



LJMU Research Online

Jauregi Zinkunegi, A and Bruno, D

Associations between the logical memory test story recall metrics and plasma biomarkers for Alzheimer's disease in individuals free of dementia

<http://researchonline.ljmu.ac.uk/id/eprint/25897/>

Article

Citation (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

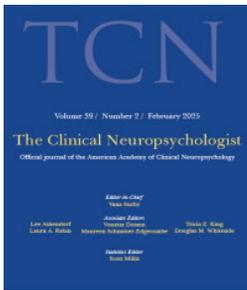
Jauregi Zinkunegi, A and Bruno, D (2025) Associations between the logical memory test story recall metrics and plasma biomarkers for Alzheimer's disease in individuals free of dementia. Clinical Neuropsychologist. ISSN 1385-4046

LJMU has developed **LJMU Research Online** for users to access the research output of the University more effectively. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LJMU Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain.

The version presented here may differ from the published version or from the version of the record. Please see the repository URL above for details on accessing the published version and note that access may require a subscription.

For more information please contact researchonline@ljmu.ac.uk

<http://researchonline.ljmu.ac.uk/>



Associations between the logical memory test story recall metrics and plasma biomarkers for Alzheimer's disease in individuals free of dementia

Ainara Jauregi-Zinkunegi, Rachael E. Wilson, Rebecca E. Langhough, Nicholas J. Ashton, Kaj Blennow, Sterling C. Johnson, Henrik Zetterberg, Davide Bruno & Kimberly D. Mueller

To cite this article: Ainara Jauregi-Zinkunegi, Rachael E. Wilson, Rebecca E. Langhough, Nicholas J. Ashton, Kaj Blennow, Sterling C. Johnson, Henrik Zetterberg, Davide Bruno & Kimberly D. Mueller (20 Mar 2025): Associations between the logical memory test story recall metrics and plasma biomarkers for Alzheimer's disease in individuals free of dementia, The Clinical Neuropsychologist, DOI: [10.1080/13854046.2025.2481119](https://doi.org/10.1080/13854046.2025.2481119)

To link to this article: <https://doi.org/10.1080/13854046.2025.2481119>



© 2025 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group



[View supplementary material](#)



Published online: 20 Mar 2025.



[Submit your article to this journal](#)



Article views: 133



[View related articles](#)



[View Crossmark data](#)

Associations between the logical memory test story recall metrics and plasma biomarkers for Alzheimer's disease in individuals free of dementia

Ainara Jauregi-Zinkunegi^a, Rachael E. Wilson^b, Rebecca E. Langhough^{b,c,d},
Nicholas J. Ashton^e, Kaj Blennow^e, Sterling C. Johnson^{b,c,d,f},
Henrik Zetterberg^{b,e,g,h,i,j}, Davide Bruno^a and Kimberly D. Mueller^{b,k,l}

^aSchool of Psychology, Liverpool John Moores University, Liverpool, UK; ^bWisconsin Alzheimer's Disease Research Center, School of Medicine and Public Health, University of Wisconsin – Madison, Madison, WI, USA; ^cWisconsin Alzheimer's Institute, School of Medicine and Public Health, University of Wisconsin – Madison, Madison, WI, USA; ^dDepartment of Medicine, School of Medicine and Public Health, University of Wisconsin—Madison, Madison, WI, USA; ^eDepartment of Psychiatry and Neurochemistry, Institute of Neuroscience and Physiology, University of Gothenburg, Mölndal, Sweden; ^fGeriatric Research Education and Clinical Center of the Wm. S. Middleton Memorial Veterans Hospital, Madison, WI, USA; ^gClinical Neurochemistry Laboratory, Sahlgrenska University Hospital, Mölndal, Sweden; ^hDepartment of Neurodegenerative Disease, UCL Institute of Neurology, Queen Square, London, UK; ⁱUK Dementia Research Institute at UCL, London, UK; ^jHong Kong Center for Neurodegenerative Diseases, Hong Kong, China; ^kDepartment of Communication Sciences and Disorders, University of Wisconsin – Madison, Madison, WI, USA; ^lWisconsin Alzheimer's Institute, University of Wisconsin School of Medicine and Public Health, University of Wisconsin – Madison, Madison, WI, USA

ABSTRACT

Objective: Blood-based biomarkers are valued for their lower cost and less invasive nature, though issues with widespread implementation and accessibility remain. Process-based scores from story recall have been shown to detect neuronal network disturbances typical of Alzheimer's disease (AD) pathology more effectively than traditional metrics. This study examined the associations between process-based scores and concurrent plasma AD biomarkers in older adults without dementia, while also comparing them to traditional metrics. Additionally, it also investigated the diagnostic utility of these metrics in detecting plasma p-tau217 positivity. **Methods:** Data from 416 participants (mean age = 66.6±7) free of dementia were extracted from the Wisconsin Registry for Alzheimer's Prevention (WRAP). Logical Memory Test (LMT) and plasma p-tau217, p-tau181, p-tau231, Aβ42/Aβ40 ratio, GFAP and NfL levels were analyzed. Bayesian regression models assessed associations between plasma biomarkers and both process-based and traditional LMT scores, controlling for the covariates. **Results:** The best-fitting model for plasma p-tau217 included Total ratio (Tr) and Immediate recall (BF10=573), but Tr showed stronger evidence of association (mean coefficient = 0.208; BFinclusion=14.4) than Immediate recall (mean coefficient=-0.007; BFinclusion=1.7). Tr was also the best predictor of plasma p-tau181 (mean coefficient = 0.144; BF10=10.5) and GFAP (mean coefficient = 0.141; BF10=5.8), outperforming traditional LMT scores. No memory

ARTICLE HISTORY

Received 6 September 2024
Accepted 13 March 2025
Published online 21
March 2025

KEYWORDS

Plasma; Biomarkers;
Alzheimer's disease;
Memory; Story Recall

CONTACT Ainara Jauregi-Zinkunegi ✉ AJauregiZinkunegi@ljmu.ac.uk School of Psychology, Liverpool John Moores University, Liverpool, UK.

Supplemental data for this article can be accessed online at <https://doi.org/10.1080/13854046.2025.2481119>.

© 2025 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited, and is not altered, transformed, or built upon in any way. The terms on which this article has been published allow the posting of the Accepted Manuscript in a repository by the author(s) or with their consent.

scores were associated with plasma p-tau231 or A β 42/40 ratio levels. Tr score was the strongest single predictor of p-tau217 positivity (BF₁₀=38). **Conclusions:** These findings suggest that process-based memory scores might be useful in enhancing the detection of neuronal network disturbances associated with AD pathology, especially in settings where biomarker testing is unavailable.

1. Introduction

Alzheimer's disease (AD) can be diagnosed and/or staged by *in vivo* abnormalities of core biomarkers, using positron emission tomography (PET) scans, cerebrospinal fluid (CSF) tests, and blood-based markers (Jack et al., 2024a). In recent years, there has been increasing interest in blood-based biomarkers, especially due to their potential accessibility, as they carry a lower cost than imaging and are less invasive than lumbar puncture (Karikari et al., 2022). Previous studies have reported reduced plasma levels of the A β 42/40 ratio (Schindler et al., 2019) and increased plasma levels of phosphorylated tau (p-tau; Ashton et al., 2021; Janelidze et al., 2022; Mattsson-Carlgrén et al., 2020; Meyer et al., 2022), glial acidic fibrillary protein (GFAP; Benedet et al., 2021), and neurofilament light (NfL; Benedet et al., 2021) in preclinical AD, when individuals are cognitively unimpaired (Milà-Alomà et al., 2022).

Among plasma biomarkers, p-tau has emerged as the leading option due to its higher diagnostic accuracy and specificity for clinical AD (Ashton et al., 2024). Plasma p-tau181 for example, is associated with tau and amyloid β pathology in the brain and predicts progression to cognitive decline and AD (Karikari et al., 2020, 2022). Similarly, plasma p-tau231 levels can identify amyloid- β positivity, while also reportedly increasing earlier than p-tau181 with underlying tau pathology in cognitively unimpaired (CU) individuals (Ashton et al., 2021). As with plasma p-tau181 and p-tau231, levels of plasma p-tau217 are also associated with amyloid- β and tau pathology (Mattsson-Carlgrén et al., 2021; Palmqvist et al., 2020). However, plasma p-tau217 has shown higher accuracy than other plasma biomarkers in discriminating AD from other neurodegenerative diseases, with comparable performance to CSF p-tau and tau PET biomarkers (Ashton et al., 2024; Janelidze et al., 2023; Palmqvist et al., 2020; Therriault et al., 2023). Furthermore, plasma p-tau217 is capable of detecting changes in preclinical AD (Mattsson-Carlgrén et al., 2020), accurately mirroring the progression of the disease pathology (Ashton et al., 2022).

Nevertheless, plasma testing is yet to be widely implemented (Blennow et al., 2023), while rural communities and remote settings continue to be affected by limited access to diagnostic tools (Anticona Huaynate et al., 2015). Therefore, alternatives to neuroimaging, lumbar puncture, and plasma remain necessary to address the increasing prevalence of dementia in low- and middle-income countries (World Health Organization, 2023). One potential solution is the use of cognitive assessment, which is relatively inexpensive and provides direct information on an individual's cognitive state, but only indirect information about possible underlying neuropathology. Nonetheless, evidence indicates that even subtle cognitive decline may precede the

development of biomarker-positive states, mild cognitive impairment (MCI) or AD (Edmonds et al. 2015), and that cognitive differences can be observed in biomarker-positive individuals who are otherwise cognitively normal (Duke Han et al., 2017). Therefore, identifying early cognitive markers is crucial to accurately monitor progression (Blennow et al., 2023).

