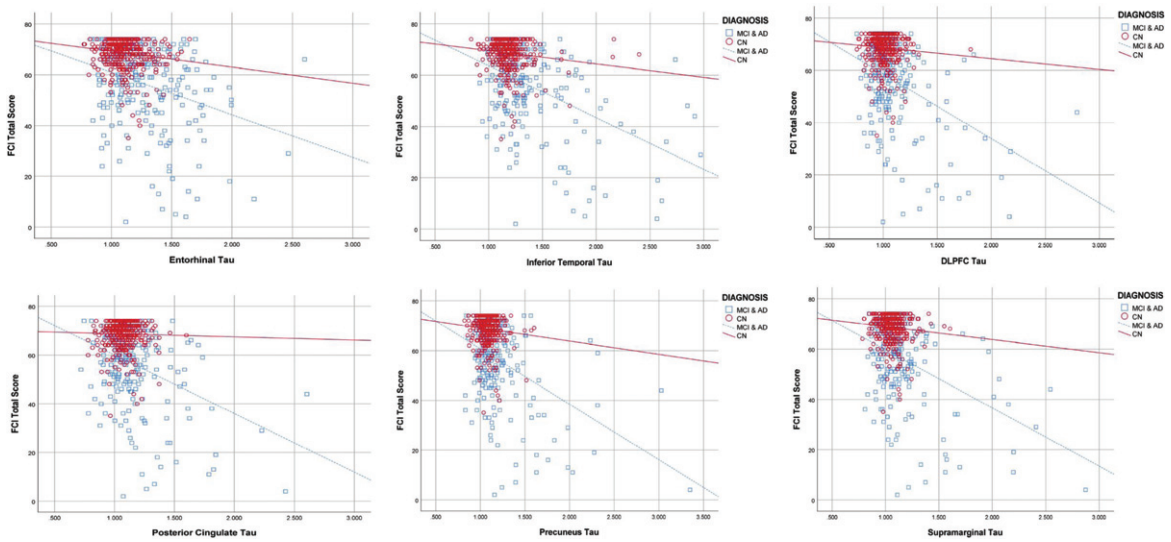


Fig. 1. Scatter plot of entorhinal cortex, inferior temporal cortex, dorsolateral prefrontal cortex (DLPFC), posterior cingulate, precuneus, and supramarginal FTP tau versus FCI-SF total score, stratified by diagnosis (CN versus symptomatic participants—combined MCI and AD dementia).

$r = -0.15, p = 0.003$ ; and inferior temporal cortex:  $r = -0.13, p = 0.008$ ) (see Fig. 1). Lastly, for the symptomatic participants (combined MCI and AD dementia), unadjusted correlation showed significant associations between the FCI-SF total score and all 6 tau regions (entorhinal cortex:  $r = -0.30, p < 0.001$ ; inferior temporal cortex:  $r = -0.46, p < 0.001$ ; dorsolateral prefrontal cortex:  $r = -0.39, p < 0.001$ ; posterior cingulate cortex:  $r = -0.36, p < 0.001$ ; precuneus:  $r = -0.42, p < 0.001$ ; and supramarginal gyrus:  $r = -0.41, p < 0.001$ ) (see Fig. 1).

Primary analyses

Linear regression models with backward elimination were run for tau regions that had significant associations with unadjusted correlations across all participants utilizing the FCI-SF total score with education, age, sex, RAVLT Total Learning Score, and TMT-B as covariates. FCI-SF total scores were sig-

nificantly associated with greater tau burden in all 6 regions (see Table 3).

Secondary analyses

As shown on Table 4, models were repeated including an interaction term between cortical amyloid and regional tau across all participants and revealed associations between FCI-SF total score and greater tau burden within participants with greater cortical amyloid in four regions (cortical amyloid and dorsolateral prefrontal cortex tau interaction, cortical amyloid and posterior cingulate tau interaction, cortical amyloid and precuneus tau interaction, and cortical amyloid and supramarginal tau interaction (see Fig. 2)).

Models were repeated separately in CN and symptomatic participants. For CN participants, no significant associations were found in any tau regions. When an interaction term between cortical amyloid

Table 3

Results of separate linear regression models across all participants with tau region as the predictor of interest, the FCI-SF total score as the dependent variable, and education, age, sex, RAVLT Total Learning Score, and TMT-B as covariates

Tau region	$\beta$	95% CI for $\beta$	pr	p
Entorhinal cortex	-6.75	-9.74, -3.75	-0.18	<0.001
Inferior temporal cortex	-6.07	-8.74, -3.39	-0.18	<0.001
Dorsolateral prefrontal cortex	-5.11	-9.14, -1.07	-0.10	0.01
Posterior cingulate	-4.61	-8.67, -0.55	-0.09	0.03
Precuneus	-7.26	-11.00, -3.53	-0.15	<0.001
Supramarginal gyrus	-5.54	-9.39, -1.68	-0.11	0.001

$\beta$ , unstandardized coefficients; CI, confidence interval; pr, partial correlation coefficient.

