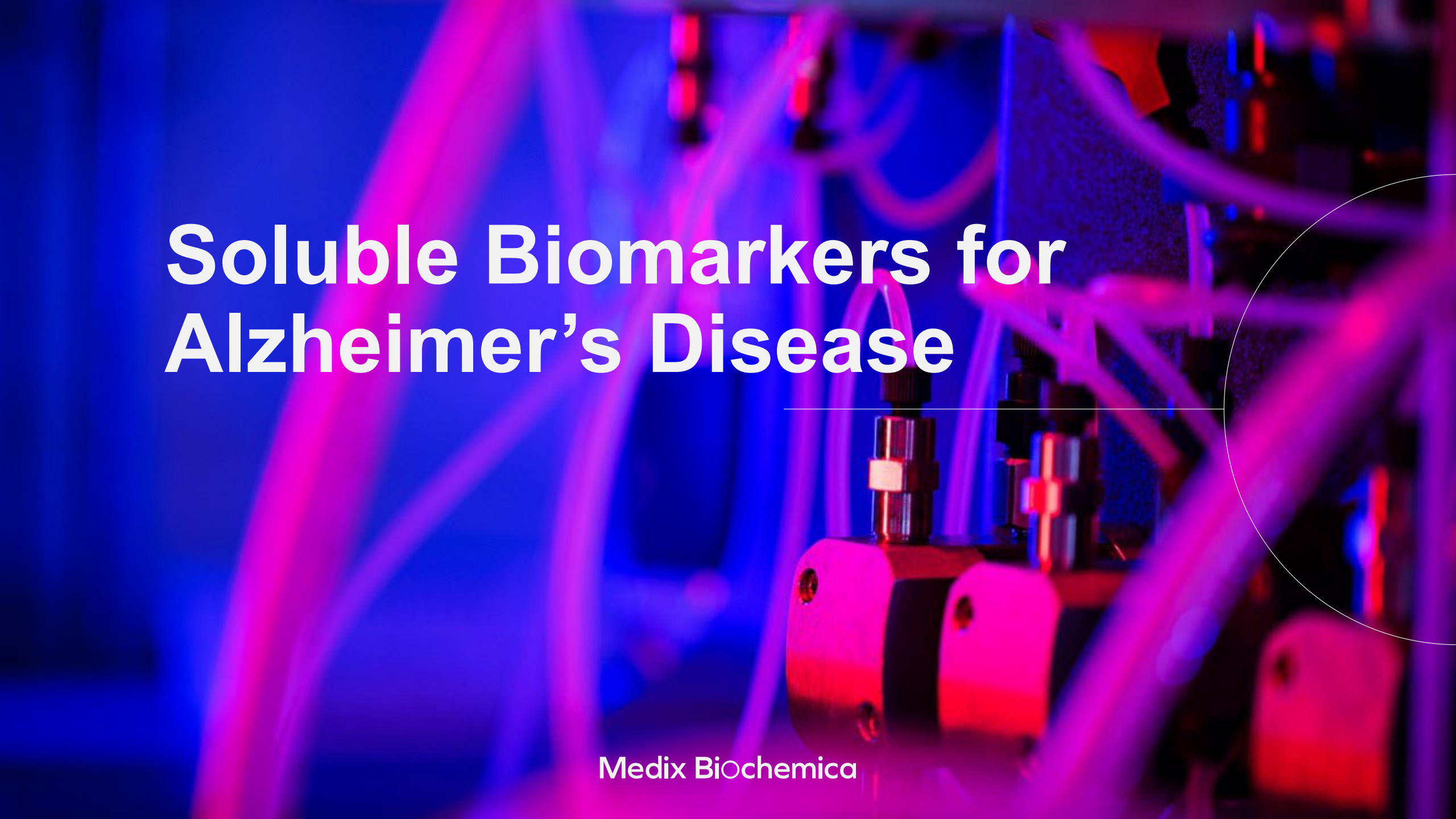
Soluble Biomarkers for Alzheimer's Disease



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Before we get started

Please use the Q&A button at the bottom of your screen to submit any questions.

There will be a Q&A session at the end of this webinar. If we do not have time to answer your questions, we will reach out by email following the webinar.

This webinar is being recorded. The recording as well as on-demand link will be released following the webinar.

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Featured Speakers

Henrik Zetterberg

Professor
Institute of Neuroscience and Physiology
University of Gothenburg, Sweden

Emilia Galli

Ph.D., R&D Manager
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Soluble biomarkers for Alzheimer's disease

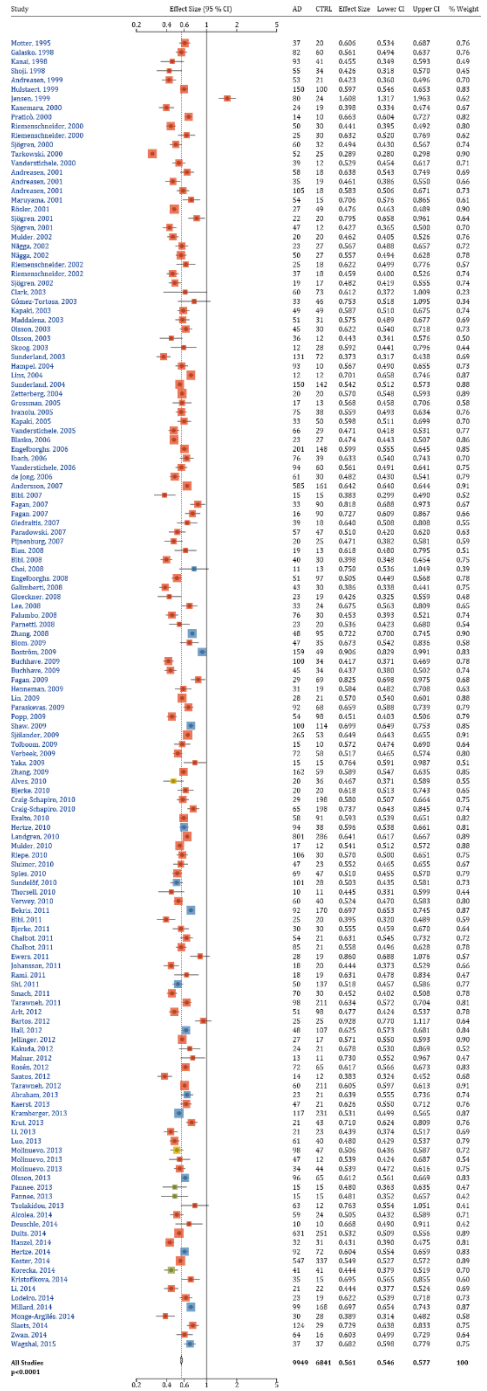
Henrik Zetterberg, MD, PhD
Department of Psychiatry and Neurochemistry, University of Gothenburg, Sweden;
Institute of Neurology and UK Dementia Research Institute, University College London, UK;
Wisconsin Alzheimer's Disease Research Center, University of Wisconsin School of Medicine and Public Health, University of Wisconsin-Madison, Madison, WI, USA

Outline

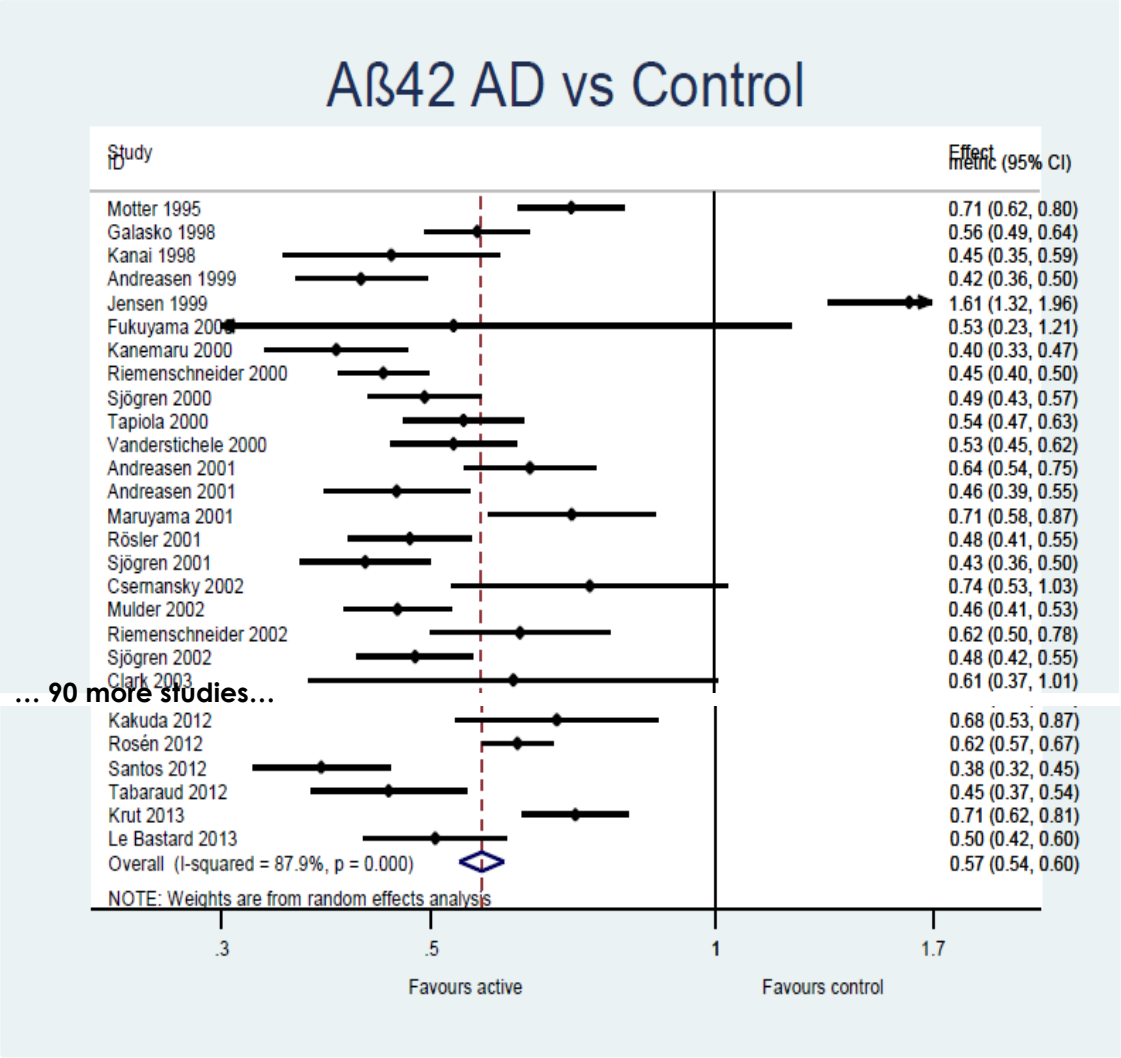
I will give an update on some of the most exciting recent developments on fluid-based biomarkers for:

- Amyloid pathology (CSF and blood)
- Tau pathology (CSF and blood)
- Neurodegeneration (CSF and blood)
- Glial activation (CSF and blood)
- Synaptic pathology (CSF)
- Synuclein pathology (CSF)
- TDP-43 pathology (CSF)

A = amyloid pathology

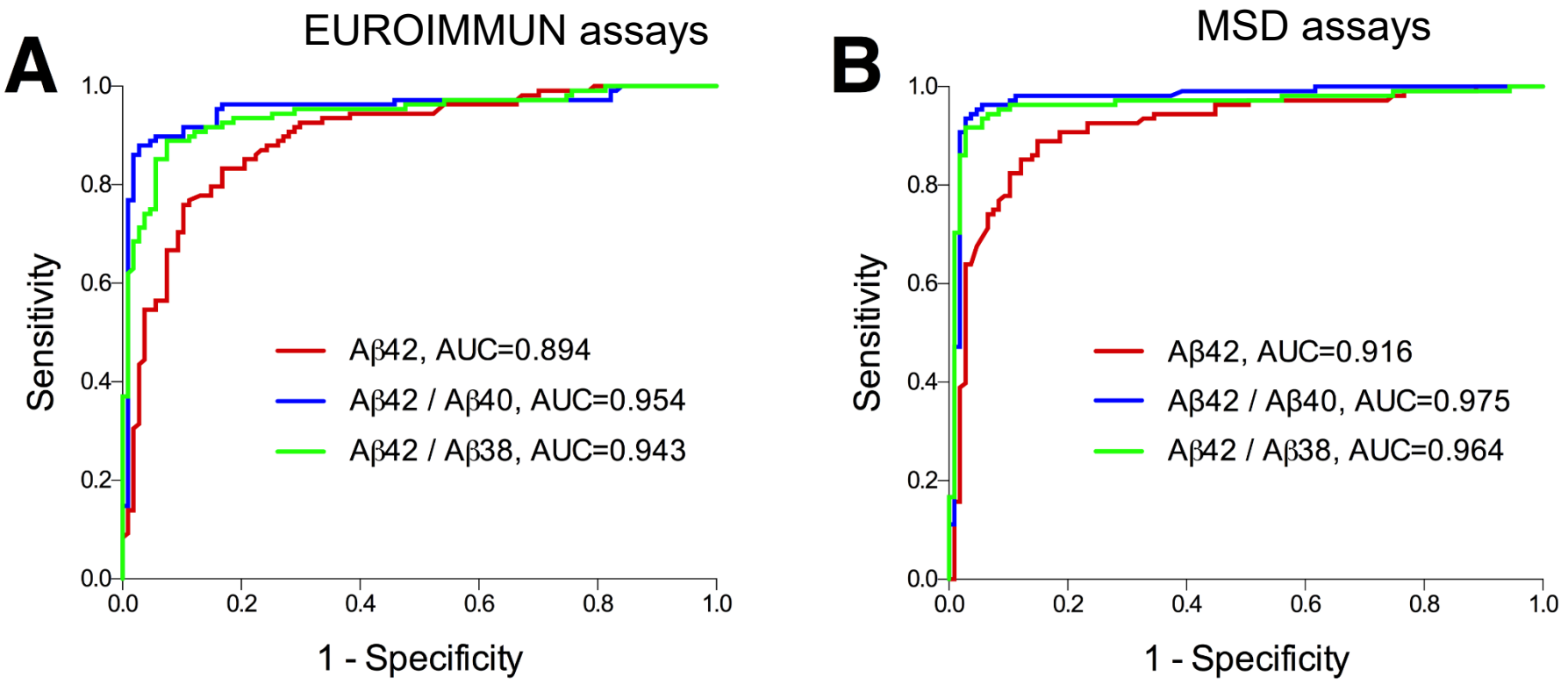


CSF Aβ42 concentration is reduced in AD



AlzBiomarker Database

CSF Aβ42/40 is (a little bit) better than Aβ42 alone



Cohort: Swedish BioFINDER
215 SCD/MCI (108 PET^{+} and 107 PET-)
PET: flutemetamol

Janelidze *et al*., Ann Clin Transl Neurol. 2016

The CSF Aβ42/Aβ40 ratio in clinical practice

DE GRUYTER Clin Chem Lab Med 2021; aop

Johan Gobom*, Lucilla Parnetti, Pedro Rosa-Neto, Martin Vyhnalek, Serge Gauthier, Samuela Cataldi, Ondrej Lerch, Jan Laczo, Katerina Cechova, Marcus Clarin, Andrea I. Benet, Tharick A. Pascoal, Neserine Rahmouni, Manu Vandijck, Else Huyck, Nathalie Le Bastard, Jenna Stevenson, Mira Chamoun, Daniel Alcolea, Alberto Lleó, Ulf Andreasson, Marcel M. Verbeek, Giovanni Bellomo, Roberta Rinaldi, Nicholas Ashton, Henrik Zetterberg, Katerina Sheardova, Jakub Hort and Kaj Blennow