Previous studies have investigated the relationship between plasma biomarkers and cognitive function. For example, plasma levels of p-tau181, GFAP, and NfL, have been found to be associated with future cognitive decline in CU individuals (Chatterjee et al., 2023). Other studies have shown that plasma p-tau217 levels correlate with longitudinal cognitive decline in preclinical and prodromal AD (Ashton et al., 2022; Mattsson-Carlgrén et al., 2020), outperforming other plasma biomarkers in CU individuals (Jack et al., 2024b; Mattsson-Carlgrén et al., 2023). Cross-sectionally, a study found that in individuals with and without cognitive impairment, plasma levels of A β 42, A β 42/A β 40, p-tau181, and NfL, were associated with global cognition, memory (delayed recall in word-list recall) and other cognitive domains; and that p-tau181 had higher correlations with these measures than other plasma biomarkers (Xiao et al., 2021). However, a recent meta-analysis revealed that none of the cross-sectional studies of CU individuals they reviewed, found associations between plasma tau biomarkers and cognitive performance; suggesting that associations might depend on the cognitive status of the sample (Garcia-Escobar et al., 2024).

Investigating if cognitive performance is cross-sectionally associated with AD pathology in older CU individuals is especially relevant. AD progression typically begins with a preclinical stage in which individuals may exhibit subtle or no cognitive decline symptoms, yet amyloid deposition and tau pathology may have already begun in certain brain regions (Dubois et al., 2016; Igarashi, 2023). From these regions, the medial temporal lobe (MTL) appears to be crucial (Song et al., 2015), especially the entorhinal cortex, where tau pathology first appears and then spreads to other MTL regions, such as the hippocampus, before progressing to cortical regions in later stages of the disease (Braak & Braak, 1991; Igarashi, 2023). Previous findings support the idea that the entorhinal cortex and hippocampus are essential for memory consolidation (Wixted, 2004; Wixted & Cai, 2013), with episodic memory loss being a key characteristic of AD (Albert et al., 2011; De Simone et al., 2017; De Tollis et al., 2021; Dubois et al., 2007). Furthermore, in preclinical AD, cognitive decline is strongly associated with episodic memory task performance (Aschenbrenner et al., 2024; Lim et al., 2013, 2016; Vos et al., 2013). Thus, the association between plasma biomarker levels and longitudinal cognitive decline observed in previous studies (e.g. Ashton et al., 2022; Chatterjee et al., 2023; Jack et al., 2024b; Mattsson-Carlgrén et al., 2020, 2023) might reflect the impact tau pathology has in memory loss. Plasma biomarkers may serve as peripheral indicators of these early neuropathological changes in the MTL, linking pathology to worse memory performance, even in CU individuals (Mattsson-Carlgrén et al., 2020).

In studies of other biomarkers of AD, such as amyloid and tau PET or CSF, we have shown that process-based scores of episodic memory tests can enrich the detection of *in vivo* AD pathology (Bruno et al., 2021, 2023a, 2023b, 2024a). For example, we demonstrated that loss of primacy recall in stories (i.e. memory for the beginning of

a story), predicts longitudinal PET amyloid load from an unimpaired baseline (Bruno et al., 2021), that loss of recency (i.e. memory for the end of a story) is associated with CSF biomarkers (Bruno et al., 2023a) and can predict amyloid and tau PET positivity (Bruno et al., 2024a), while both loss of primacy and, to a lesser extent, loss of recency, predict biomarker-determined AD cross-sectionally (Bruno et al., 2023b), as measured by CSF levels of the p-tau/A β 42 ratio. The process-based scores examined in these studies are based on the analysis of serial position effects, where patterns of recall are considered alongside “traditional” scores (Bruno et al., 2013; Díaz-Orueta et al., 2018; Grant & Adams, 2009; Talamonti et al., 2020). The serial position curve is a common pattern observed in memory tests, where individuals often recall items from the beginning (primacy) and/or end (recency) of a list better than those in the middle, creating an U-shaped curve (e.g. Murdock, 1962). Even though little is known about how serial position effects relate to plasma biomarkers, in a recent unpublished study, Bruno et al. (2024b) found that serial position scores derived from word-list recall, and especially delayed primacy, are cross-sectionally associated with plasma p-tau231 levels in CU individuals. However, this study did not investigate the association with plasma p-tau217, and considering the high diagnostic accuracy and predictive ability of this biomarker (Ashton et al., 2024; Jack et al., 2024b; Mattsson-Carlgrén et al., 2023; Palmqvist et al., 2020), the examination of their potential link is promising. Additionally, whether process-based scores derived from story recall are also linked to plasma biomarkers of AD and how they compare to traditional scores is unknown.

In the current study, we examined whether process-based scores derived from the Logical Memory Task, a test of episodic memory, were associated to cross-sectional plasma biomarkers of AD, specifically p-tau217, p-tau181, p-tau231, A β 42/A β 40 ratio, GFAP, and NFL, in a cohort of older adults free of dementia. We also explored whether these scores outperformed traditional scores of the same test. Moreover, considering that the most recent guidelines for diagnosing and staging AD include plasma p-tau217 as a core biomarker (Jack et al., 2024a), due to its higher diagnostic accuracy and specificity for clinical AD (Ashton et al., 2024), further analyses were carried out with this biomarker. In particular, we investigated the ability of process-based and traditional scores in detecting plasma p-tau217 positivity and the diagnostic accuracy of these scores. Based on previous findings with other biomarkers of AD (Bruno et al., 2021, 2023a, 2023b, 2024a), we hypothesized process-based scores of story recall would be associated with cross-sectional plasma biomarkers of AD, outperforming traditional metrics, while also showing high diagnostic accuracy.

2. Methods

2.1. Participants

Data were drawn from the Wisconsin Registry of Alzheimer’s Prevention (WRAP), an ongoing longitudinal cohort study based at the University of Wisconsin–Madison, USA. Participants are volunteers who were recruited through advertisements, word of mouth, or memory clinics where a parent was diagnosed or treated; yet those with a dementia diagnosis or evidence of dementia at enrollment are excluded. Participants attend regular visits, the first follow-up occurs 2 to 4 years after baseline and then

every 2 years (for details, see Johnson et al., 2018). To be included in the analysis, participants had to have measures of plasma samples and story recall data, derived from the LMT (Wechsler, 1987). In cases where multiple plasma samples were available for a participant, we selected the most recent sample and the corresponding cognitive assessment. Typically, blood extraction and cognitive assessment were concurrent, yet there were some exceptions. Participants were excluded if the time between the blood extraction and cognitive assessment exceeded one year. In addition, participants had to be classified as cognitively unimpaired (stable or declining) or with mild cognitive impairment at cognitive assessment visit. Cognitive statuses were assessed using a two-tiered consensus conference approach (for details, see Johnson et al., 2018; Langhough Kosciak et al., 2021), based on core clinical criteria developed by the National Institute on Aging and the Alzheimer's Association (Albert et al., 2011; McKhann et al., 2011). All in all, these criteria left us with 416 individuals, means and standard deviations of demographic variables are reported in Table 1. Among the 416 participants, 360 were classified as cognitively unimpaired stable, 42 as cognitively unimpaired declining, and 14 with mild cognitive impairment, at cognitive assessment visit. Of these 416 participants, five (1.20%) reported their race as American Indian or Alaska Native, one (0.24%) as Asian, 10 (2.40%) as Black or African American, 397 (95.43%) as White, and three (0.72%) as unknown. Some participants included in this study were also included in previous publications from our group (Bruno et al., 2021, 2023a, 2023b, 2024a), yet these studies focused on either PET or CSF biomarkers, and not plasma biomarkers. All activities for this study were approved by the ethics committees of the authors' universities and conducted in accordance with the Declaration of Helsinki. All participants provided informed consent prior to testing.

2.2. Cognitive assessment

The Logical Memory Task (LMT) is a subtest of the Wechsler Memory Scale Revised (WMS-R; Wechsler, 1987), and comprises two stories ("A" and "B"), each with 25 items

Table 1. Mean and standard deviation (or median and minimum-maximum for cognitive visit number) of the study variables.

	Total (n=416)	CUS (n=360)	CUD (n=42)	MCI (n=14)
Age at blood extraction	66.60 (6.8)	66.13 (6.8)	68.20 (6.2)	73.83 (3.5)
Elapsed time (years)	0.00 (0.0)	0.0 (0.0)	0.00 (0.0)	0.03 (0.1)
APOE Risk score	1.17 (0.7)	1.15 (0.7)	1.21 (0.8)	1.65 (1.1)
Education (years)	16.60 (2.8)	16.65 (2.9)	16.33 (2.5)	16.07 (2.5)
Cognitive visit number	6 (1–7)	6 (1–7)	6 (2–7)	6 (4–7)
Plasma p-tau217	0.50 (0.4)	0.45 (0.3)	0.63 (0.4)	1.29 (0.8)
Plasma p-tau181	2.68 (1.4)	2.55 (1.2)	3.26 (1.9)	4.30 (1.4)
Plasma p-tau231	12.0 (5.2)	11.7 (5.1)	13.4 (5.3)	16.4 (4.3)
Plasma Aβ42/40	0.07 (0.0)	0.07 (0.0)	0.07 (0.0)	0.06 (0.0)
Plasma GFAP	119 (67.8)	113 (63.5)	131 (70.8)	224 (78.4)
Plasma NfL	22.1 (16.3)	20.8 (11.7)	30.1 (36.9)	30.0 (10.2)
LMT Immediate recall	14.55 (3.4)	15.18 (3.03)	10.70 (3.0)	9.71 (3.2)
LMT Delayed recall	13.21 (3.9)	13.99 (3.3)	8.77 (3.1)	6.43 (3.2)
LMT Recency ratio	1.05 (0.2)	1.03 (0.1)	1.15 (0.4)	1.21 (0.3)
LMT Total ratio	1.13 (0.2)	1.10 (0.1)	1.24 (0.2)	1.57 (0.5)
LMT Primacy ratio	1.22 (0.3)	1.18 (0.3)	1.34 (0.3)	1.72 (0.5)

Note. CUS: cognitively unimpaired stable; CUD: cognitively unimpaired declining; MCI: mild cognitive impairment; APOE: apolipoprotein E; p-tau: phosphorylated tau; Aβ: amyloid-beta; GFAP: glial fibrillary acidic protein; NfL: neurofilament light chain; LMT: Logical Memory Task. Plasma biomarker levels in pg/mL.