Table 4

Results of separate linear regression models across all participants with the interaction of cortical amyloid and regional tau as the predictor of interest, the FCI-SF total score as the dependent variable, and education, age, sex, RAVLT Total Learning Score, and TMT-B as covariates

Amyloid x tau interaction	$\beta$	95% CI for $\beta$	pr	p
Amyloid x dorsolateral prefrontal cortex tau	-5.28	-8.95 $\pm$ -1.60	-0.15	0.005
Amyloid x posterior cingulate tau	-2.95	-4.97 $\pm$ -0.92	-0.16	0.005
Amyloid x precuneus tau	-3.61	-5.58 $\pm$ -1.63	-0.19	<0.001
Amyloid x supramarginal gyrus tau	-3.12	-5.08 $\pm$ -1.16	-0.17	0.002

$\beta$ , unstandardized coefficients; CI, confidence interval; pr, partial correlation coefficient.

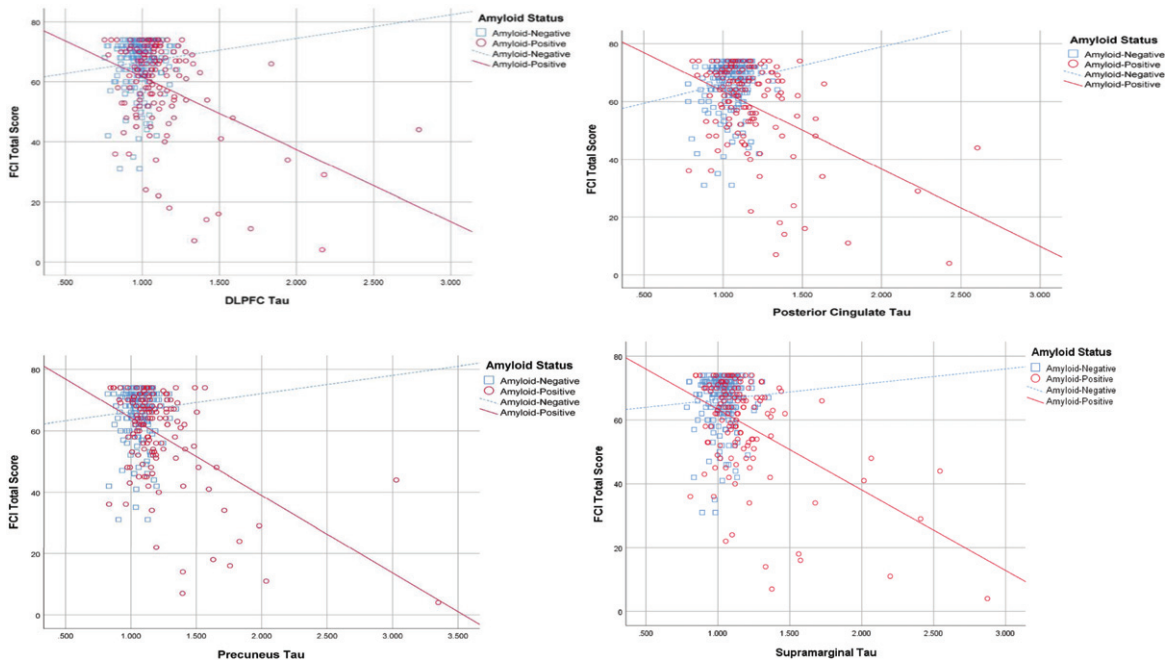


Fig. 2. Scatter plot of dorsolateral prefrontal cortex (DLPFC), posterior cingulate, precuneus, and supramarginal FTP tau versus FCI-SF total score in all participants, stratified by Amyloid status (Amyloid-Positive=florbetapir SUVR > 1.10; Amyloid-Negative=florbetapir SUVR  $\leq$  1.10).

and regional tau was added for CN participants, FCI-SF total score was significantly associated with 2 regions (cortical amyloid and inferior temporal cortex tau interaction:  $\beta = -31.00$ , 95% CI =  $-55.37$ ,  $-6.62$ ,  $pr = -0.17$ ,  $p = 0.01$ ; cortical amyloid and precuneus tau interaction:  $\beta = -34.69$ , 95% CI =  $-69.32$ ,  $-0.05$ ,  $pr = -0.13$ ,  $p = 0.05$ ). In symptomatic par-

ticipants, FCI-SF total score was significantly associated with two tau regions (inferior temporal cortex:  $\beta = -4.83$ , 95% CI =  $-9.04$ ,  $-0.61$ ,  $pr = -0.15$ ,  $p = 0.03$ ; and precuneus:  $\beta = -6.51$ , 95% CI =  $-12.27$ ,  $-0.76$ ,  $pr = -0.15$ ,  $p = 0.03$ ). However, no significant associations were found in any regions when an interaction term between cortical amyloid and regional tau

was added in symptomatic participants. Models were repeated using the five FCI-SF domain scores (see the Supplementary Material).