Validation of the LUMIPULSE automated immunoassay for the measurement of core AD biomarkers in cerebrospinal fluid

- Clinical validation of fully automated assays for the core AD CSF biomarkers
- High repeatability (intra-assay CVs) and reproducibility (inter-lassay CVs)

- Unselected patients in clinical routine
- No healthy controls
- 12012 samples during 3 years
- Analyses done 2 times per week using different lots of reagents and calibrators

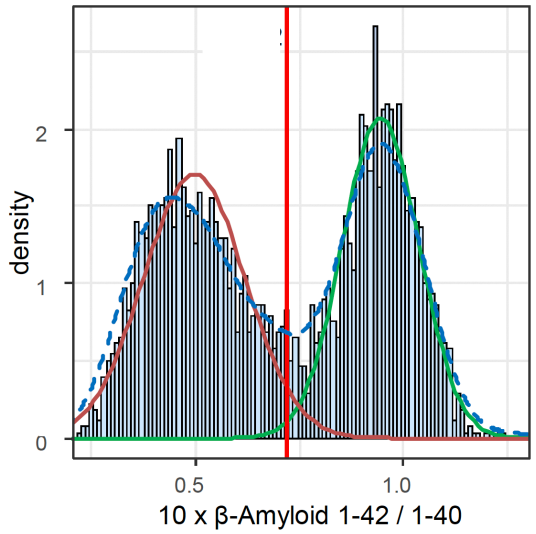
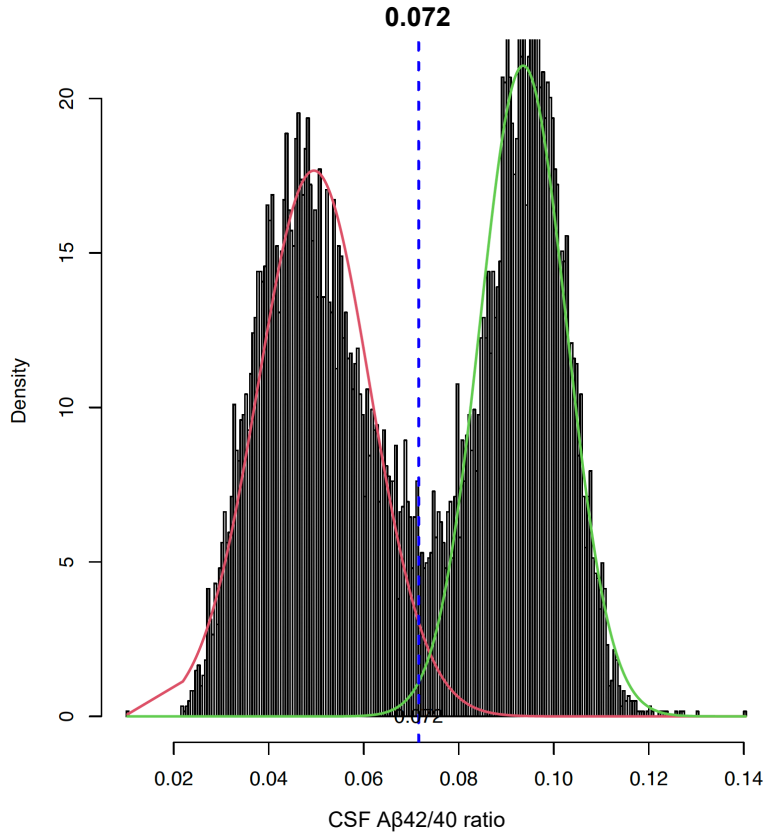


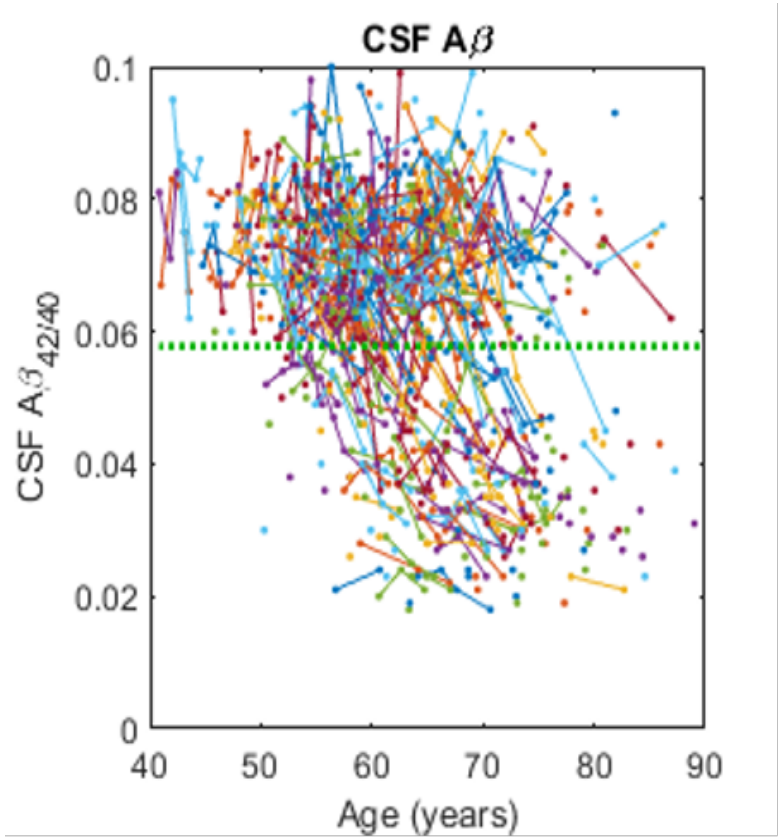
Figure 5: Mixture model analysis of β-amyloid 1-42/1-40 of data from patient CSF samples (n=2,782) analyzed in a routine laboratory setting.

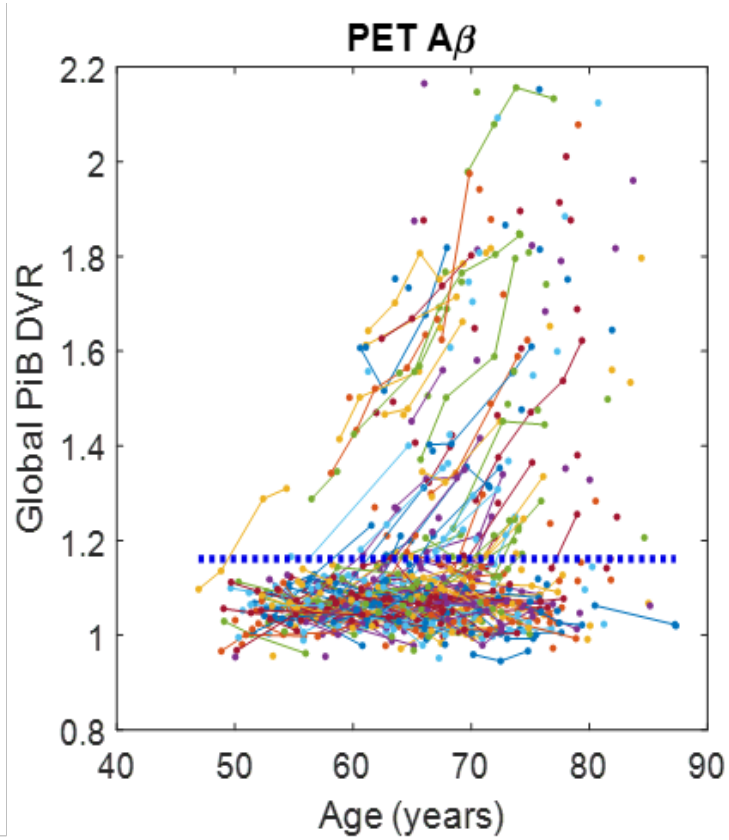


➔ **High clinical performance with two very distinct populations**

➔ **Cut-off stable at 0.072**

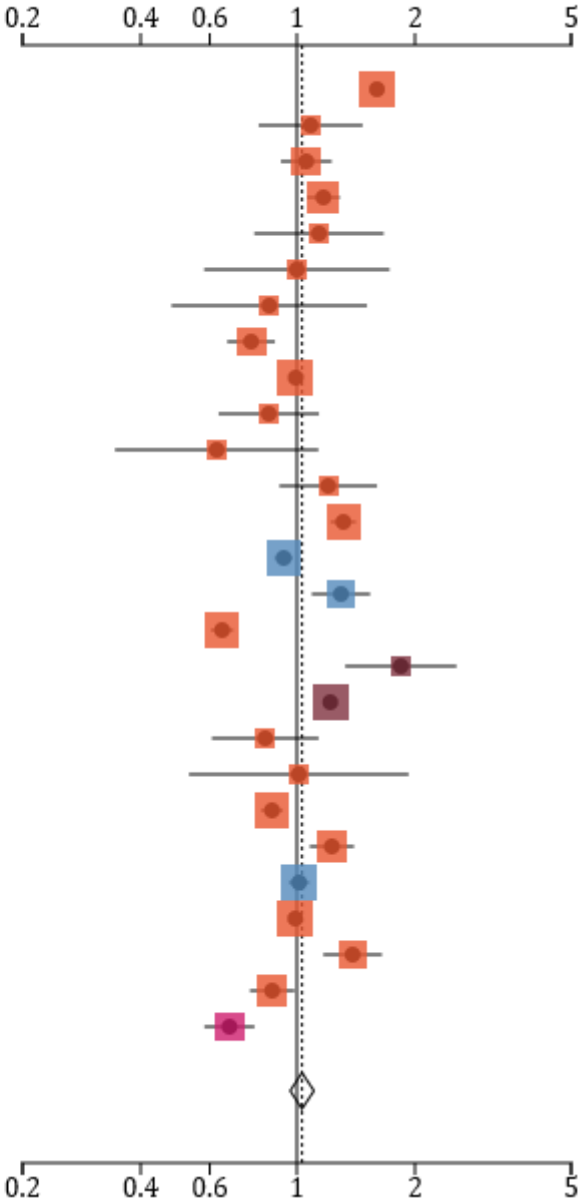
CSF Aβ42/Aβ40 ratio – longitudinal data





Betthauser T et al., unpublished

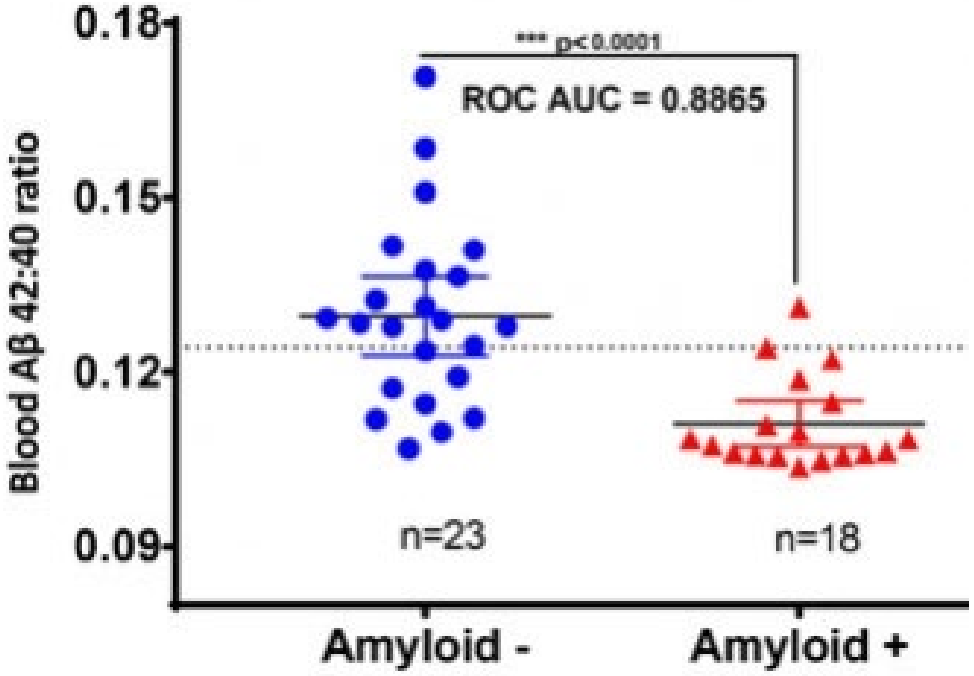
How about plasma Aβ?



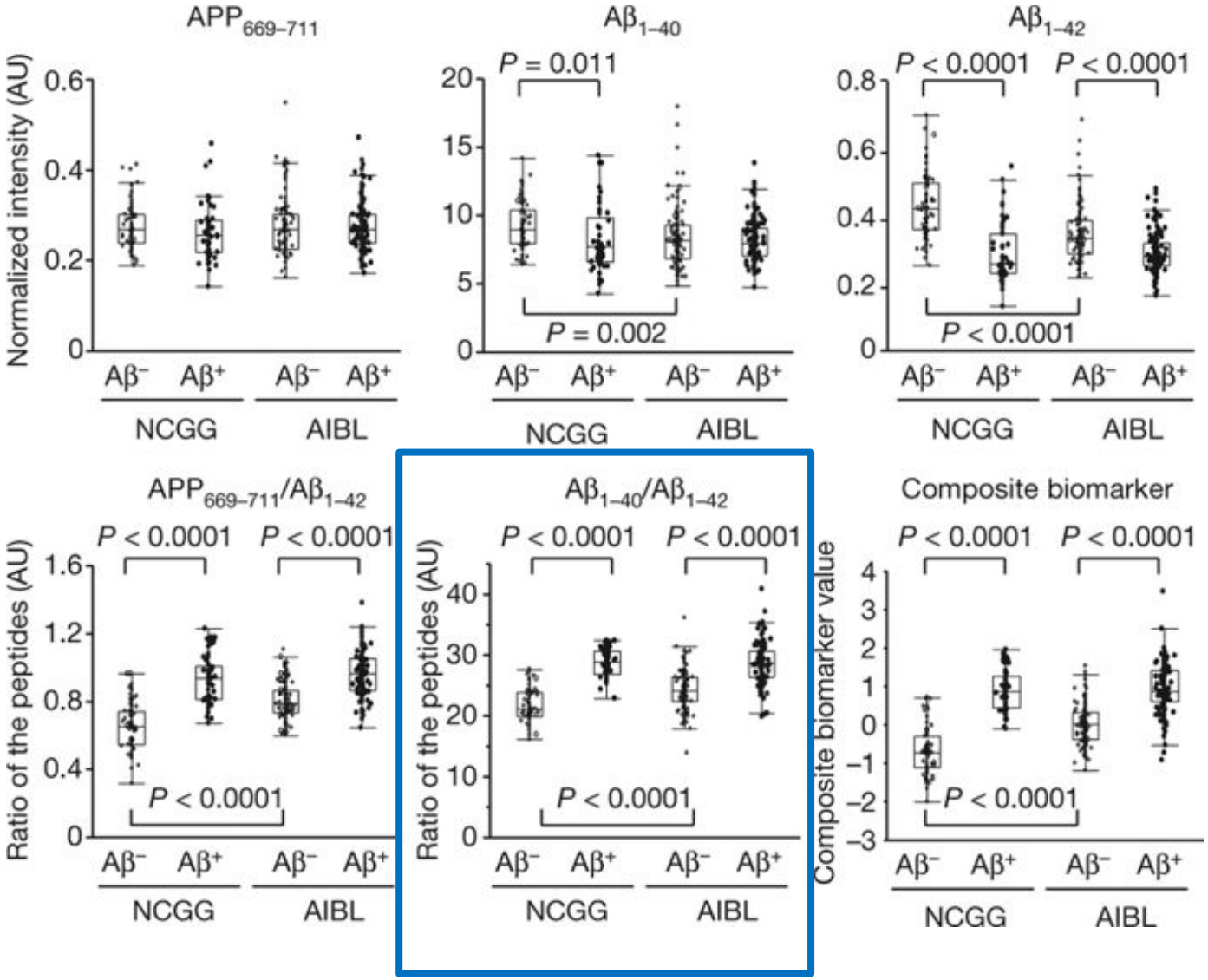
Study	AD	CTRL	Effect Size	Lower CI	Upper CI	% Weight
Mayeux, 1999	64	105	1.600	1.553	1.648	5.19
Arvanitakis, 2002	220	59	1.085	0.800	1.471	2.63
Fukumoto, 2003	146	92	1.057	0.911	1.227	4.23
Mayeux, 2003	79	365	1.168	1.061	1.287	4.76
Fagan, 2007	33	65	1.139	0.779	1.665	2.06
Fagan, 2007	16	65	1.000	0.580	1.723	1.26
Giedraitis, 2007	39	18	0.850	0.479	1.509	1.16
Pesaresi, 2007	192	126	0.765	0.665	0.879	4.33
Schupf, 2008	104	1021	0.994	0.986	1.002	5.23
Xu, 2008	113	155	0.848	0.632	1.139	2.72
Fagan, 2009	29	69	0.625	0.344	1.135	1.09
Roher, 2009	17	21	1.202	0.903	1.601	2.79
Sedaghat, 2009	29	16	1.313	1.219	1.415	4.95
Lui, 2010	186	724	0.926	0.878	0.977	5.08
Sundelöf, 2010	101	28	1.295	1.091	1.538	3.99
Head, 2011	17	12	0.645	0.604	0.689	5.01
Chiu, 2013	30	107	1.841	1.328	2.552	2.45
Chiu, 2013	31	107	1.217	1.175	1.261	5.17
Ruiz, 2013	51	53	0.831	0.607	1.136	2.56
Yamamoto, 2013	23	13	1.012	0.531	1.927	0.96
Zhang, 2013	153	120	0.865	0.811	0.922	5.02
Bu, 2014	128	135	1.228	1.079	1.399	4.44
Rembach, 2014	125	577	1.013	0.953	1.077	5.04
Wang, 2014	97	122	0.991	0.979	1.003	5.23
Jiao, 2015	156	129	1.387	1.168	1.648	3.98
Kim, 2015	100	46	0.865	0.759	0.986	4.43
Janelidze, 2016	57	274	0.673	0.581	0.780	4.25
All Studies	**2336**	**4452**	**1.031**	**0.962**	**1.106**	**100**