“idea units”). Each story is read aloud to the participant, and then the participant is asked to recall each story immediately after presentation, and again after a 25–30 min delay. Participants are free to recall the items in any order they prefer. Scoring procedures from the WMS-R manual were applied. Although the scoring criteria permits some alteration from the original item (for a hypothetical example, “slid off the table” is allowed instead of “fell off the table”), certain items must be recalled verbatim, e.g. numerical expressions or proper names. Two conventional clinical metrics were extracted from LMT (averaging over A and B): LMT Immediate recall, derived from the total number of idea units recalled immediately after learning the story; and LMT Delayed recall, derived from the total number of idea units recalled after the delay. The reliability of Immediate and Delayed recall has been reported as .76 and .74, respectively (The Psychological Corporation, 1997), and as large practice effects have been found with this test (Lo et al., 2012), the number of visits was also accounted for in the statistical analyses. Process scores were primacy ratio, indexing primacy forgetting of idea units from immediate to delayed recall (Pr; Bruno et al., 2021); recency ratio, indexing recency forgetting of idea units from immediate to delayed recall (Rr; Bruno et al., 2018); and total ratio, which indexes overall forgetting of idea units from immediate to delayed recall (Tr; Bruno et al., 2021; Jauregi Zinkunegi et al., 2024). Primacy and recency were defined as the first and final eight idea units of the story (out of 25) respectively, as in previous studies (Bruno et al., 2021, 2023a, 2023b). All three scores were calculated by dividing immediate performance by delayed performance, after applying $a+1$ correction at each term to compensate for possible 0 scores; thus, higher numbers indicate more forgetting. Participants’ cognitive data were taken from whichever visit was closest to the plasma visit. Among the 416 participants, all had concurrent cognitive assessment to plasma visit except six, and to account for this time difference, elapsed times were calculated and controlled for in the statistical analyses.

2.3. Blood biomarkers

Plasma samples were analyzed at the Department of Psychiatry and Neurochemistry, University of Gothenburg (see Ashton et al., 2024 for details). Plasma amyloid-beta (A β) 42/40, glial fibrillary acidic protein (GFAP), and neurofilament light chain (NfL) were quantified using the Simoa[®] Human Neurology 4-Plex E assay. Plasma phosphorylated tau (p-tau) 231 was measured using Simoa[®] assays developed at the University of Gothenburg, while plasma p-tau181 was quantified using the commercial Advantage V2.1 kit. Plasma p-tau 217 levels were measured using the Simoa[®] ALZpath p-tau 217 assay. All plasma levels were analyzed using Quanterix’s HD-X[™] Analyzer. Plasma p-tau217 positivity was determined using the clinical cutoff of 0.42 pg/mL (Ashton et al., 2024), which was derived to optimally distinguish individuals with amyloid-PET positivity from those without. Positivity indicates a high likelihood of underlying amyloid pathology.

2.4. Genotyping

DNA was extracted from whole blood. Samples were aliquoted on 96-well plates for determination of apolipoprotein E (APOE) genotypes. An APOE risk score was calculated

based on the odds ratios of the $\epsilon 2/\epsilon 3/\epsilon 4$ genotype, as previously reported (McKhann et al., 2011).

2.5. Statistical analyses

Partial correlations, controlling for age at blood draw, gender, years of education, *APOE* risk score, elapsed time between cognitive assessment and blood extraction, and cognitive visit number, were used to explore the relationship between the LMT scores and plasma biomarker levels. Steiger's Z tests (Steiger, 1980) were conducted on partial correlation coefficients between any two significant predictors, to determine if the strength of one association between one memory score and each biomarker was stronger than the association between another memory score and the same biomarker, by using a calculator (<http://quantpsy.org>; Lee & Preacher, 2013). We adjusted for multiple testing using a false discovery rate-based approach (FDR; Benjamini & Hochberg, 1995) for all the comparisons, corrected across biomarkers.

To examine whether process-based and traditional scores of story recall were cross-sectionally associated to plasma biomarkers of AD, and whether process-based scores outperformed traditional scores of the same test, Bayesian linear regression analyses were carried out. These analyses were conducted separately with plasma p-tau217, p-tau231, p-tau181, GFAP, NfL, and A β 42/40 ratio as outcomes; for each outcome, memory scores, including traditional metrics and serial position scores, were included as predictors (individually or in every possible combination of LMT scores). Age at blood draw, gender, years of education, *APOE* risk score, elapsed time between cognitive assessment and blood extraction, and visit number to account for practice effects, were used as control variables, forming the *null model*. The posterior summary was calculated based on the best model and credible intervals (CIs) were set to 95%. The prior was set to the default JZS, and the model prior was set to Uniform. One thousand Markov chain Monte-Carlo simulations were conducted to determine parameters and compensate for possible violations of normality, but we also evaluated q-q plots of residuals to estimate normality.

We then specifically focused our analyses on plasma p-tau217, to determine if process-based and traditional scores can detect a positive result, based on the clinical cutoff of 0.42 pg/mL (Ashton et al., 2024), and discriminate between p-tau217 positive and negative individuals, in the same sample. Bayesian logistic regression analyses were carried out to identify the best predictors of plasma p-tau217 positivity, binary plasma p-tau217 status (negative vs. positive) was included as outcome, traditional and process-based scores were included as predictors, and the same covariates as above. Next, the memory scores that emerged as the strongest predictors of plasma p-tau217 positivity in the logistic regression were examined further using separate Bayesian Analysis of Covariance (ANCOVAs). In these models, the identified memory scores were included as dependent variables, binary plasma p-tau217 status as independent variable, and the same covariates as in previous analyses.

In addition, to compare the diagnostic performance of these scores, classification analyses were conducted using a fixed probability threshold of 0.5 to ensure consistency. Diagnostic performance was evaluated using sensitivity (i.e. hit, or true positive, rate), specificity (i.e. correct rejection, or true negative, rate), positive predictive value

(PPV; i.e. the probability that a positive response is a hit rather than a false alarm, i.e. a false positive), and negative predictive value (NPV; i.e. the probability that a negative response is a correct rejection rather than a miss, i.e. a false negative). The area under the receiver operating characteristic curve (AUC) was calculated for each score to assess their ability to discriminate between plasma p-tau217 negativity and positivity. Analyses were conducted using JASP (0.18.3; <https://jasp-stats.org/>).

3. Results

Table 1 reports demographic variables, memory scores, and plasma levels, by whole sample and by diagnosis at cognitive assessment visit. Visual inspection of q-q plots suggested that plasma p-tau217, p-tau231, p-tau181, GFAP, NfL and A β 42/40 ratio displayed some degree of non-normality, so we applied log10 transformations, which gave us more linear q-q plots.

3.1. Partial correlations

Partial correlations indicated LMT scores significantly correlated with plasma p-tau217, p-tau181, GFAP, and NfL, all log10 transformed; see **Table 2** for partial correlation coefficients. Steiger's Z-tests, corrected for multiple comparisons with FDR, showed that for p-tau217, the association with Tr was significantly stronger than with Immediate recall ($Z=4.42$; unadjusted- $p < .001$; adjusted- $p = .001$), Delayed recall ($Z=4.26$; unadjusted- $p < .001$; adjusted- $p = .001$), Pr ($Z=2.27$; unadjusted- $p = .023$; adjusted- $p = .034$) and Rr ($Z=2.25$; unadjusted- $p = .024$; adjusted- $p = .034$). For plasma p-tau181, the association with Tr was significantly stronger than with Delayed recall ($Z=2.81$; unadjusted- $p = .005$, adjusted- $p = .012$), but not than with Rr ($Z=0.13$; unadjusted- $p = .893$; adjusted- $p = .893$). For plasma GFAP, the association with Tr was not significantly stronger than with Pr ($Z=0.92$; unadjusted- $p = .360$; adjusted- $p = .420$), while for plasma NfL, a significant association was only found with Delayed recall scores. See **Figure S1** in **Supplementary materials** for scatterplots of the strongest associations.

3.2. Cross-sectional associations with plasma biomarker levels

Plasma p-tau217. The linear regression analysis showed that the best fitting model ($BF_{10} = 573.58$, extreme evidence) included two predictors: Tr ($BF_{inclusion} = 14.44$) and Immediate recall ($BF_{inclusion} = 1.74$). $BF_{inclusion}$ values indicate how much more likely the

Table 2. Partial correlations between plasma biomarkers and Logical Memory Task scores.

Variable	p-tau217	p-tau231	p-tau181	A β 42/40	NfL	GFAP
Immediate recall	-0.144**	-0.011	-0.073	.073	-0.096	-0.042
Delayed recall	-0.172***	-0.023	-0.105*	.060	-0.103*	-0.070
Primacy ratio	.116*	.006	.069	-0.048	.033	.098*
Recency ratio	.097*	.058	.133**	.001	.049	.067
Total ratio	.197***	.069	.139**	-0.013	.057	.131**

Note. $N=416$. Control variables: gender, education years, apolipoprotein E (APOE) risk score, age at blood extraction, cognitive visit number, elapsed time between blood extraction and cognitive assessment. p-tau: phosphorylated tau; A β : amyloid-beta; NfL: neurofilament light chain; GFAP: glial fibrillary acidic protein. * $p < .05$, ** $p < .01$, *** $p < .001$. Bold: indicates the stronger partial coefficients in the column after correcting for multiple comparisons.

data are under a model that includes a given variable compared to a model without it. For example, the model with Tr is 14.4 times more likely than the model with only the covariates, while the model with Immediate recall is 1.7 times more likely than the model with covariates alone. Cross-sectionally, higher Tr scores, representing more forgetting, were associated with increased plasma p-tau217 levels (mean coefficient = 0.208, SD = 0.062, CIs 0.086 to 0.331), and higher Immediate recall scores, reflecting better recall, were associated with decreased plasma p-tau217 levels (mean coefficient = -0.007, SD = 0.003, CIs -0.014 to 0.000).