## DISCUSSION

This study explored the cross-sectional association between the regional cerebral accumulation of tau and financial capacity across CN, MCI, and AD dementia participants. Our findings suggest that greater regional tau in the entorhinal, inferior temporal, dorsolateral prefrontal, posterior cingulate, precuneus, and supramarginal gyrus are modestly associated with greater financial capacity impairment across these diagnostics groups. The current study, which employed a performance-based IADL test, the FCI-SF, confirms results of previous studies, which employed an IADL questionnaire, the Functional Activities Questionnaire (FAQ), and showed an association between IADL impairment and greater tau burden in temporal and frontal regions in MCI and AD dementia [16, 17].

Our study also examined the associations between financial capacity and the interaction of tau and amyloid. Our results showed that dorsolateral prefrontal cortical tau, posterior cingulate tau, precuneus tau, and supramarginal tau were associated with greater financial capacity impairment in individuals with elevated cortical amyloid. Halawa et al. previously found an association between IADL and the interaction of entorhinal and inferior temporal cortex tau with cortical amyloid [16], while Marshall et al. found an association between IADL and the interaction of entorhinal and orbitofrontal cortex tau and cortical amyloid [17]. The differences in regional associations could be due to a difference in the test used to assess IADL—both of the previous studies used a study partner-reported questionnaire targeting a variety of IADL, the FAQ, while the current study used a performance-based test targeting financial capacity, the FCI-SF.

Additional analyses explored tau accumulation separately within diagnostic groups. When looking only at the asymptomatic (CN) group, a modest association between greater IADL impairment and inferior temporal and precuneus tau within individuals with elevated cortical amyloid was seen. In the symptomatic (combined MCI and AD dementia) group, an association between greater IADL impairment and inferior temporal and precuneus tau was observed. However, the interactions of regional tau

and cortical amyloid were not significant in the symptomatic group. This finding was contrary to Halawa et al., as they found an association within the symptomatic group but not within the asymptomatic group. While association were modest in strength, this suggests that the FCI-SF, used in the current study, may be more sensitive to detecting the earliest IADL changes in the preclinical stage of AD, while the FAQ, used in the previous study, may be more geared toward detecting IADL changes at the stage of MCI and mild AD dementia. A few prior studies have shown an association between IADL changes and cortical amyloid within CN older adults [20, 38]. However, to our knowledge, this is the first study to show an association between regional tau burden and IADL within CN older adults.

When assessing the regional tau association with each of the FCI-SF five domains (see the Supplementary Material), similar results were obtained, suggesting that within this sample there was not a particular domain of financial capacity that drove the findings.

A strength of the current study was that we utilized the ADNI dataset, which represents a multicenter study of clinical and multi-modal imaging in a well-characterized cohort across early-stage AD. The FCI-SF was added to the third iteration of ADNI (ADNI-3) and currently few participants had repeat assessments. Since longitudinal tau and clinical data are collected in ADNI-3, when the data becomes available, we plan to extend our analyses to determine the longitudinal relationship between financial capacity and regional tau burden.

Limitations to our study consisted of a lack of diversity within the ADNI-3 participants. Most participants (about 91%) reported being White and this contributed to the data being less generalizable to the older population, since we know that Blacks and African Americans are twice as likely to develop AD [39]. Moreover, the sample was highly educated with over 16 years of education on average, again making it less generalizable. Another limitation is that the same size of the AD dementia group was much smaller than that of the other two diagnostic groups (CN and MCI). For that reason, we combined the symptomatic groups together for the analyses that were divided by diagnostic group. An additional limitation is that while the FCI-SF serves as a promising tool for the baby boomer generation, it may no longer be relevant to future generations since the usage of monetary tools and transfers have been more and more transitioned to electronic interfaces. The cur-

rent study did not correct for multiple comparisons; therefore, the results should be interpreted with caution. Future studies need to replicate these findings. Another limitation is that the current study focused on cross-sectional PET and FCI-SF total scores. As mentioned above, we plan on analyzing the longitudinal data as it becomes available. Lastly, the current study focused on the regional cerebral tau burden associations with financial capacity. In future studies using ADNI and other datasets, we would like to determine the association between CSF and plasma tau markers, financial capacity, and other sensitive IADL tests.

In summary, our study showed that greater regional tau burden was modestly associated with financial capacity impairment in early-stage AD. These associations were independent of age, education, sex, memory, and executive function. We also showed that the interaction of regional tau and cortical amyloid was associated with financial capacity changes, even within CN older adults. Future longitudinal analyses will help determine how well AD pathology predicts changes financial capacity at the earliest stages of AD. This in turn will help guide the use of clinically meaningful assessments of IADL, such as the FCI-SF, in upcoming clinical trials in early-stage AD aiming to find better treatments and slowing disease progression.

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Authors' disclosures available online (<https://www.j-alz.com/manuscript-disclosures/20-1122r1>).

## SUPPLEMENTARY MATERIAL

The supplementary material is available in the electronic version of this article: <https://dx.doi.org/10.3233/JAD-201122>.

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