p=0.38718

Highly sensitive and precise mass spec methods work



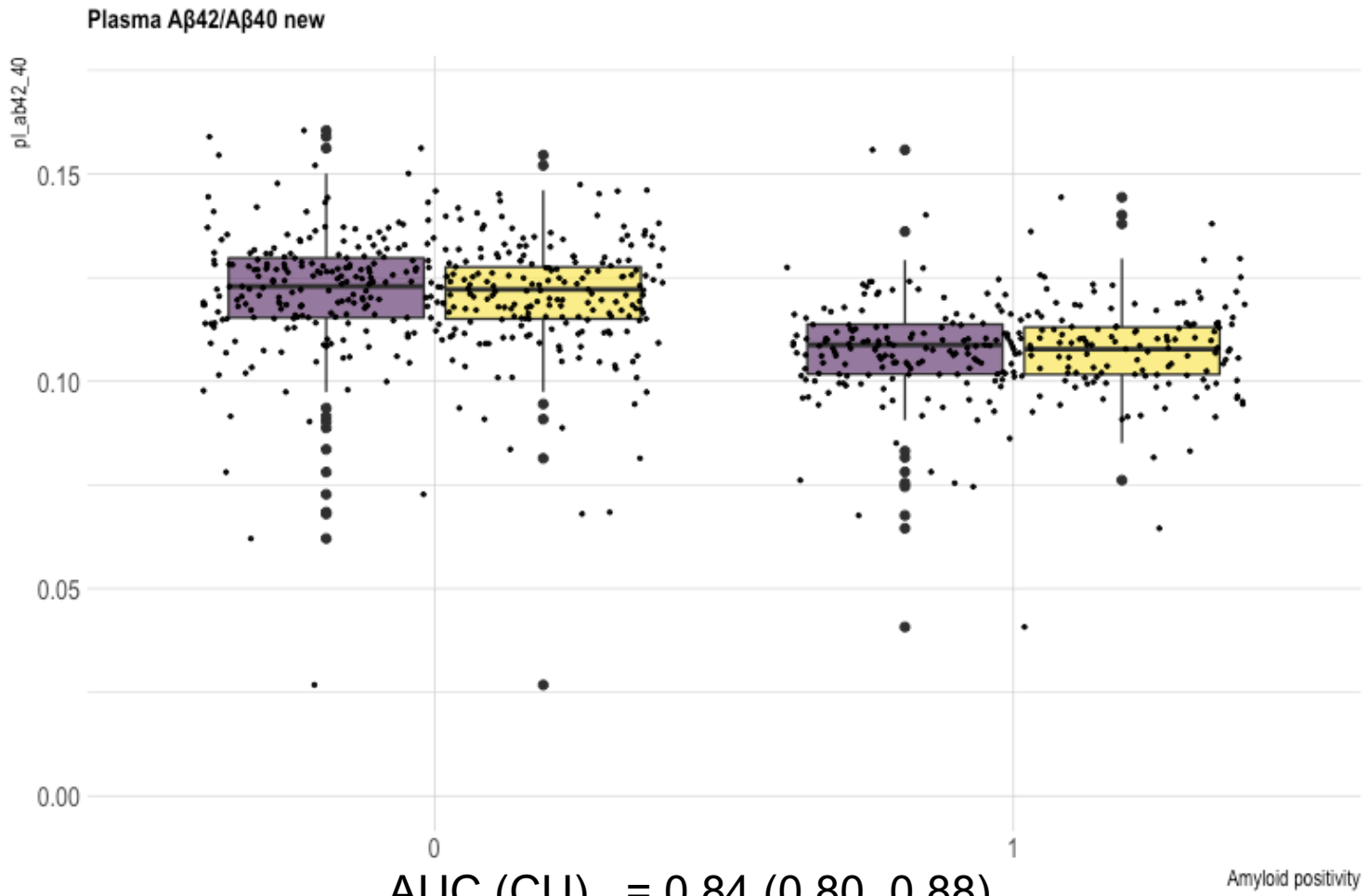
Ovod et al. A&D, 2017



Nakamura et al., Nature, 2018

Plasma Aβ42/Aβ40 ratio using a fully automated Cobas assay

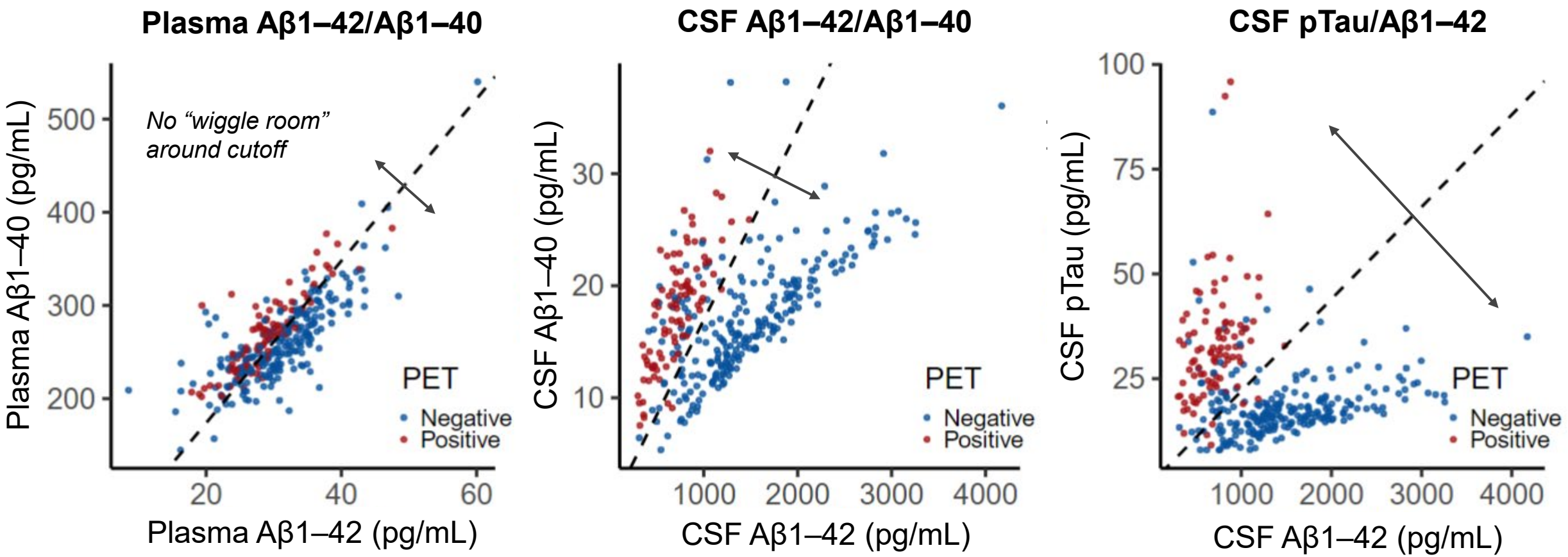
- Purple = CU
- Yellow = MCI



AUC (CU) = 0.84 (0.80, 0.88)
AUC (MCI) = 0.79 (0.72, 0.85)

Palmqvist *et al*., 2022

The challenge



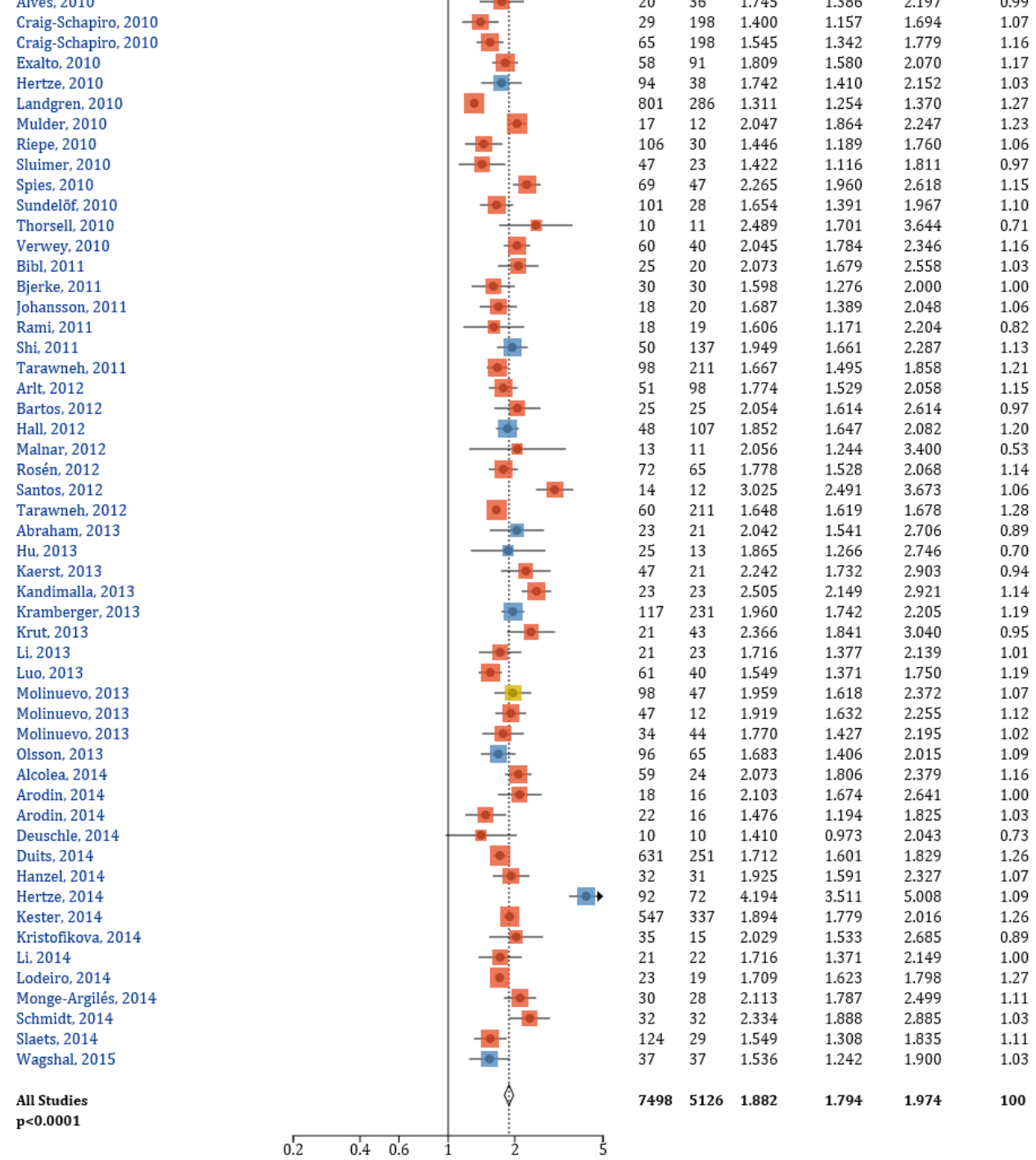
Rabe C *et al*., 2022

Diagnosing amyloid pathology with a blood test: are we there yet?