As the dependent variable was log10 transformed, the mean coefficients were exponentiated with base 10 to obtain the multiplicative factor on the original scale (Benoit, 2011), this calculation was also carried out with credible intervals. Cross-sectionally, a one-unit increase in Tr corresponded to 61.4% higher plasma p-tau217 levels, with credible intervals ranging from 21.9% to 114.3%, whereas one-unit decrease in Immediate recall corresponded to 1.6% higher plasma p-tau217 levels, with credible intervals ranging from -3.2% to 0%. However, the scales of Tr and Immediate recall are not directly comparable due to how each scores are calculated (for details, see section 2.2). For example, an individual with an Immediate recall score of 13, approximately half of the 25 items, would have 21% higher plasma p-tau217 levels than an individual who recalled all the items, i.e. had a score of 25. On the other hand, an individual with a Tr score of 2, who forgot half of the items from the immediate to the delayed trial, would have 61% higher plasma p-tau217 levels than an individual who did not forget any, and thus, had a Tr score of 1.

Plasma p-tau181. The linear regression analysis yielded a best fitting model with Tr alone ($BF_{10} = 10.54$, strong evidence; $BF_{inclusion} = 1.40$). Cross-sectionally, higher Tr scores were associated with increased plasma p-tau181 levels (mean coefficient = 0.144, SD = 0.053, CIs 0.040 to 0.247). As the dependent variable was log10 transformed, the mean coefficient and credible intervals were exponentiated, as explained above. Cross-sectionally, a one-unit increase in Tr corresponded to 39.3% higher plasma p-tau181 levels, with credible intervals ranging from 9.6% to 76.6%.

Plasma p-tau231. The linear regression analysis showed that the *null model* was the best fitting model.

Plasma GFAP. The linear regression analysis yielded a best fitting model with Tr alone ($BF_{10} = 5.82$, moderate evidence; $BF_{inclusion} = 2.56$). Cross-sectionally, higher Tr scores were associated with increased plasma GFAP levels (mean coefficient = 0.141, SD = 0.054, CIs 0.035 to 0.247). After exponentiating the mean coefficients, a one-unit increase in Tr corresponded to 38.4% higher plasma GFAP levels, with credible intervals ranging from 8.4% to 76.6%.

Plasma NFL. The linear regression analysis yielded a best fitting model with Delayed recall ($BF_{10} = 1.49$, anecdotal evidence; $BF_{inclusion} = 0.70$).

Plasma A β 42/40 ratio. The linear regression analysis showed that the *null model* was the best fitting model.

3.3. Additional analyses with plasma p-tau217

To identify the best predictors of plasma p-tau217 positivity, we ran Bayesian logistic regression analyses. The logistic regression analysis (164 of 416 individuals were

classified as p-tau217 positive; 39%) showed that the best fitting model ($BF_{10} = 79.69$, very strong evidence) included three predictors: Tr ($BF_{inclusion} = 9.59$), Immediate recall ($BF_{inclusion} = 1.78$) and Delayed recall ($BF_{inclusion} = 1.58$). $BF_{inclusion}$ values indicate that model odds increase when including either variable 9.6, 1.8 or 1.6 times, respectively. The second-best model included Tr alone ($BF_{10} = 38.43$, very strong evidence). See [Figure 1](#) for a plot of the probability of being classified as plasma p-tau217 positive versus LMT Tr, Immediate and Delayed recall scores.

To investigate whether there was evidence for differences between plasma p-tau217- and p-tau217+ groups in the identified memory scores, while also controlling for the covariates, we ran separate Bayesian ANCOVAs with Tr, Immediate and Delayed recall scores, as dependent variables. There was strong evidence that Tr scores were influenced by plasma p-tau217 classification ($BF_{10} = 10.28$), and a post-hoc comparison showed that Tr scores discriminated between p-tau217- and p-tau217+ individuals ($BF_{10} = 566.97$; extreme evidence). There was anecdotal evidence that Delayed recall scores were influenced by p-tau217 classification ($BF_{10} = 2.14$), yet a post-hoc comparison showed that Delayed recall scores discriminated between groups ($BF_{10} = 25.49$, strong evidence). These analyses also showed Immediate recall scores were not influenced by p-tau217 classification ($BF_{10} = 0.58$), as the null model was better.

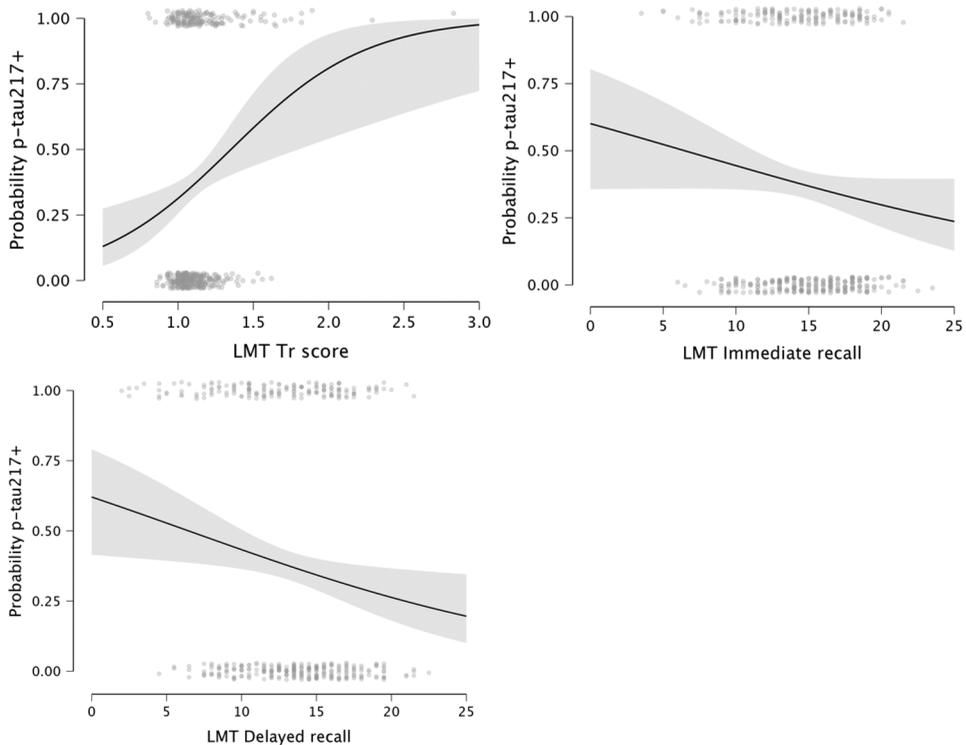


Figure 1. Plots of the probability of being classified as plasma phosphorylated tau (p-tau) 217 positive versus Logical Memory Task Total ratio, Immediate and Delayed recall scores, with 95% confidence interval, $N=416$.

Finally, to compare the diagnostic performance of the three metrics, classification analyses were performed using a fixed probability threshold of 0.5. Tr score had a sensitivity of 50.0%, specificity of 85.7%, PPV of 69.5%, NPV of 72.5% and AUC value of 0.733, indicating good discriminatory ability. Immediate recall showed a sensitivity of 47.0%, specificity of 85.7%, PPV of 68.1%, NPV of 71.3%, and an AUC of 0.725. Similarly, Delayed recall scores showed a sensitivity of 47.0%, specificity of 83.7%, PPV of 65.3%, NPV of 70.8%, and AUC of 0.726.

4. Discussion

In this study, we examined whether process-based scores in story recall were associated with cross-sectional plasma biomarkers of AD in a cohort of older adults free of dementia, while also comparing their sensitivity to that of traditional scores of the same test. In addition, considering that among blood-based biomarkers, plasma p-tau217 has shown higher diagnostic accuracy and specificity for clinical AD (Ashton et al., 2024), further analyses were carried out to determine the ability of story recall metrics in detecting plasma p-tau217 positivity and the diagnostic accuracy of these scores. We hypothesized that process-based scores of story recall would be associated with cross-sectional plasma biomarkers of AD, outperforming traditional metrics, while also showing high diagnostic accuracy. Our results showed that story recall scores were cross-sectionally associated with plasma p-tau217, p-tau181 and GFAP levels in this population. Process-based scores, particularly the Total ratio, which is an index of total forgetting, outperformed traditional LMT scores such as Immediate and Delayed recall. However, we did not find evidence for associations between story recall metrics and plasma p-tau231, NfL or A β 42/40 ratio levels. Specific analyses with plasma p-tau217 status, based on a clinical cutoff of this biomarker (Ashton et al., 2024), indicated that while Delayed recall and Tr scores discriminated between p-tau217- and p-tau217+ individuals, Tr was the strongest single predictor and showed better diagnostic performance.

Past studies have shown plasma biomarkers are associated with longitudinal cognitive decline in CU individuals (Chatterjee et al., 2023; Jack et al., 2024b), and in preclinical and prodromal AD (Ashton et al., 2022; Mattsson-Carlgrén et al., 2020, 2023). However, findings on cross-sectional associations have been mixed, while studies with mixed samples found associations between cognitive tests and plasma A β 42/40 and/or p-tau181 (Karikari et al., 2020; Sun et al., 2022; Tsai et al., 2019; Wang et al., 2021; Weigand et al., 2023; Xiao et al., 2021), some studies of CU individuals have not (Aschenbrenner et al., 2024; Snellman et al., 2023). Contradictory findings have been suggested to be due to differences in study populations and the cognitive tests used, which might not be sensitive enough to detect the subtle cognitive changes occurring in unimpaired individuals (García-Escobar et al., 2024). One way to improve the sensitivity of cognitive tests is to implement an item-level analysis of cognitive responses (Bruno et al., 2024c), which aims at identifying the underlying neurocognitive processes leading to cognitive performance (Mueller et al., 2022). An example of this approach is the analysis of serial position effects, which has been shown to enhance the detection of *in vivo* AD pathology, as measured by amyloid and tau PET

or CSF (Bruno et al., 2021, 2023a, 2023b, 2024a; Jauregi-Zinkunegi et al., 2025), even in samples comprised of CU individuals only.