Group level enrichment/screening: Yes

Individual diagnostics: No, or maybe, but with great caution

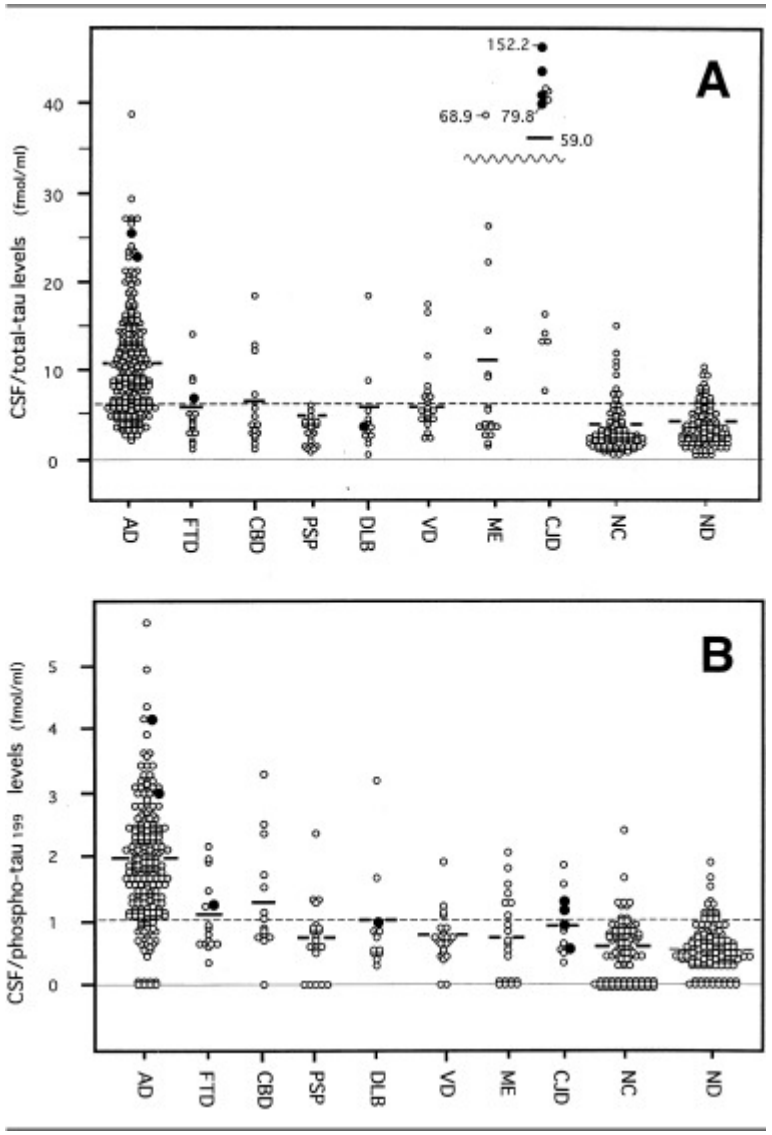
T = tau pathology



CSF P-tau is increased in AD

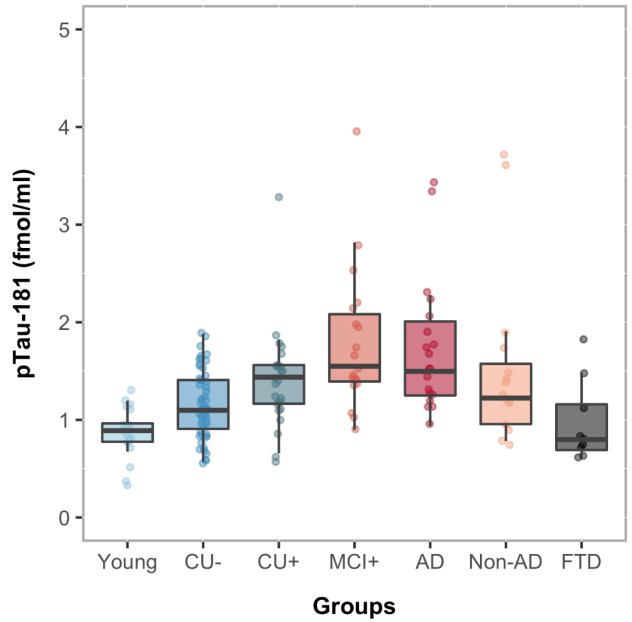
AlzBiomarker Database

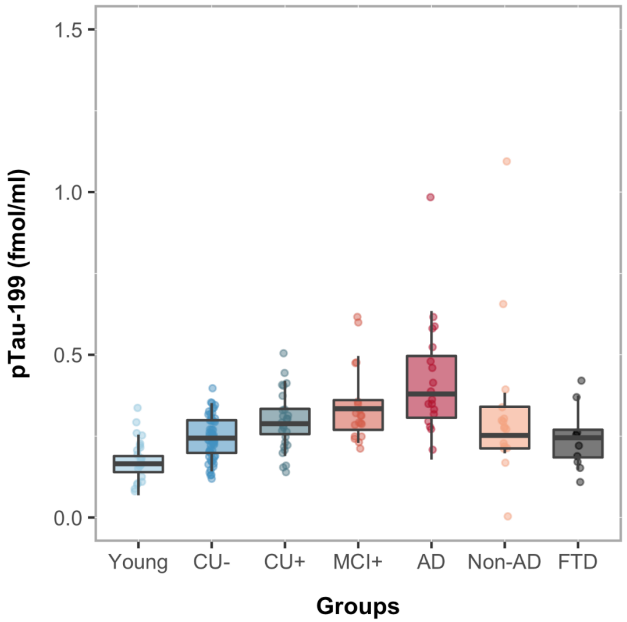
CSF P-tau increase only in AD, not in (most) other neurodegenerative diseases

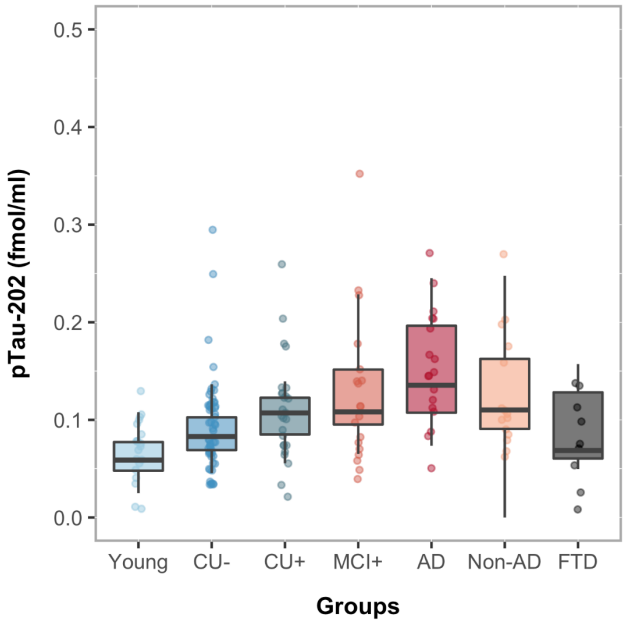


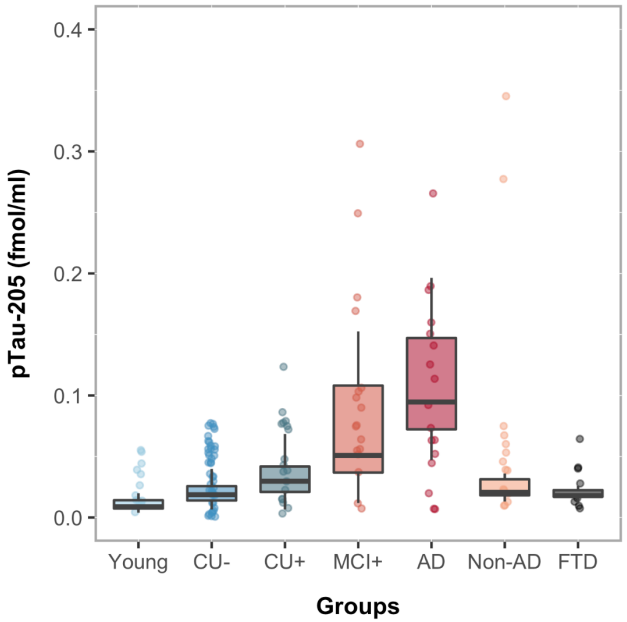
Itoh *et al*., Ann Neurol 2001

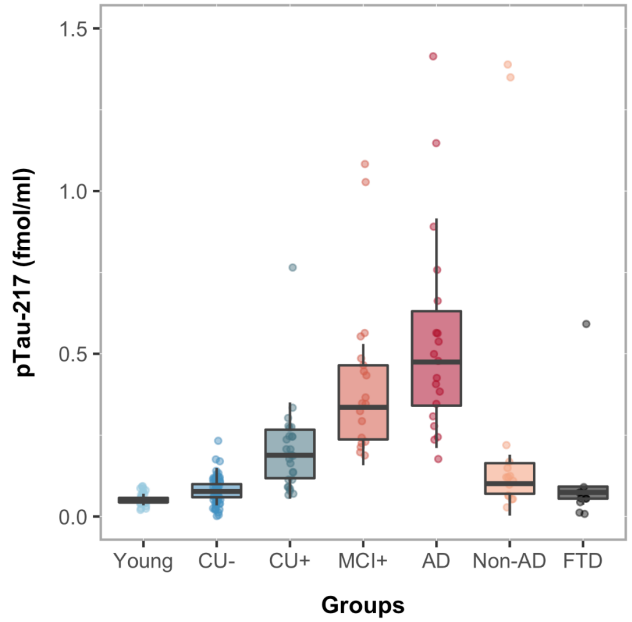
Differential detection of AD measuring different phospho-forms of tau in CSF

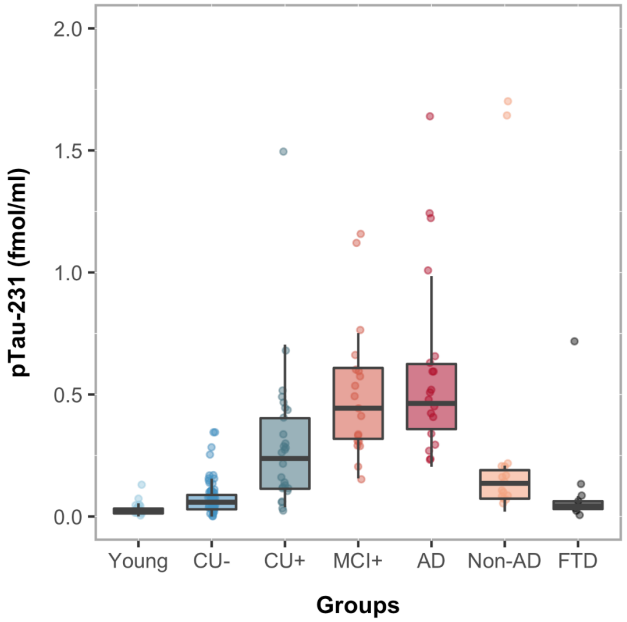


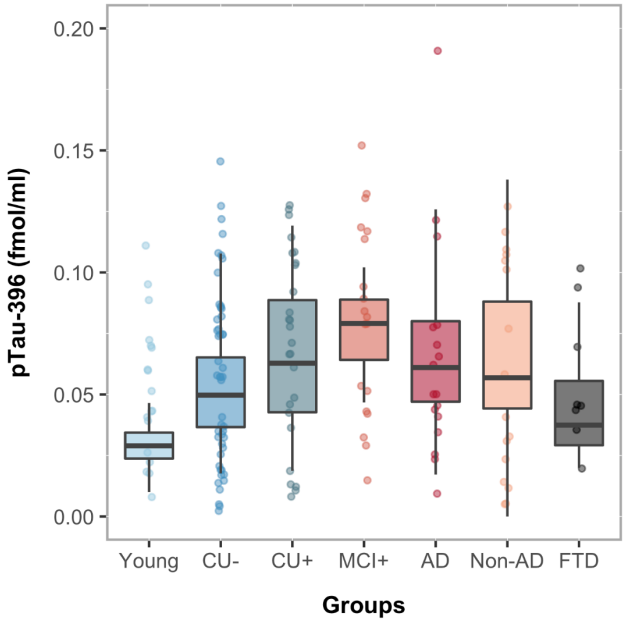


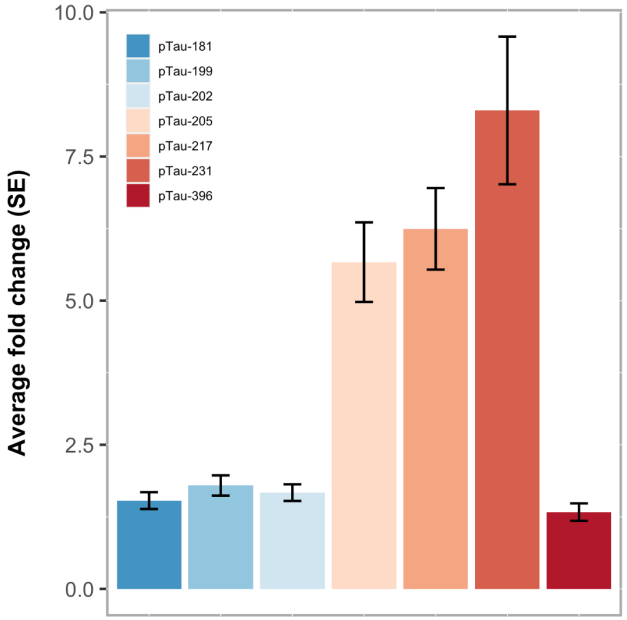






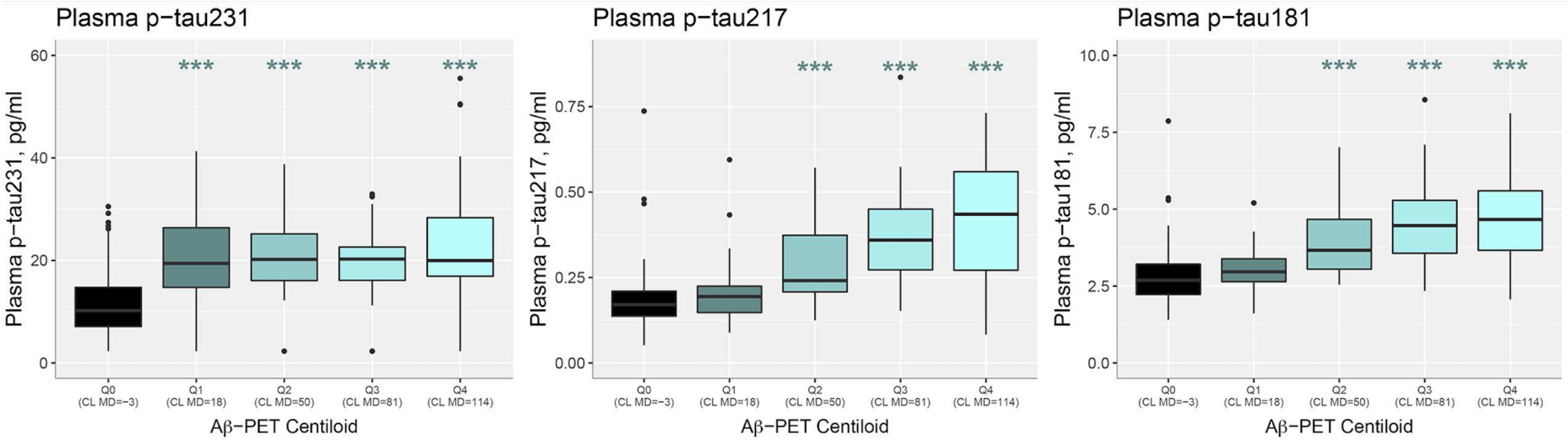






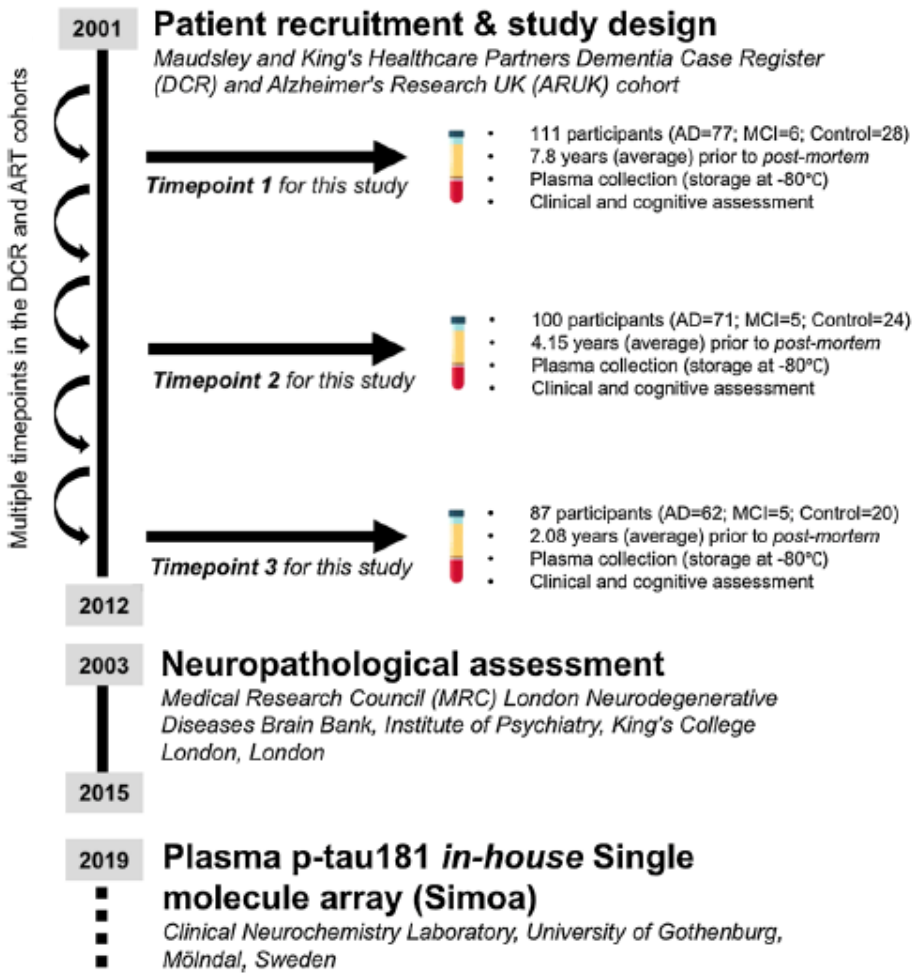
Gobom *et al.*, Mol Neurodegen. 2023

Different phospho-forms of tau can be measured in plasma



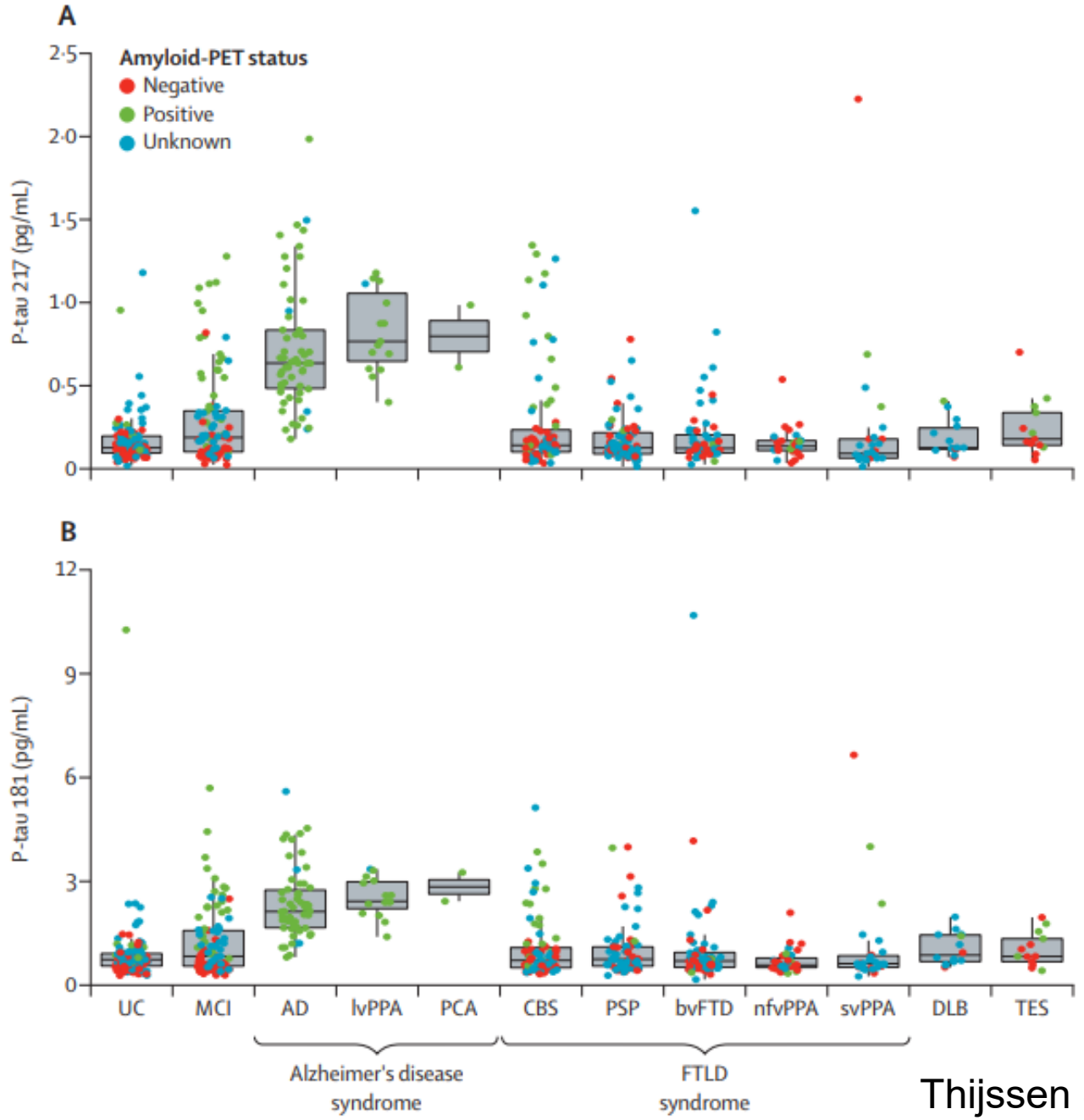
Ashton *et al.*, Nature Med. 2022

Plasma p-tau indicates AD pathology (including amyloid)



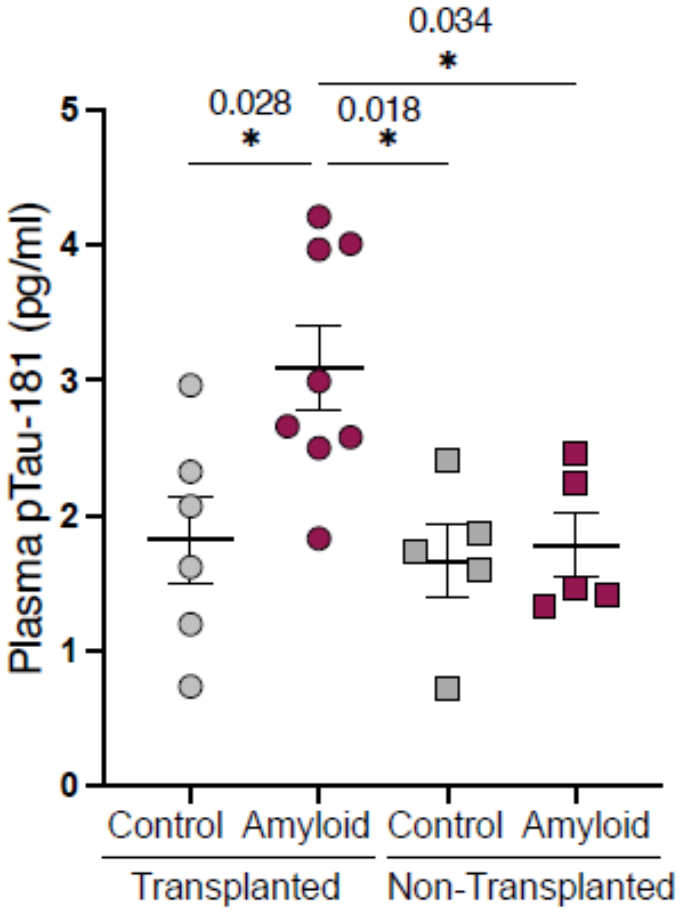
Lantero-Rodriguez et al., 2020 Acta Neuropathologica (PMID: 32720099)

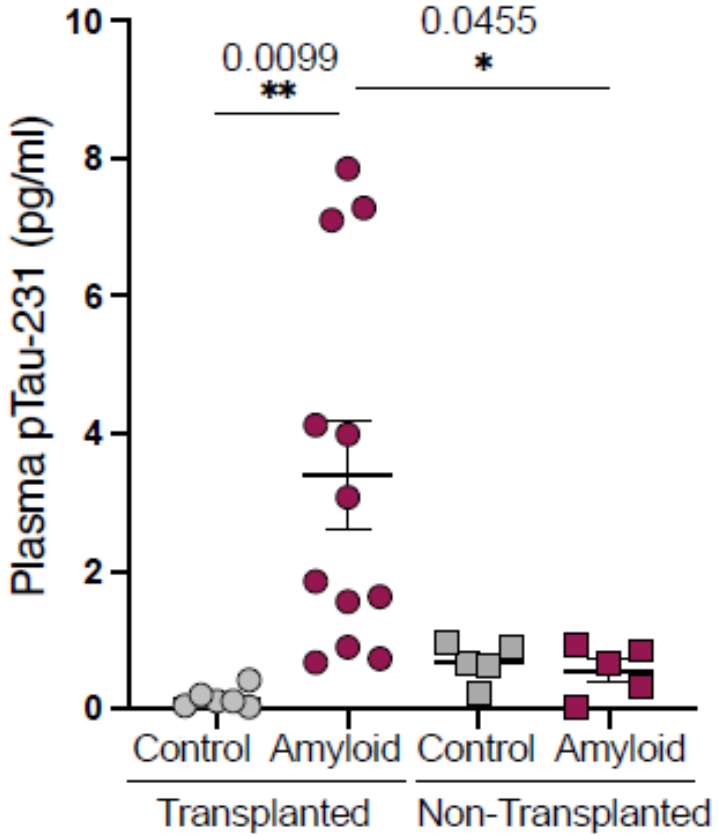
Plasma P-tau across neurodegenerative diseases



Thijssen *et al.* Lancet Neurology 2021

Plasma P-tau in relation to amyloid



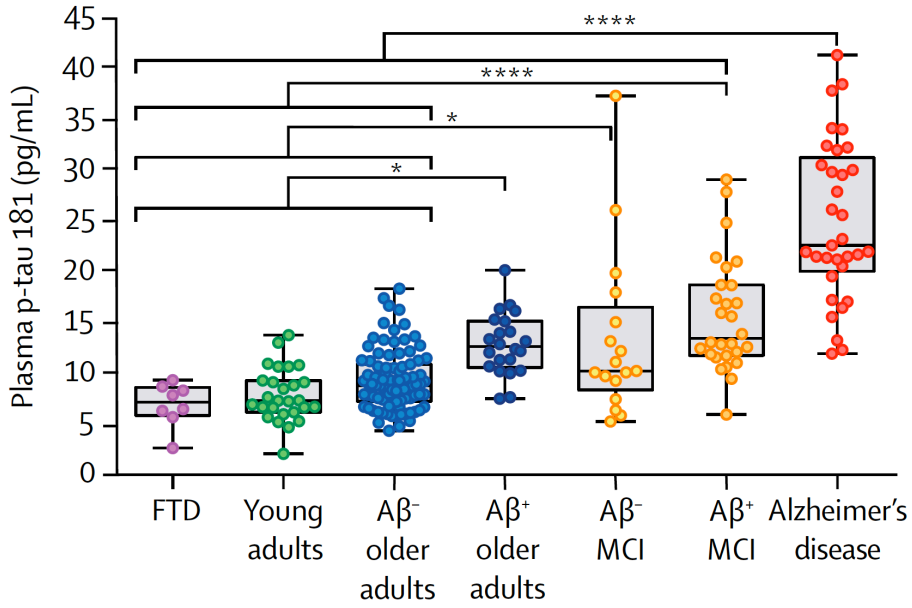


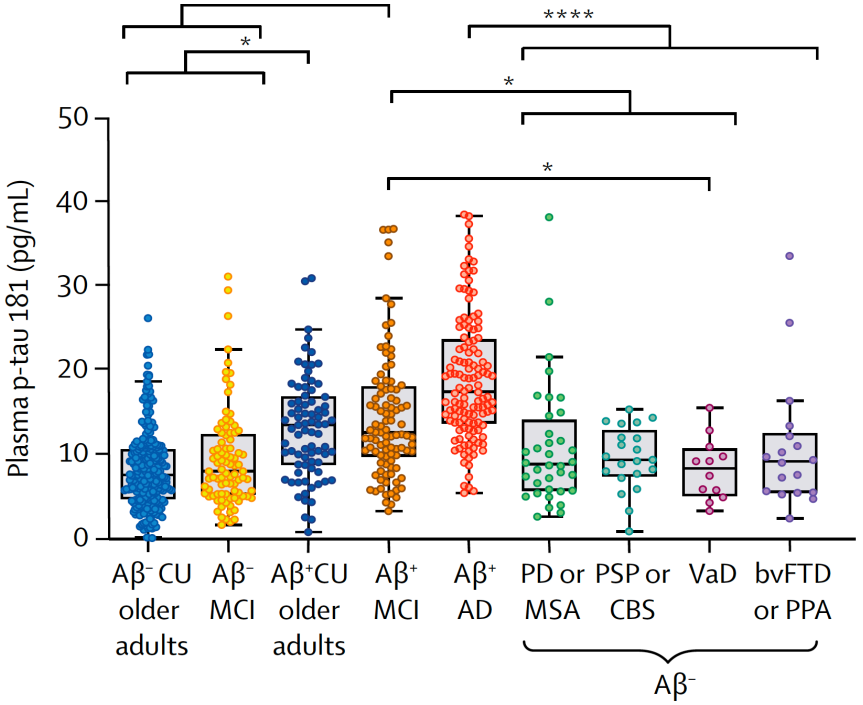
Balusu *et al*., Science 2023

Plasma p-tau in the Alzheimer's disease continuum

Blood phosphorylated tau 181 as a biomarker for Alzheimer's disease: a diagnostic performance and prediction modelling study using data from four prospective cohorts

Thomas K Karikari, Tharick A Pascoal*, Nicholas J Ashton, Shorena Janelidze, Andréa Lessa Benedet, Juan Lantero Rodriguez, Mira Chamoun, Melissa Savard, Min Su Kang, Joseph Therriault, Michael Schöll, Gassan Massarweh, Jean-Paul Soucy, Kina Höglund, Gunnar Brinkmalm, Niklas Mattsson, Sebastian Palmqvist, Serge Gauthier, Erik Stomrud, Henrik Zetterberg, Oskar Hansson†, Pedro Rosa-Neto†, Kaj Blennow†*





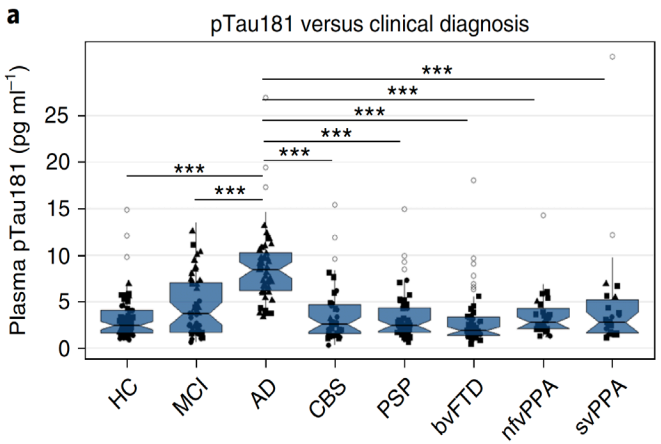
Karikari, Pascoal et al., 2020 Lancet Neurology (PMID: 32333900)

Plasma p-tau in the Alzheimer's disease continuum

nature medicine
ARTICLES
https://doi.org/10.1038/s41591-020-0762-2
Check for updates

Diagnostic value of plasma phosphorylated tau181 in Alzheimer's disease and frontotemporal lobar degeneration