The findings presented here provide evidence for cross-sectional associations between plasma biomarkers and story recall, especially for process-based scores derived from serial position analysis. Consistent with previous studies, the regression analyses show strong associations between memory scores and concurrent p-tau biomarkers, specifically p-tau217 and p-tau181. However, in contrast to others (Sun et al., 2022; Xiao et al., 2021), we found no associations with plasma A β 42/40 ratio levels. Notably, most studies indicate that plasma p-tau biomarkers have stronger associations with cognition than other plasma biomarkers (for a review, see Garcia-Escobar et al., 2024). Additionally, our results offer novel evidence supporting the utility of process-based scores, specifically Total ratio, in relation to plasma p-tau217, p-tau181, and GFAP biomarker levels, as they outperformed traditional scores of the same test; thus, extending previous findings involving other biomarkers of AD.

To be certain current results are also found in individuals who are cognitively unimpaired, especially considering the contradictory findings reported depending on cognitive status, we carried out a post hoc analysis excluding those with MCI at cognitive assessment visit ($N=14$). Analyses showed that with plasma p-tau217 levels as outcome, Tr remained the strongest single predictor, while the model with Rr was the strongest for plasma p-tau181 levels, followed by Tr (for details, see Tables S1 and S2 in Supplementary materials); yet the null model was better for all other biomarkers. Moreover, when inspecting all regression models, analyses show neither Immediate nor Delayed recall scores alone were cross-sectionally associated with plasma p-tau181 levels, while with plasma p-tau217, the evidence for associations with traditional scores was none or anecdotal. These results indicate that not only process-based scores of story recall outperform traditional scores of the same test in older individuals free of dementia, but also remain associated with plasma p-tau biomarkers in CU individuals, whereas traditional scores do not.

Overall, it could be argued that process-based scores derived from story recall might be more strongly associated with plasma biomarkers of AD than traditional metrics of the same test, especially in older individuals without cognitive impairment. While current findings suggest process-based scores might aid in the early detection of AD, further research, and particularly longitudinal studies, are needed to confirm and expand these results.

Considering the advantages p-tau217 offers over other plasma biomarkers, as described before and elsewhere (Ashton et al., 2022, 2024; Mattsson-Carlgrén et al., 2020, 2021, 2023, Palmqvist et al., 2020), whether story recall-derived measures also predict plasma p-tau217 status was investigated using a clinical cutoff from a previous study of WRAP data (Ashton et al., 2024). Results from logistic regression and ANCOVA analyses revealed Total ratio showed the strongest association with plasma p-tau217 positivity, and provided the strongest evidence for discriminating between p-tau217- and p-tau217+ groups. These findings suggest this process-based score of story recall reflects the differences in memory performance that might be vulnerable to tau pathology. Considering that early pathological changes appear to begin in the MTL,

which has a key role in episodic memory, the association and discriminative ability of Tr to plasma p-tau217 positivity may reflect the disruptions already occurring in encoding, consolidation and retrieval processes, in individuals who are free of dementia. In contrast, the weaker associations with Delayed recall, or the lack of discriminative ability of Immediate recall, suggest simpler recall measures might not be able to fully capture pathological changes in this population. As for diagnostic performance, results indicated that although Tr, Immediate and Delayed recall scores had modest sensitivity, they had high specificity, with Tr for example, correctly identifying plasma p-tau217– individuals approximately 85% of the time. The positive and negative predictive values of the three scores were modest, approximately 70%, with Tr showing the highest and Delayed recall the lowest, yet all three showed a reasonable balance between correctly identifying p-tau217+ and excluding p-tau217– individuals, making it particularly valuable in screening or early detection in clinical and research settings.

To be certain these results are also found in individuals who are CU, we carried out a *post hoc* analysis in which individuals with MCI were excluded. Analyses showed that Tr remained the strongest predictor of plasma p-tau217 status (see [Table S3](#)), while also being able to discriminate between negative and positive individuals, whereas Immediate and Delayed recall scores did not (see [Table S4](#) in Supplementary materials). Furthermore, if among cognitive unimpaired individuals, only those who were stable were included in the analyses ($N=360$), and those declining were excluded, results indicated Tr was still the strongest single predictor of plasma p-tau217 status, while also discriminating between negative and positive individuals, albeit anecdotally, but traditional scores did not (see [Tables S5 and S6](#) in Supplementary materials). Overall, current findings emphasize the early vulnerability of episodic memory to tau pathology, even in CU individuals, and highlight the utility of measures like Tr to detect these changes, which should be considered along traditional scores to maximize their predictive ability.

A potential limitation of the current study is that most of the sample consisted of individuals who identified as non-Hispanic and white (95.43%), and as a result, other races and ethnicities were underrepresented. Considering the importance of including a more diverse range of ethnicities and backgrounds in AD research (Manly et al., 2021; Morris et al., 2019), we believe future research should consider exploring whether the patterns observed in the current study would also apply to a more diverse sample. Another limitation is that even though we initially planned to assess the diagnostic accuracy of story recall metrics in relation to other plasma biomarkers too, we ultimately decided against it due to the lack of established cutoff values within this cohort. Given that cutoffs vary based on analytical methods and reference populations, current recommendations advise establishing population-specific thresholds to ensure interpretability (Pais et al., 2023). Additionally, although we report cross-sectional associations between story recall-derived scores and plasma biomarkers, we did not analyze repeated measurements across visits. Future studies should consider investigating whether process-based scores of story recall also predict plasma biomarker levels longitudinally.

In summary, this cross-sectional study showed that in individuals free of dementia, story recall-derived memory scores, and particularly Total ratio, a measure of total

forgetting, are associated with plasma p-tau217, p-tau181 and GFAP levels, but not with plasma p-tau231, NfL or A β 42/40 ratio levels. Furthermore, Total ratio was also the strongest predictor of plasma p-tau217 positivity, outperforming traditional metrics of story recall. These findings showed that tracking forgetting in LMT, as opposed to simply measuring total recall performance, is a better option when predicting plasma p-tau217 in older adults free of dementia. We believe process-based scores of story recall can be particularly useful in settings without biomarker testing or in combination with plasma biomarkers to detect clinically relevant brain A β and tau pathology.

Acknowledgments

We would like to acknowledge WRAP and WADRC study participants and staff for their invaluable contributions to this research. We also acknowledge the entire staff of the Cognitive-Communication in Aging and Neurogenic Disorders Laboratory (CCANDL) at University of Wisconsin – Madison for their efforts in coding and managing the data for this study.

Disclosure statement

HZ has served at scientific advisory boards and/or as a consultant for Abbvie, Acumen, Alector, Alzinova, ALZPath, Amylyx, Annexon, Apellis, Artery Therapeutics, AZTherapies, Cognito Therapeutics, CogRx, Denali, Eisai, LabCorp, Merry Life, Nervgen, Novo Nordisk, Optoceutics, Passage Bio, Pinteon Therapeutics, Prothena, Red Abbey Labs, reMYND, Roche, Samumed, Siemens Healthineers, Triplet Therapeutics, and Wave, has given lectures in symposia sponsored by Alzecure, Biogen, Cellectricon, Fujirebio, Lilly, Novo Nordisk, and Roche, and is a co-founder of Brain Biomarker Solutions in Gothenburg AB (BBS), which is a part of the GU Ventures Incubator Program (outside submitted work).

Funding

This secondary analysis of WRAP data was funded by a NIH-NIA (R01AG07940-01) grant to KDM, in which DB and REL are co-investigators. Primary data collection for WRAP data was supported by NIH-NIA grants 5R01AG027161-13 and 5R01AG021155-18 on which SCJ is principal investigator and REL is co-investigator, and data from the Wisconsin Alzheimer's Disease Research Center Clinical Core was supported by NIH-NIA grant 5P50AG033514-07. HZ is a Wallenberg Scholar and a Distinguished Professor at the Swedish Research Council supported by grants from the Swedish Research Council (#2023-00356; #2022-01018 and #2019-02397), the European Union's Horizon Europe research and innovation programme under grant agreement No 101053962, Swedish State Support for Clinical Research (#ALFGBG-71320), the Alzheimer Drug Discovery Foundation (ADDF), USA (#201809-2016862), the AD Strategic Fund and the Alzheimer's Association (#ADSF-21-831376-C, #ADSF-21-831381-C, #ADSF-21-831377-C, and #ADSF-24-1284328-C), the European Partnership on Metrology, co-financed from the European Union's Horizon Europe Research and Innovation Programme and by the Participating States (NEuroBioStand, #22HLT07), the Bluefield Project, Cure Alzheimer's Fund, the Olav Thon Foundation, the Erling-Persson Family Foundation, Familjen Rönströms Stiftelse, Stiftelsen för Gamla Tjänarinnor, Hjärnfonden, Sweden (#FO2022-0270), the European Union's Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie grant agreement No 860197 (MIRIADE), the European Union Joint Programme – Neurodegenerative Disease Research (JPND2021-00694), the National Institute for Health and Care Research University College London Hospitals Biomedical Research Center, and the UK Dementia Research Institute at UCL (UKDRI-1003).

Data availability

WRAP data can be requested from: <https://wrap.wisc.edu/data-requests-2/>. Data will be released to internal and external investigators following confirmation of IRB approval together with an evaluation of scientific merit and resource availability.