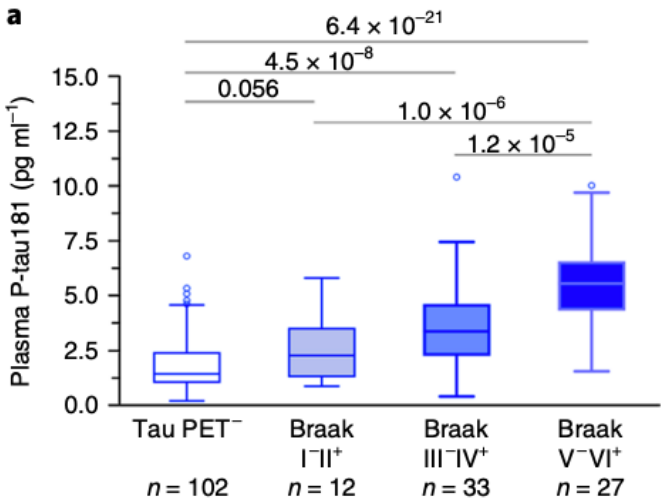
Elisabeth H. Thijssen[1,2], Renaud La Joie[1], Amy Wolf[1], Amelia Strom[1], Ping Wang[1], Leonardo Iaccarino[1], Viktoriya Bourakova[1], Yann Cobigo[1], Hilary Heuer[1], Salvatore Spina[1], Lawren VandeVrede[1], Xiyun Chai[3], Nicholas K. Proctor[3], David C. Airey[3], Sergey Shcherbinin[3], Cynthia Duggan Evans[3], John R. Sims[3], Henrik Zetterberg[4,5,6,7], Kaj Blennow[4,5], Anna M. Karydas[1], Charlotte E. Teunissen[2], Joel H. Kramer[1], Lea T. Grinberg[1,8], William W. Seeley[1,8], Howie Rosen[1], Bradley F. Boeve[9], Bruce L. Miller[1], Gil D. Rabinovici[1,10], Jeffrey L. Dage[3], Julio C. Rojas[1], Adam L. Boxer[1]✉ and Advancing Research and Treatment for Frontotemporal Lobar Degeneration (ARTFL) investigators*



nature medicine
ARTICLES
https://doi.org/10.1038/s41591-020-0755-1

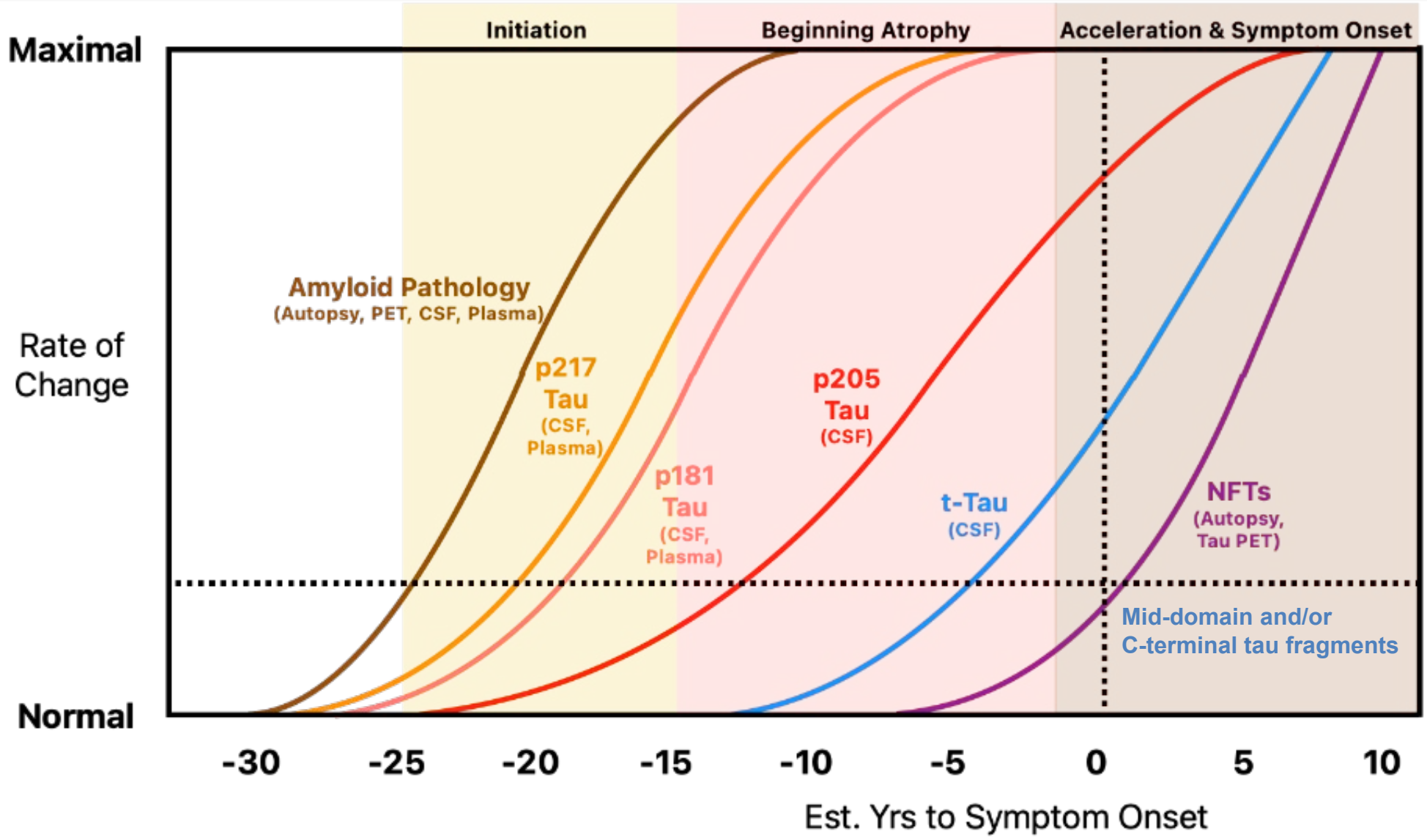
Plasma P-tau181 in Alzheimer's disease: relationship to other biomarkers, differential diagnosis, neuropathology and longitudinal progression to Alzheimer's dementia

Shorena Janelidze[1,13]*, Niklas Mattsson[1,2,3,13], Sebastian Palmqvist[1,2], Ruben Smith[1,2], Thomas G. Beach[4], Geidy E. Serrano[4], Xiyun Chai[5], Nicholas K. Proctor[5], Udo Eichenlaub[6], Henrik Zetterberg[7,8,9,10], Kaj Blennow[7,8], Eric M. Reiman[11], Erik Stomrud[1,12], Jeffrey L. Dage[5] and Oskar Hansson[1,12]*



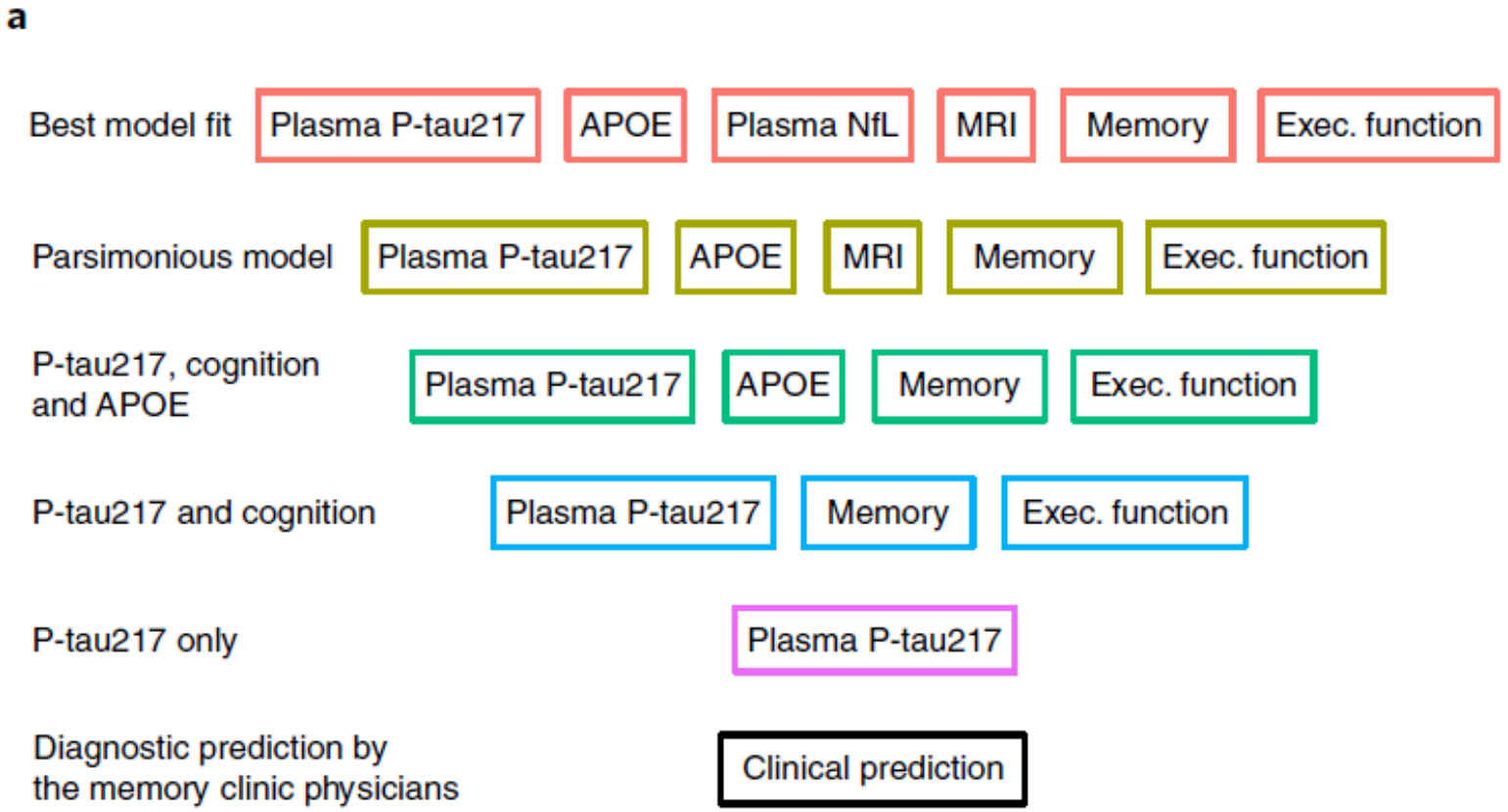
Thijssen et al., 2020 Nature Medicine (PMID: 32123386) / Janelidze et al., 2020 Nature Medicine (PMID: 32123385) / Mielke et al., 2018 Alzheimer's & Dementia (PMID: 29626426)

Biomarker-based staging of AD



Kolster *et al*., manuscript in preparation

Plasma tests as clinical tools to predict AD-type dementia in patients with subjective or mild cognitive impairment

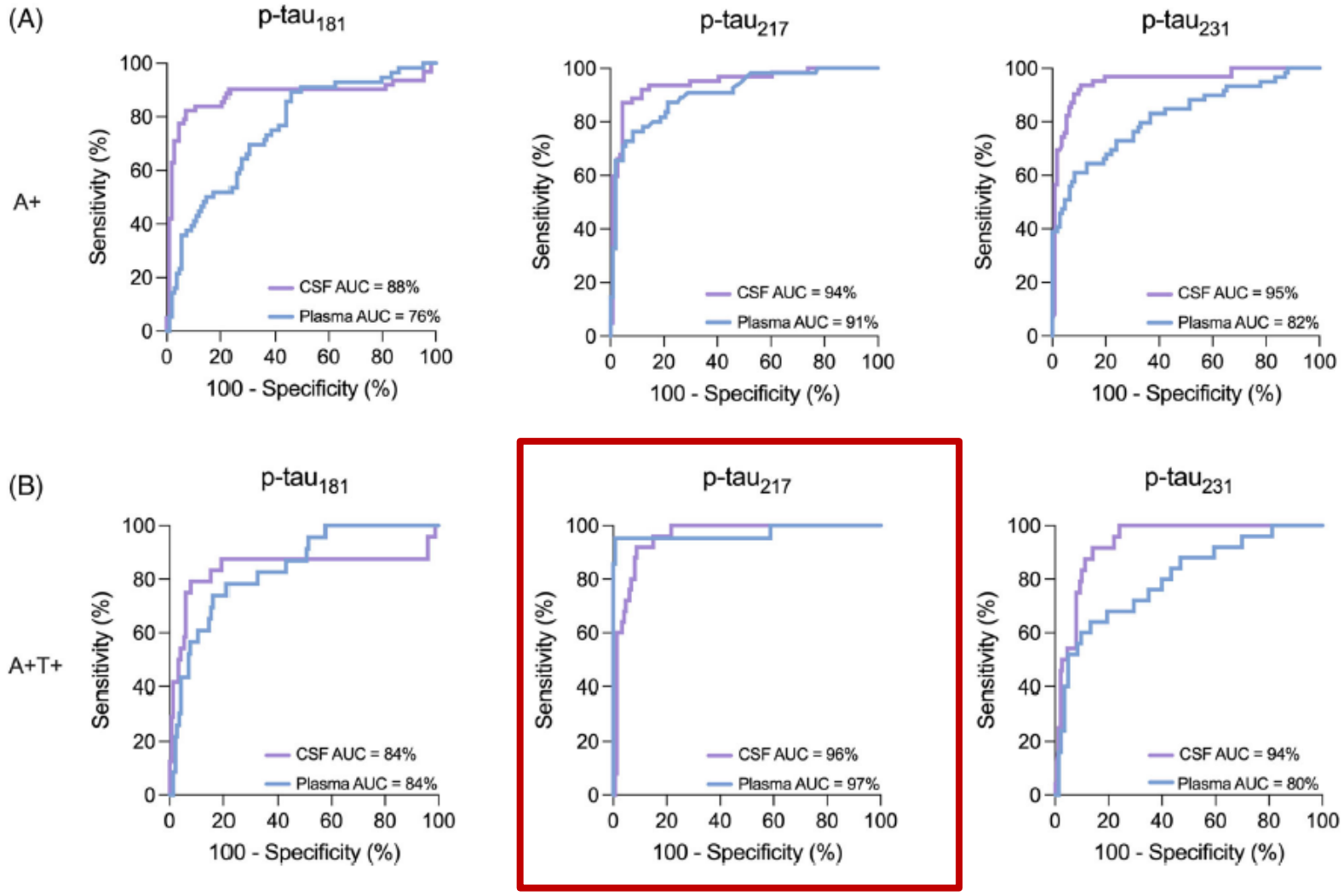


	AUC (95% CI)	AIC
Best model fit	0.92 (0.89–0.95)**	159
Parsimonious model	0.92 (0.88–0.95)**	161
P-tau217, cognition and APOE	0.90 (0.86–0.94)**	166
P-tau217 and cognition	0.89 (0.84–0.93)**	171
P-tau217 only	0.81 (0.75–0.87)*	207
Diagnostic prediction by the memory clinic physicians	0.72 (0.65–0.78)	228

*$P < 0.05$; **$P < 0.001$ vs the clinical prediction

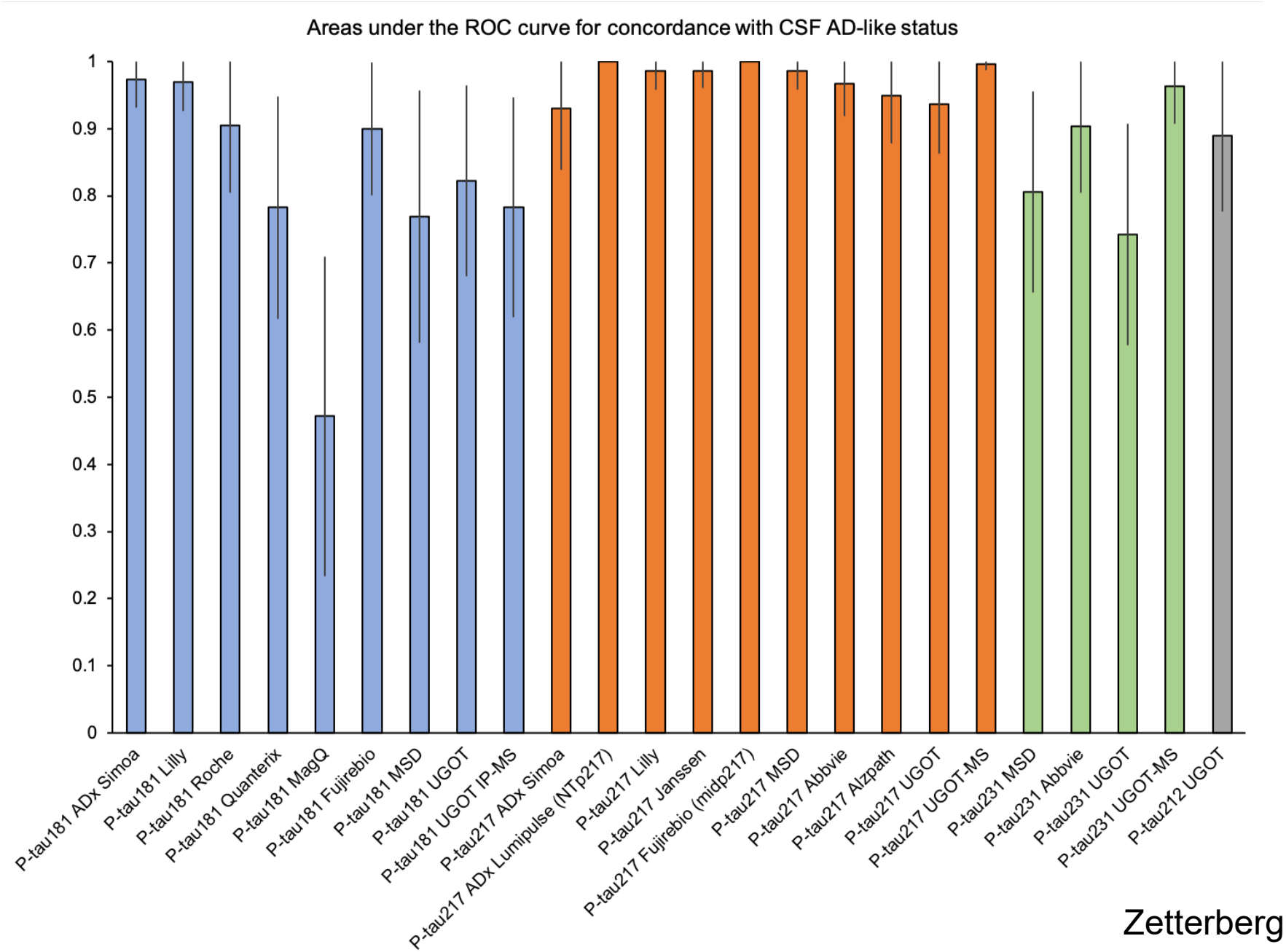
Palmqvist *et al.*, Nature Med. 2021

Equivalence of plasma p-tau217 with cerebrospinal fluid in the diagnosis of Alzheimer's disease



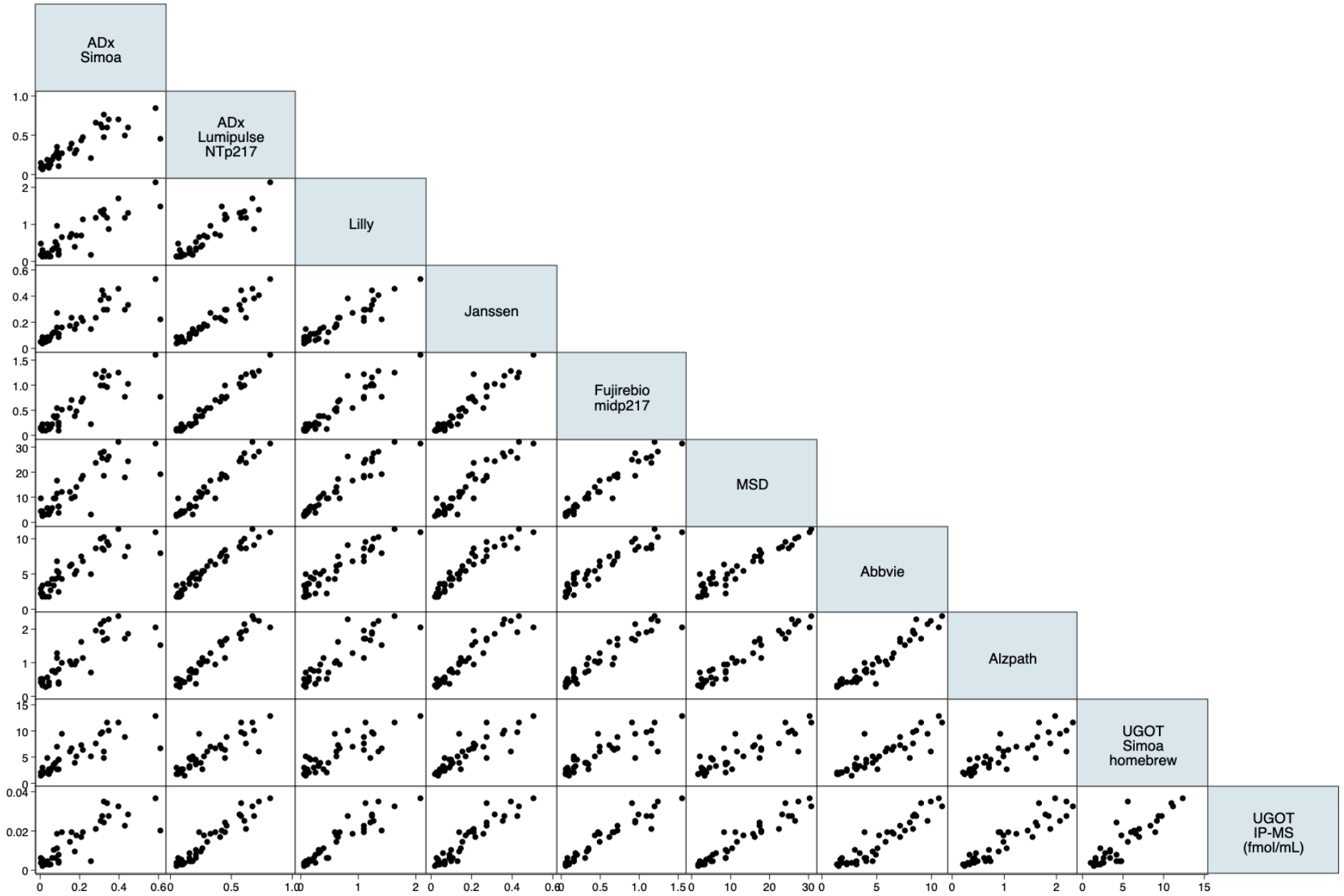
Therriault *et al*., A&D, 2023

The Alzheimer's Association Round Robin Study



Zetterberg *et al.*, unpublished

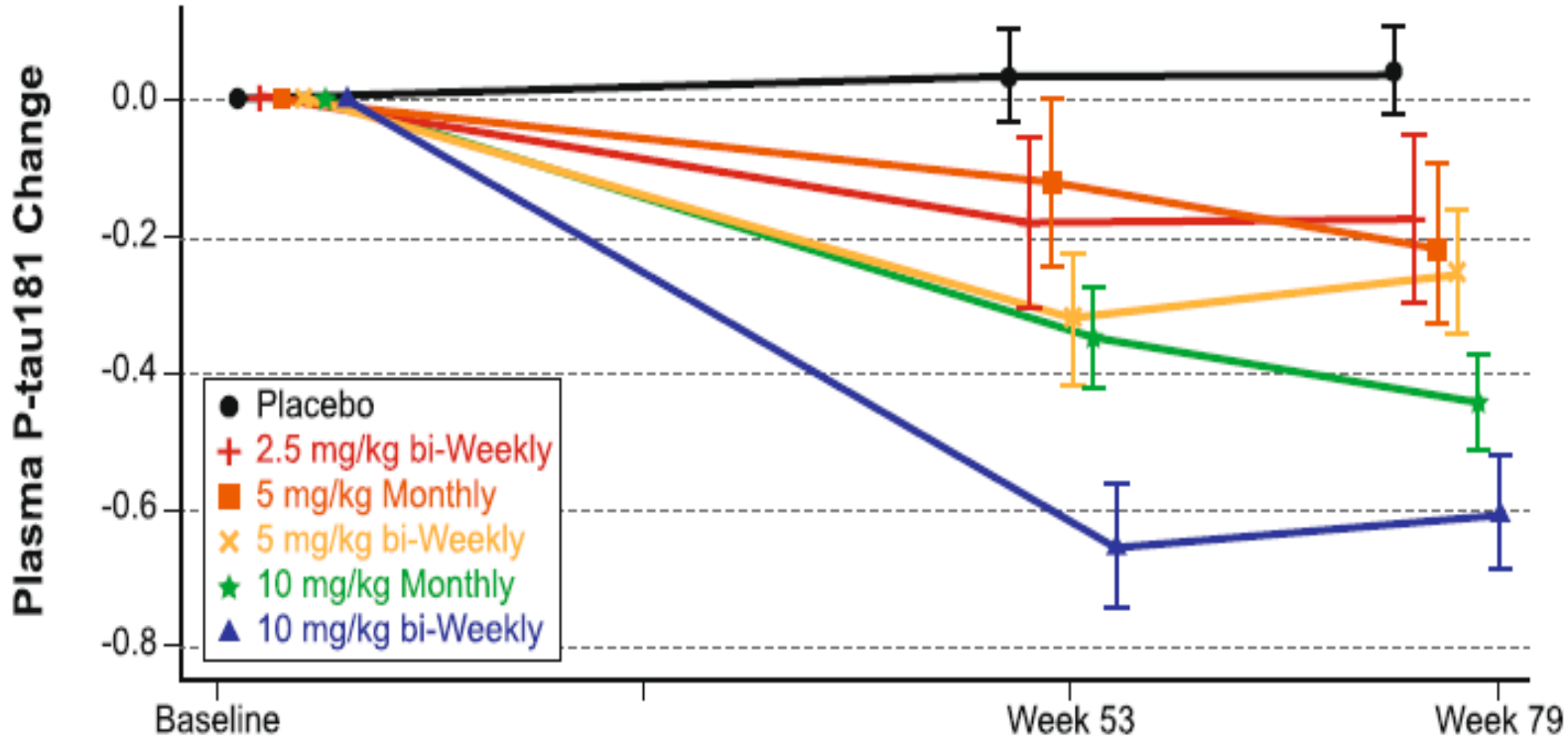
The Alzheimer's Association Round Robin Study



Zetterberg *et al*., unpublished

Plasma P-tau markers in clinical trials: lecanemab

Results from the lecanemab proof-of-concept 201 core trial

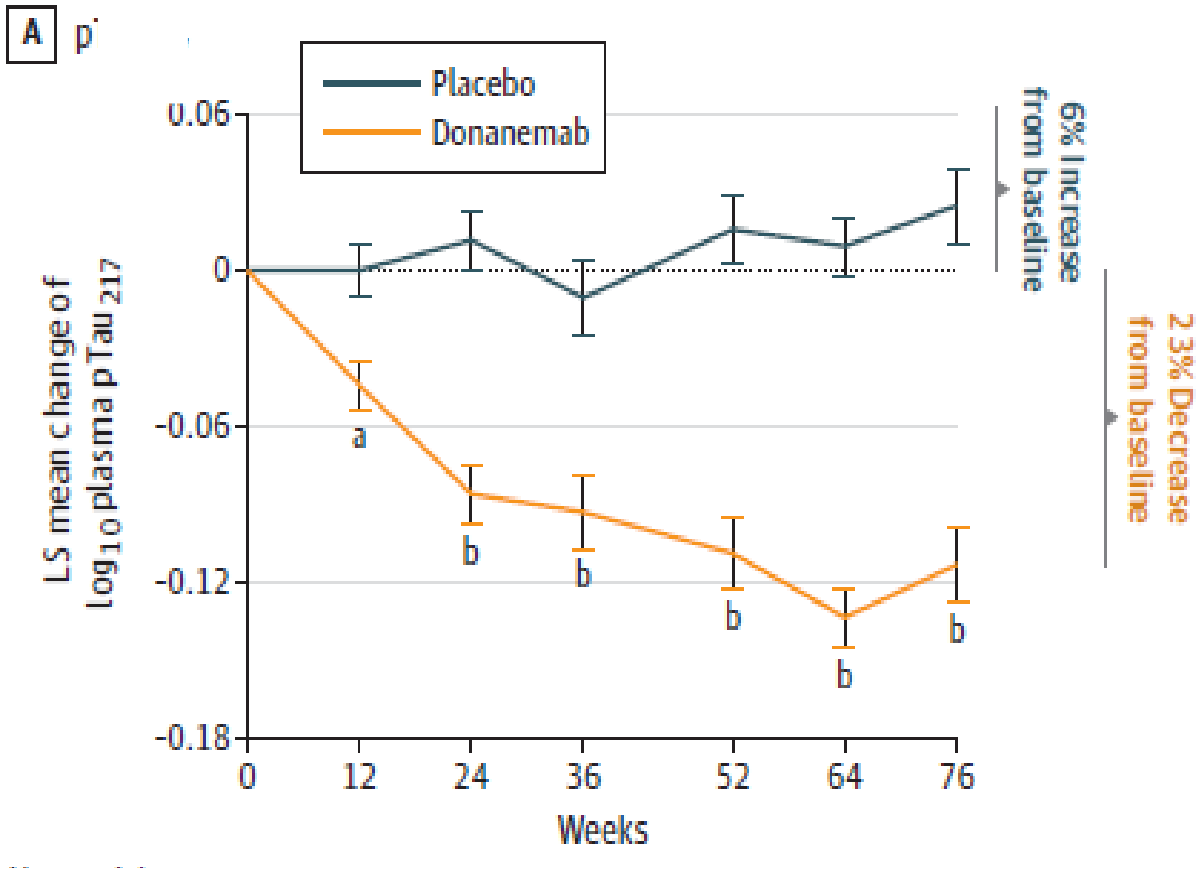


McDade *et al*., AR&T, 2022

Plasma P-tau markers in clinical trials: donanemab

Results *TRAILBLAZER-ALZ*:

- Reduced cognitive decline in iADRS after 76 weeks
- Plasma pTau217 decrease from baseline in the treatment arm