References

- Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., Gamst, A., Holtzman, D. M., Jagust, W. J., Petersen, R. C., Snyder, P. J., Carrillo, M. C., Thies, B., & Phelps, C. H. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging- Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 7(3), 270–279. <https://doi.org/10.1016/j.jalz.2011.03.008>
- Anticona Huaynate, C. F., Pajuelo Travezaño, M. J., Correa, M., Mayta Malpartida, H., Oberhelman, R., Murphy, L. L., & Paz-Soldan, V. A. (2015). Diagnostics barriers and innovations in rural areas: Insights from junior medical doctors on the frontlines of rural care in Peru. *BMC Health Services Research*, 15(1), 454. <https://doi.org/10.1186/s12913-015-1114-7>
- Aschenbrenner, A. J., Hassenstab, J. J., Schindler, S. E., Janelidze, S., Hansson, O., Morris, J. C., & Grober, E. (2024). Free recall outperforms story recall in associations with plasma biomarkers in preclinical Alzheimer disease. *The Journal of Prevention of Alzheimer's Disease*, 11(6), 1696–1702. <https://doi.org/10.14283/jpad.2024.130>
- Ashton, N. J., Pascoal, T. A., Karikari, T. K., Benedet, A. L., Lantero-Rodriguez, J., Brinkmalm, G., Snellman, A., Schöll, M., Troakes, C., Hye, A., Gauthier, S., Vanmechelen, E., Zetterberg, H., Rosa-Neto, P., & Blennow, K. (2021). Plasma p-tau231: A new biomarker for incipient Alzheimer's disease pathology. *Acta Neuropathologica*, 141(5), 709–724. <https://doi.org/10.1007/s00401-021-02275-6>
- Ashton, N. J., Janelidze, S., Mattsson-Carlgen, N., Binette, A. P., Strandberg, O., Brum, W. S., Karikari, T. K., González-Ortiz, F., Di Molfetta, G., Meda, F. J., Jonaitis, E. M., Kosciak, R. L., Cody, K., Betthausen, T. J., Li, Y., Vanmechelen, E., Palmqvist, S., Stomrud, E., Bateman, R. J., ... Hansson, O. (2022). Differential roles of A β 42/40, p-tau231, and p-tau217 for Alzheimer's trial selection and disease monitoring. *Nature Medicine*, 28(12), 2555–2562. <https://doi.org/10.1038/s41591-022-02074-w>
- Ashton, N. J., Brum, W. S., Di Molfetta, G., Benedet, A. L., Arslan, B., Jonaitis, E., Langhough, R. E., Cody, K., Wilson, R., Carlsson, C. M., Vanmechelen, E., Montoliu-Gaya, L., Lantero-Rodriguez, J., Rahmouni, N., Tissot, C., Stevenson, J., Servaes, S., Therriault, J., Pascoal, T., ... Zetterberg, H. (2024). Diagnostic accuracy of a plasma phosphorylated tau 217 immunoassay for Alzheimer disease pathology. *JAMA Neurology*, 81(3), 255–263. <https://doi.org/10.1001/jamaneurol.2023.5319>
- Benedet, A. L., Milà-Alomà, M., Vrillon, A., Ashton, N. J., Pascoal, T. A., Lussier, F., Karikari, T. K., Hourregue, C., Cognat, E., Dumurgier, J., Stevenson, J., Rahmouni, N., Pallen, V., Poltronetti, N. M., Salvadó, G., Shekari, M., Operto, G., Gispert, J. D., Minguillon, C., ... Suárez-Calvet, M. (2021). Differences between plasma and cerebrospinal fluid glial fibrillary acidic protein levels across the Alzheimer disease continuum. *JAMA Neurology*, 78(12), 1471–1483. <https://doi.org/10.1001/jamaneurol.2021.3671>
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society Series B: Statistical Methodology*, 57(1), 289–300. <https://doi.org/10.1111/j.2517-6161.1995.tb02031.x>
- Benoit, K. (2011). Linear regression models with logarithmic transformations. *London School of Economics*, 22(1), 23–36.
- Blennow, K., Galasko, D., Perneczky, R., Quevenço, F. C., van der Flier, W. M., Akinwonmi, A., Carboni, M., Jethwa, A., Suridjan, I., & Zetterberg, H. (2023). The potential clinical value of plasma biomarkers in Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 19(12), 5805–5816. <https://doi.org/10.1002/alz.13455>

- Braak, H., & Braak, E. (1991). Neuropathological staging of Alzheimer-related changes. *Acta Neuropathologica*, 82(4), 239–259. <https://doi.org/10.1007/BF00308809>
- Bruno, D., Reiss, P. T., Petkova, E., Sidtis, J. J., & Pomara, N. (2013). Decreased recall of primacy words predicts cognitive decline. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, 28(2), 95–103. <https://doi.org/10.1093/arclin/acs116>
- Bruno, D., Kosciak, R. L., Woodard, J. L., Pomara, N., & Johnson, S. C. (2018). The recency ratio as predictor of early MCI. *International Psychogeriatrics*, 30(12), 1883–1888. <https://doi.org/10.1017/S1041610218000467>
- Bruno, D., Mueller, K. D., Betthausen, T., Chin, N., Engelman, C. D., Christian, B., Kosciak, R. L., & Johnson, S. C. (2021). Serial position effects in the Logical Memory Test: Loss of primacy predicts amyloid positivity. *Journal of Neuropsychology*, 15(3), 448–461. <https://doi.org/10.1111/jnp.12235>
- Bruno, D., Jauregi Zinkunegi, A., Kollmorgen, G., Suridjan, I., Wild, N., Carlsson, C., Bendlin, B., Okonkwo, O., Chin, N., Hermann, B. P., Asthana, S., Zetterberg, H., Blennow, K., Langhough, R., Johnson, S. C., & Mueller, K. D. (2023a). The recency ratio assessed by story recall is associated with cerebrospinal fluid levels of neurodegeneration biomarkers. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 159, 167–174. <https://doi.org/10.1016/j.cortex.2022.12.004>
- Bruno, D., Jauregi Zinkunegi, A., Kollmorgen, G., Carboni, M., Wild, N., Carlsson, C., Bendlin, B., Okonkwo, O., Chin, N., Hermann, B. P., Asthana, S., Blennow, K., Langhough, R., Johnson, S. C., Pomara, N., Zetterberg, H., & Mueller, K. D. (2023b). A comparison of diagnostic performance of word-list and story recall tests for biomarker-determined Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, 45(8), 763–769. <https://doi.org/10.1080/13803395.2023.2240060>
- Bruno, D., Jauregi-Zinkunegi, A., Betthausen, T., Carlsson, C., Bendlin, B. B., Okonkwo, O., Chin, N. A., Asthana, S., Langhough, R. E., Johnson, S. C., & Mueller, K. D. (2024a). Story recall performance and AT classification via positron emission tomography: A comparison of logical memory and Craft Story 21. *Journal of the Neurological Sciences*, 464, 123148. <https://doi.org/10.1016/j.jns.2024.123148>
- Bruno, D., Reichert, C., Jauregi Zinkunegi, A., Ashton, N. J., Zetterberg, H., Blennow, K., & Pomara, N. (2024b). Associations of plasma p-tau231 with serial position recall performance in cognitively unimpaired individuals. Pre-print, SSRN. <https://doi.org/10.2139/ssrn.4874374>
- Bruno, D., Gicas, K. M., Jauregi-Zinkunegi, A., Mueller, K. D., & Lamar, M. (2024c). Delayed primacy recall performance predicts post mortem Alzheimer's disease pathology from unimpaired ante mortem cognitive baseline. *Alzheimer's & Dementia (Amsterdam, Netherlands)*, 16(1), e12524. <https://doi.org/10.1002/dad2.12524>
- Chatterjee, P., Pedrini, S., Doecke, J. D., Thota, R., Villemagne, V. L., Doré, V., Singh, A. K., Wang, P., Rainey-Smith, S., Fowler, C., Taddei, K., Sohrabi, H. R., Molloy, M. P., Ames, D., Maruff, P., Rowe, C. C., Masters, C. L., ... Martins, R. N. (2023). Plasma A β 42/40 ratio, p-tau181, GFAP, and NFL across the Alzheimer's disease continuum: A cross-sectional and longitudinal study in the AIBL cohort. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 19(4), 1117–1134. <https://doi.org/10.1002/alz.12724>
- De Simone, M. S., Perri, R., Fadda, L., De Tollis, M., Turchetta, C. S., Caltagirone, C., & Carlesimo, G. A. (2017). Different deficit patterns on word lists and short stories predict conversion to Alzheimer's disease in patients with amnesic mild cognitive impairment. *Journal of Neurology*, 264(11), 2258–2267. <https://doi.org/10.1007/s00415-017-8623-8>
- De Tollis, M., De Simone, M. S., Perri, R., Fadda, L., Caltagirone, C., & Carlesimo, G. A. (2021). Verbal and spatial memory spans in mild cognitive impairment. *Acta Neurologica Scandinavica*, 144(4), 383–393. <https://doi.org/10.1111/ane.13470>
- Díaz-Orueta, U., Blanco-Campal, A., & Burke, T. (2018). Rapid review of cognitive screening instruments in MCI: Proposal for a process-based approach modification of overlapping tasks in select widely used instruments. *International Psychogeriatrics*, 30(5), 663–672. <https://doi.org/10.1017/S1041610217002253>

- Dubois, B., Feldman, H. H., Jacova, C., Dekosky, S. T., Barberger-Gateau, P., Cummings, J., Delacourte, A., Galasko, D., Gauthier, S., Jicha, G., Meguro, K., O'Brien, J., Pasquier, F., Robert, P., Rossor, M., Salloway, S., Stern, Y., Visser, P. J., & Scheltens, P. (2007). Research criteria for the diagnosis of Alzheimer's disease: revising the NINCDS-ADRDA criteria. *The Lancet. Neurology*, 6(8), 734–746. [https://doi.org/10.1016/S1474-4422\(07\)70178-3](https://doi.org/10.1016/S1474-4422(07)70178-3)
- Dubois, B., Hampel, H., Feldman, H. H., Scheltens, P., Aisen, P., Andrieu, S., Bakardjian, H., Benali, H., Bertram, L., Blennow, K., Broich, K., Cavado, E., Crutch, S., Dartigues, J.-F., Duyckaerts, C., Epelbaum, S., Frisoni, G. B., Gauthier, S., Genthon, R., ... Jack, C. R. (2016). Preclinical Alzheimer's disease: Definition, natural history, and diagnostic criteria. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 12(3), 292–323.
- Duke Han, S., Nguyen, C. P., Stricker, N. H., & Nation, D. A. (2017). Detectable neuropsychological differences in early preclinical Alzheimer's disease: A meta-analysis. *Neuropsychology Review*, 27(4), 305–325. <https://doi.org/10.1007/s11065-017-9345-5>
- Edmonds, E. C., Delano-Wood, L., Galasko, D., Salmon, D. P., & Bondi, M. W. (2015). Subtle cognitive decline and biomarker staging in preclinical Alzheimer's disease. *Journal of Alzheimer's Disease*, 47, 231–242.
- García-Escobar, G., Manero, R. M., Fernández-Lebrero, A., Ois, A., Navalpotro-Gómez, I., Puente-Periz, V., Contador-Muñana, J., Estragués-Gazquez, I., Puig-Pijoan, A., & Jiménez-Balado, J. (2024). Blood biomarkers of Alzheimer's disease and cognition: A literature review. *Biomolecules*, 14(1), 93. <https://doi.org/10.3390/biom14010093>
- Grant, I. E., & Adams, K. M. (2009). *Neuropsychological assessment of neuropsychiatric and neurological disorders* (3rd ed.). Oxford University Press.
- Igarashi, K. M. (2023). Entorhinal cortex dysfunction in Alzheimer's disease. *Trends in Neurosciences*, 46(2), 124–136. <https://doi.org/10.1016/j.tins.2022.11.006>
- Jack, C. R., Andrews, J. S., Beach, T. G., Buracchio, T., Dunn, B., Graf, A., Hansson, O., Ho, C., Jagust, W., McDade, E., & Molinuevo, J. L. (2024a). Revised criteria for diagnosis and staging of Alzheimer's disease: Alzheimer's Association Workgroup. *Alzheimer's & Dementia*, 20(8), 5143–5169. <https://doi.org/10.1002/alz.13859>
- Jack, C. R., Wiste, H. J., Algeciras-Schimmich, A., Weigand, S. D., Figdore, D. J., Lowe, V. J., Vemuri, P., Graff-Radford, J., Ramanan, V. K., Knopman, D. S., Mielke, M. M., Machulda, M. M., Fields, J., Schwarz, C. G., Cogswell, P. M., Senjem, M. L., Therneau, T. M., & Petersen, R. C. (2024b). Comparison of plasma biomarkers and amyloid PET for predicting memory decline in cognitively unimpaired individuals. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 20(3), 2143–2154. <https://doi.org/10.1002/alz.13651>
- Janelidze, S., Palmqvist, S., Leuzy, A., Stomrud, E., Verberk, I. M. W., Zetterberg, H., Ashton, N. J., Pesini, P., Sarasa, L., Allué, J. A., Teunissen, C. E., Dage, J. L., Blennow, K., Mattsson-Carlsson, N., & Hansson, O. (2022). Detecting amyloid positivity in early Alzheimer's disease using combinations of plasma A β 42/A β 40 and p-tau. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 18(2), 283–293. <https://doi.org/10.1002/alz.12395>
- Janelidze, S., Bali, D., Ashton, N. J., Barthélemy, N. R., Vanbrabant, J., Stoops, E., Vanmechelen, E., He, Y., Dolado, A. O., Triana-Baltzer, G., Pontecorvo, M. J., Zetterberg, H., Kolb, H., Vandijck, M., Blennow, K., Bateman, R. J., & Hansson, O. (2023). Head-to-head comparison of 10 plasma phospho-tau assays in prodromal Alzheimer's disease. *Brain: A Journal of Neurology*, 146(4), 1592–1601. <https://doi.org/10.1093/brain/awac333>
- Jauregi Zinkunegi, A., Bruno, D., Betthausen, T. J., Langhough, R., Asthana, S., Chin, N. A., Hermann, B. P., Johnson, S. C., & Mueller, K. D. (2024). A comparison of story-recall metrics to predict hippocampal volume in older adults with and without cognitive impairment. *The Clinical Neuropsychologist*, 38(2), 453–470. <https://doi.org/10.1080/13854046.2023.2223389>
- Jauregi-Zinkunegi, A., Betthausen, T., Carlsson, C. M., Bendlin, B. B., Okonkwo, O., Chin, N. A., Asthana, S., Langhough, R. E., Johnson, S. C., Mueller, K. D., & Bruno, D. (2025). Delayed primacy recall in AVLT is associated with medial temporal tau PET burden in cognitively unimpaired adults. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 184, 47–57. <https://doi.org/10.1016/j.cortex.2024.12.012>

- Johnson, S. C., Kosciak, R. L., Jonaitis, E. M., Clark, L. R., Mueller, K. D., Berman, S. E., Bendlin, B. B., Engelman, C. D., Okonkwo, O. C., Hogan, K. J., Asthana, S., Carlsson, C. M., Hermann, B. P., & Sager, M. A. (2018). The Wisconsin Registry for Alzheimer's Prevention: A review of findings and current directions. *Alzheimer's & Dementia (Amsterdam, Netherlands)*, *10*, 130–142. <https://doi.org/10.1016/j.dadm.2017.11.007>
- Karikari, T. K., Pascoal, T. A., Ashton, N. J., Janelidze, S., Benedet, A. L., Rodriguez, J. L., Chamoun, M., Savard, M., Kang, M. S., Therriault, J., Schöll, M., Massarweh, G., Soucy, J.-P., Höglund, K., Brinkmalm, G., Mattsson, N., Palmqvist, S., Gauthier, S., Stomrud, E., ... Blennow, K. (2020). Blood phosphorylated tau 181 as a biomarker for Alzheimer's disease: A diagnostic performance and prediction modelling study using data from four prospective cohorts. *The Lancet. Neurology*, *19*(5), 422–433. [https://doi.org/10.1016/S1474-4422\(20\)30071-5](https://doi.org/10.1016/S1474-4422(20)30071-5)
- Karikari, T. K., Ashton, N. J., Brinkmalm, G., Brum, W. S., Benedet, A. L., Montoliu-Gaya, L., Lantero-Rodriguez, J., Pascoal, T. A., Suárez-Calvet, M., Rosa-Neto, P., Blennow, K., & Zetterberg, H. (2022). Blood phospho-tau in Alzheimer disease: Analysis, interpretation, and clinical utility. *Nature Reviews. Neurology*, *18*(7), 400–418. <https://doi.org/10.1038/s41582-022-00665-2>
- Langhough Kosciak, R., Hermann, B. P., Allison, S., Clark, L. R., Jonaitis, E. M., Mueller, K. D., Betthausen, T. J., Christian, B. T., Du, L., Okonkwo, O., Birdsill, A., Chin, N., Gleason, C., & Johnson, S. C. (2021). Validity evidence for the research category, "Cognitively Unimpaired—Declining," as a risk marker for mild cognitive impairment and Alzheimer's disease. *Frontiers in Aging Neuroscience*, *13*, 688478. <https://doi.org/10.3389/fnagi.2021.688478>
- Lee, I. A., & Preacher, K. J. (2013, September). *Calculation for the test of the difference between two dependent correlations with one variable in common* [Computer software]. <http://quantpsy.org>
- Lim, Y. Y., Pietrzak, R. H., Ellis, K. A., Jaeger, J., Harrington, K., Ashwood, T., Szoeki, C., Martins, R. N., Bush, A. I., Masters, C. L., Rowe, C. C., Villemagne, V. L., Ames, D., Darby, D., & Maruff, P. (2013). Rapid decline in episodic memory in healthy older adults with high amyloid- β . *Journal of Alzheimer's Disease: JAD*, *33*(3), 675–679. <https://doi.org/10.3233/JAD-2012-121516>
- Lim, Y. Y., Snyder, P. J., Pietrzak, R. H., Ukiqi, A., Villemagne, V. L., & Ames, D. (2016). Sensitivity of composite scores to amyloid burden in preclinical Alzheimer's disease: Introducing the Z-scores of Attention, Verbal fluency, and Episodic memory for Nondemented older adults composite score. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring*, *2*, 19–26.
- Lo, A. H., Humphreys, M., Byrne, G. J., & Pachana, N. A. (2012). Test–retest reliability and practice effects of the Wechsler Memory Scale-III. *Journal of Neuropsychology*, *6*(2), 212–231. <https://doi.org/10.1111/j.1748-6653.2011.02023.x>
- Manly, J. J., Avila, J. F., Vonk, J. M., Rentería, M. A., Turney, I. C., Lao, P. J., Seblova, D., Martinez, M. N., Gutierrez, J., Mayeux, R., & Schupf, N. (2021). Racial differences in cognitive resilience to parental history of AD and cognitive impairment: The offspring study. *Alzheimer's & Dementia*, *17*, e051874.
- Mattsson-Carlgrén, N., Janelidze, S., Palmqvist, S., Cullen, N., Svenningsson, A. L., Strandberg, O., Mengel, D., Walsh, D. M., Stomrud, E., Dage, J. L., & Hansson, O. (2020). Longitudinal plasma p-tau217 is increased in early stages of Alzheimer's disease. *Brain: A Journal of Neurology*, *143*(11), 3234–3241. <https://doi.org/10.1093/brain/awaa286>
- Mattsson-Carlgrén, N., Janelidze, S., Bateman, R. J., Smith, R., Stomrud, E., Serrano, G. E., Reiman, E. M., Palmqvist, S., Dage, J. L., Beach, T. G., & Hansson, O. (2021). Soluble P-tau217 reflects amyloid and tau pathology and mediates the association of amyloid with tau. *EMBO Molecular Medicine*, *13*(6), e14022. <https://doi.org/10.15252/emmm.202114022>
- Mattsson-Carlgrén, N., Salvadó, G., Ashton, N. J., Tideman, P., Stomrud, E., Zetterberg, H., Ossenkoppele, R., Betthausen, T. J., Cody, K. A., Jonaitis, E. M., Langhough, R., Palmqvist, S., Blennow, K., Janelidze, S., Johnson, S. C., & Hansson, O. (2023). Prediction of longitudinal cognitive decline in preclinical Alzheimer disease using plasma biomarkers. *JAMA Neurology*, *80*(4), 360–369. <https://doi.org/10.1001/jamaneurol.2022.5272>
- McKhann, G. M., Knopman, D. S., Chertkow, H., Hyman, B. T., Jack, C. R., Jr, Kawas, C. H., Klunk, W. E., Koroshetz, W. J., Manly, J. J., Mayeux, R., Mohs, R. C., Morris, J. C., Rossor, M. N., Scheltens, P., Carrillo, M. C., Thies, B., Weintraub, S., & Phelps, C. H. (2011). The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's

- Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 7(3), 263–269. <https://doi.org/10.1016/j.jalz.2011.03.005>
- Meyer, P.-F., Ashton, N. J., Karikari, T. K., Strikwerda-Brown, C., Köbe, T., Gonneaud, J., Pichet Binette, A., Ozlen, H., Yakoub, Y., Simrén, J., Pannee, J., Lantero-Rodriguez, J., Labonté, A., Baker, S. L., Schöll, M., Vanmechelen, E., Breitner, J. C. S., Zetterberg, H., Blennow, K., Poirier, J., & Villeneuve, S. (2022). Plasma p-tau231, p-tau181, PET biomarkers, and cognitive change in older adults. *Annals of Neurology*, 91(4), 548–560. <https://doi.org/10.1002/ana.26308>
- Milà-Alomà, M., Ashton, N. J., Shekari, M., Salvadó, G., Ortiz-Romero, P., Montoliu-Gaya, L., Benedet, A. L., Karikari, T. K., Lantero-Rodriguez, J., Vanmechelen, E., Day, T. A., González-Escalante, A., Sánchez-Benavides, G., Minguillon, C., Fauria, K., Molinuevo, J. L., Dage, J. L., Zetterberg, H., Gispert, J. D., Suárez-Calvet, M., & Blennow, K. (2022). Plasma p-tau231 and p-tau217 as state markers of amyloid-beta pathology in preclinical Alzheimer's disease. *Nature Medicine*, 28(9), 1797–1801. <https://doi.org/10.1038/s41591-022-01925-w>
- Morris, J. C., Schindler, S. E., McCue, L. M., Moulder, K. L., Benzinger, T. L. S., Cruchaga, C., Fagan, A. M., Grant, E., Gordon, B. A., Holtzman, D. M., & Xiong, C. (2019). Assessment of racial disparities in biomarkers for Alzheimer disease. *JAMA Neurology*, 76(3), 264–273. <https://doi.org/10.1001/jamaneurol.2018.4249>
- Mueller, K. D., Du, L., Bruno, D., Betthausen, T., Christian, B., Johnson, S., Hermann, B., & Kosciak, R. L. (2022). Item-level story recall predictors of amyloid-beta in late middle-aged adults at increased risk for Alzheimer's disease. *Frontiers in Psychology*, 13, Article 908651. <https://doi.org/10.3389/fpsyg.2022.908651>
- Murdock, B. B. (1962). The serial position effect of free recall. *Journal of Experimental Psychology*, 64(5), 482–488. <https://doi.org/10.1037/h0045106>
- Pais, M. V., Forlenza, O. V., & Diniz, B. S. (2023). Plasma biomarkers of Alzheimer's disease: A review of available assays, recent developments, and implications for clinical practice. *Journal of Alzheimer's Disease Reports*, 7(1), 355–380. <https://doi.org/10.3233/ADR-230029>
- Palmqvist, S., Janelidze, S., Quiroz, Y. T., Zetterberg, H., Lopera, F., Stomrud, E., Su, Y., Chen, Y., Serrano, G. E., Leuzy, A., Mattsson-Carlgrén, N., Strandberg, O., Smith, R., Villegas, A., Sepulveda-Falla, D., Chai, X., Proctor, N. K., Beach, T. G., Blennow, K., ... Hansson, O. (2020). Discriminative accuracy of plasma phospho-tau217 for Alzheimer disease vs other neurodegenerative disorders. *JAMA*, 324(8), 772–781. <https://doi.org/10.1001/jama.2020.12134>
- Schindler, S. E., Bollinger, J. G., Ovod, V., Mawuenyega, K. G., Li, Y., Gordon, B. A., Holtzman, D. M., Morris, J. C., Benzinger, T. L. S., Xiong, C., Fagan, A. M., & Bateman, R. J. (2019). High-precision plasma beta-amyloid 42/40 predicts current and future brain amyloidosis. *Neurology*, 93(17), e1647–e1659. <https://doi.org/10.1212/WNL.00000000000008081>
- Snellman, A., Ekblad, L. L., Ashton, N. J., Karikari, T. K., Lantero-Rodriguez, J., Pietilä, E., Koivumäki, M., Helin, S., Karrasch, M., Zetterberg, H., Blennow, KAJ., & Rinne, J. O. (2023). Head-to-head comparison of plasma p-tau181, p-tau231 and glial fibrillary acidic protein in clinically unimpaired elderly with three levels of APOE4-related risk for Alzheimer's disease. *Neurobiology of Disease*, 183, 106175. <https://doi.org/10.1016/j.nbd.2023.106175>
- Song, Z., Insel, P. S., Buckley, S., Yohannes, S., Mezher, A., Simonson, A., Wilkins, S., Tosun, D., Mueller, S., Kramer, J. H., Miller, B. L., & Weiner, M. W. (2015). Brain amyloid- β burden is associated with disruption of intrinsic functional connectivity within the medial temporal lobe in cognitively normal elderly. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 35(7), 3240–3247. <https://doi.org/10.1523/JNEUROSCI.2092-14.2015>
- Steiger, J. H. (1980). Tests for comparing elements of a correlation matrix. *Psychological Bulletin*, 87(2), 245–251. <https://doi.org/10.1037/0033-2909.87.2.245>
- Sun, Q., Ni, J., Wei, M., Long, S., Li, T., Fan, D., ... & Tian, J. (2022). Plasma β -amyloid, tau, neurodegeneration biomarkers and inflammatory factors of probable Alzheimer's disease dementia in Chinese individuals. *Frontiers in Aging Neuroscience*, 14, 963845.
- Talamonti, D., Kosciak, R., Johnson, S., & Bruno, D. (2020). Predicting early mild cognitive impairment with free recall: The primacy of primacy. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, 35(2), 133–142. <https://doi.org/10.1093/arclin/acz013>

- The Psychological Corporation. (1997). *WAIS-III – WMS-III technical manual*.
- Therriault, J., Servaes, S., Tissot, C., Rahmouni, N., Ashton, N. J., Benedet, A. L., Karikari, T. K., Macedo, A. C., Lussier, F. Z., Stevenson, J., Wang, Y.-T., Fernandez-Arias, J., Stevenson, A., Socualaya, K. Q., Haeger, A., Nazneen, T., Aumont, É., Hosseini, A., Rej, S., ... Rosa-Neto, P. (2023). Equivalence of plasma p-tau217 with cerebrospinal fluid in the diagnosis of Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 19(11), 4967–4977. <https://doi.org/10.1002/alz.13026>
- Tsai, C.-L., Liang, C.-S., Lee, J.-T., Su, M.-W., Lin, C.-C., Chu, H.-T., Tsai, C.-K., Lin, G.-Y., Lin, Y.-K., & Yang, F.-C. (2019). Associations between Plasma Biomarkers and Cognition in Patients with Alzheimer's Disease and Amnesic Mild Cognitive Impairment: A Cross-Sectional and Longitudinal Study. *Journal of Clinical Medicine*, 8(11), 1893. <https://doi.org/10.3390/jcm8111893>
- Vos, S. J., Xiong, C., Visser, P. J., Jasielec, M. S., Hassenstab, J., Grant, E. A., Cairns, N. J., Morris, J. C., Holtzman, D. M., & Fagan, A. M. (2013). Preclinical Alzheimer's disease and its outcome: A longitudinal cohort study. *The Lancet. Neurology*, 12(10), 957–965. [https://doi.org/10.1016/S1474-4422\(13\)70194-7](https://doi.org/10.1016/S1474-4422(13)70194-7)
- Wang, Y. L., Chen, J., Du, Z. L., Weng, H., Zhang, Y., Li, R., ... & Alzheimer's Disease Neuroimaging Initiative. (2021). Plasma p-tau181 level predicts neurodegeneration and progression to Alzheimer's dementia: a longitudinal study. *Frontiers in neurology*, 12, 695696.
- Wechsler, D. (1987). *WMS-R: Wechsler memory scale-revised*. Psychological Corporation.
- Weigand, A. J., Ortiz, G., Walker, K. S., Galasko, D. R., Bondi, M. W., & Thomas, K. R. (2023). APOE differentially moderates cerebrospinal fluid and plasma phosphorylated tau181 associations with multi-domain cognition. *Neurobiology of Aging*, 125, 1–8. <https://doi.org/10.1016/j.neurobiolaging.2022.10.016>
- Wixted, J. T. (2004). The psychology and neuroscience of forgetting. *Annual Review of Psychology*, 55, 235–269. <https://doi.org/10.1146/annurev.psych.55.090902.141555>
- Wixted, J. T., & Cai, D. J. (2013). Memory consolidation. In *The Oxford handbook of cognitive neuroscience* (vol. 1, pp. 436–456).
- World Health Organization. (2023, March 15). *Dementia*. <https://www.who.int/news-room/fact-sheets/detail/dementia>
- Xiao, Z., Wu, X., Wu, W., Yi, J., Liang, X., Ding, S., Zheng, L., Luo, J., Gu, H., Zhao, Q., Xu, H., & Ding, D. (2021). Plasma biomarker profiles and the correlation with cognitive function across the clinical spectrum of Alzheimer's disease. *Alzheimer's Research & Therapy*, 13(1), 1–12. <https://doi.org/10.1186/s13195-021-00864-x>