Pontecorvo *et al.*, JAMA Neurol., 2022

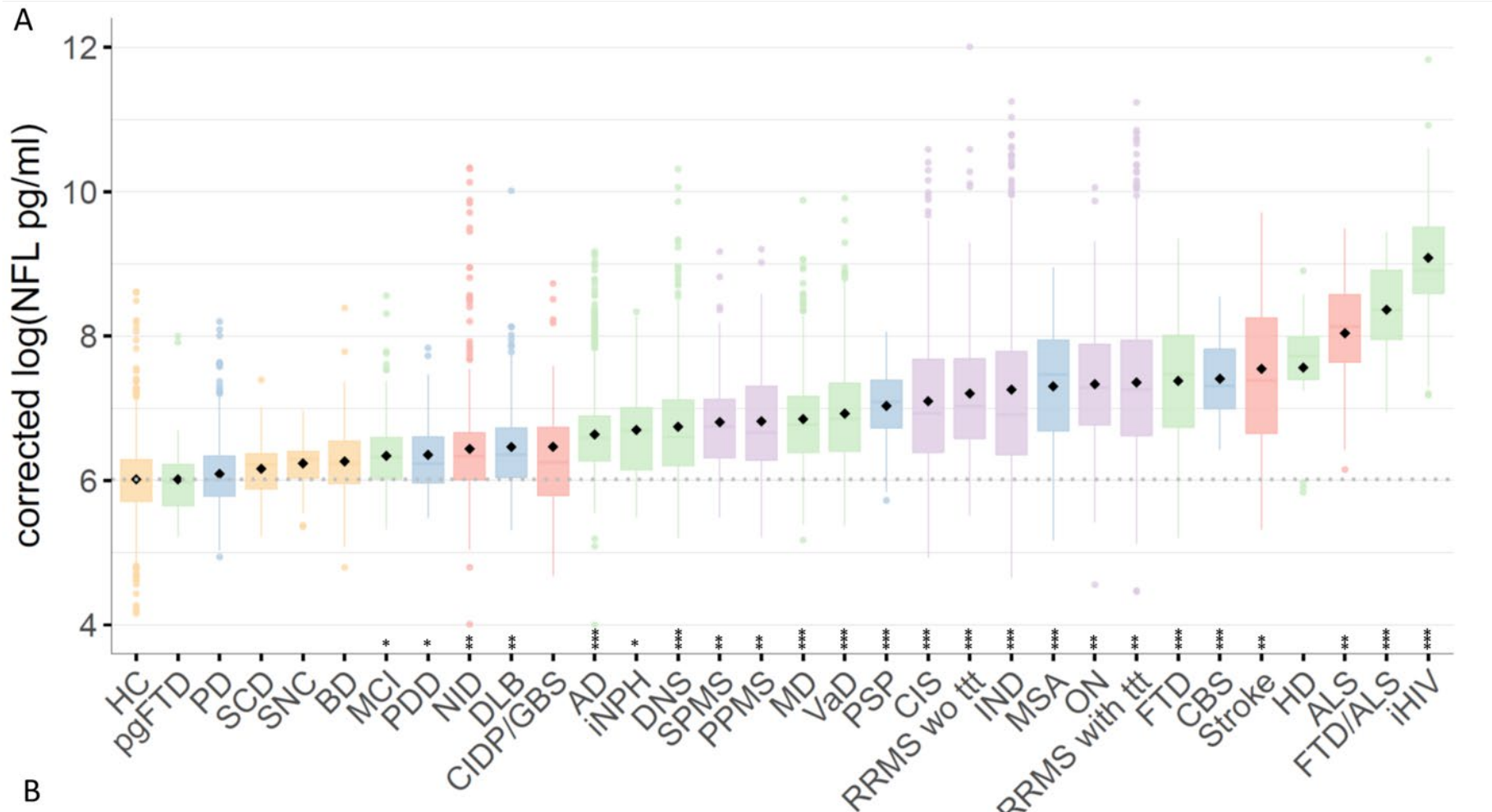
Diagnosing AD-type tau pathophysiology with a blood test: are we there yet?

Group level enrichment/screening: Yes

Individual diagnostics: Yes, at least we are getting there

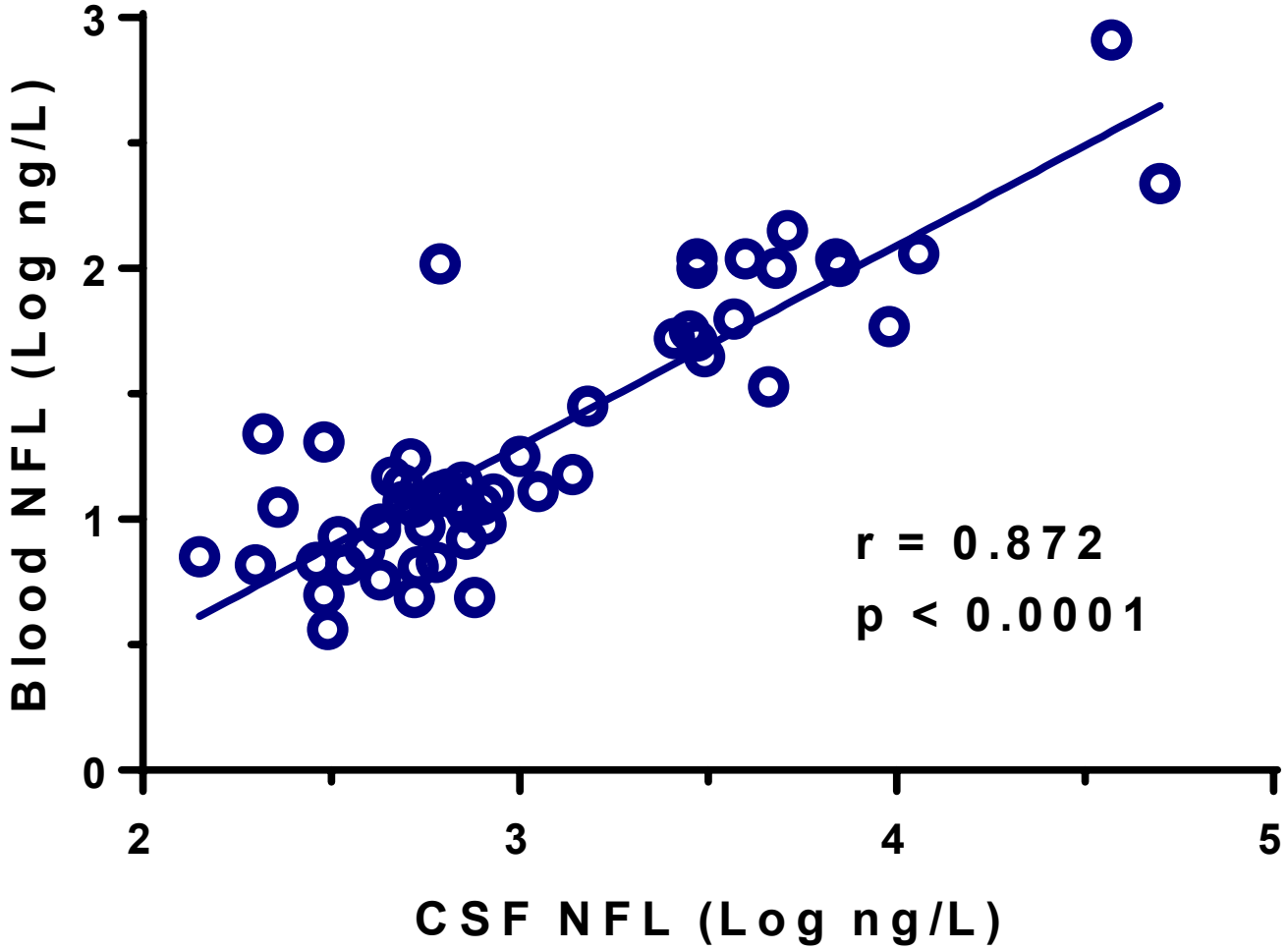
N = neurodegeneration

CSF NfL across neurodegenerative diseases



Bridel C *et al.*, JAMA Neurol 2019

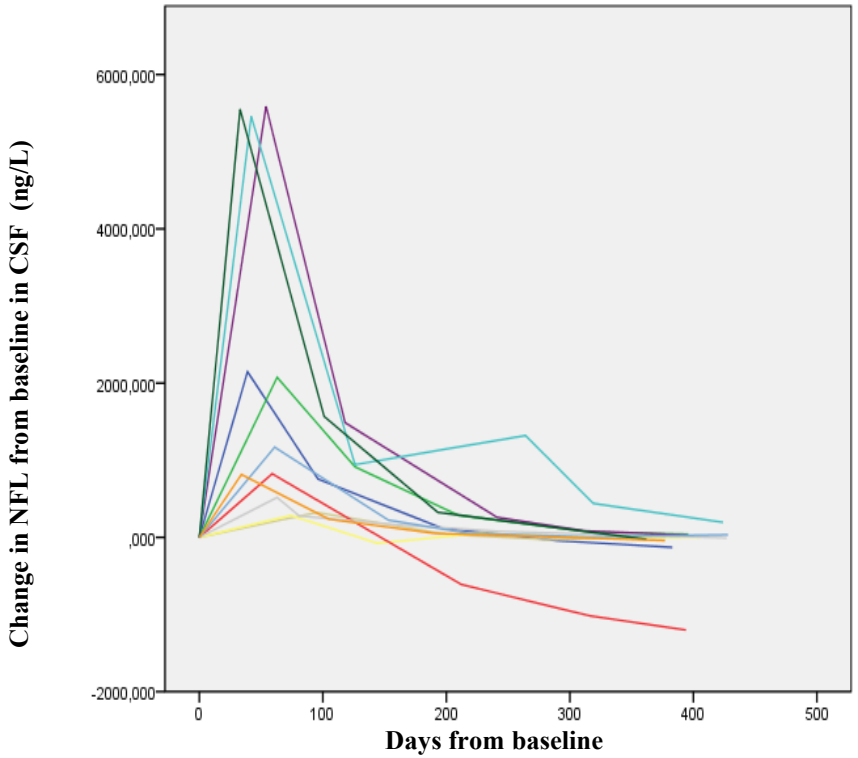
Plasma NfL correlates with CSF NfL…



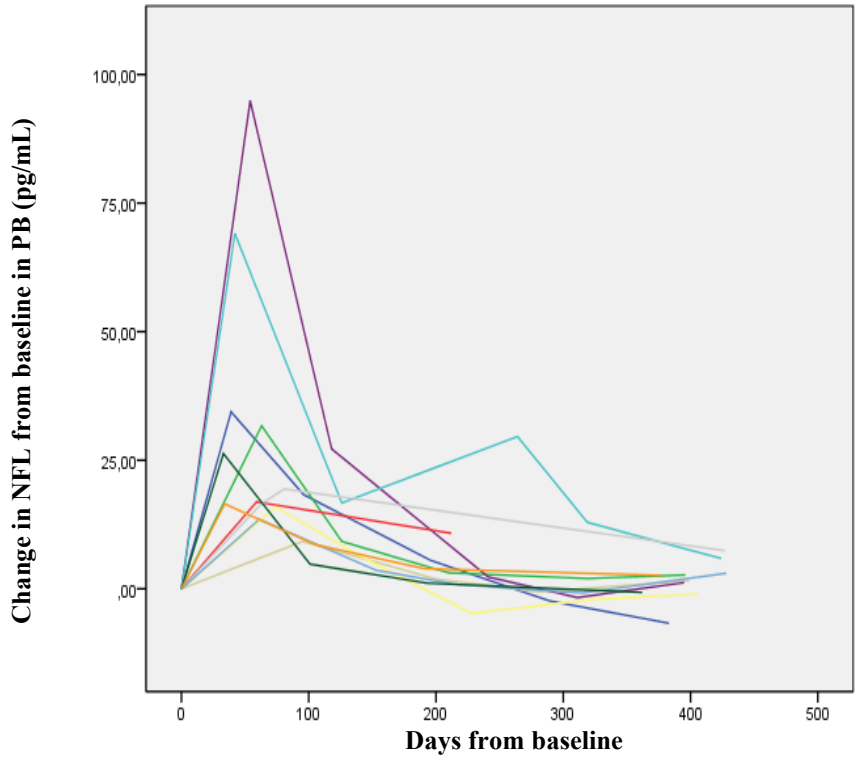
Gisslén *et al*., 2015

…and shows similar dynamics

A

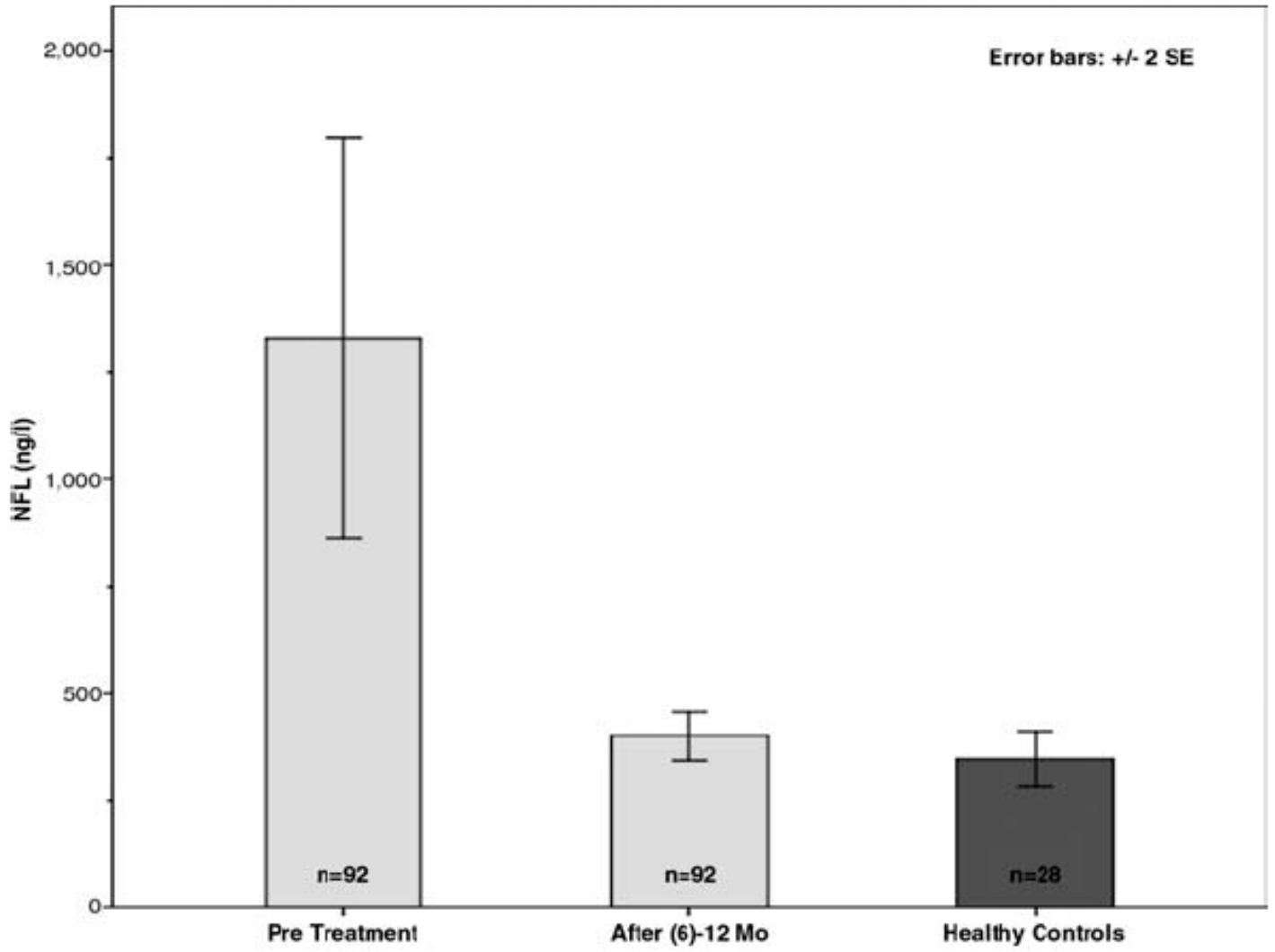


B



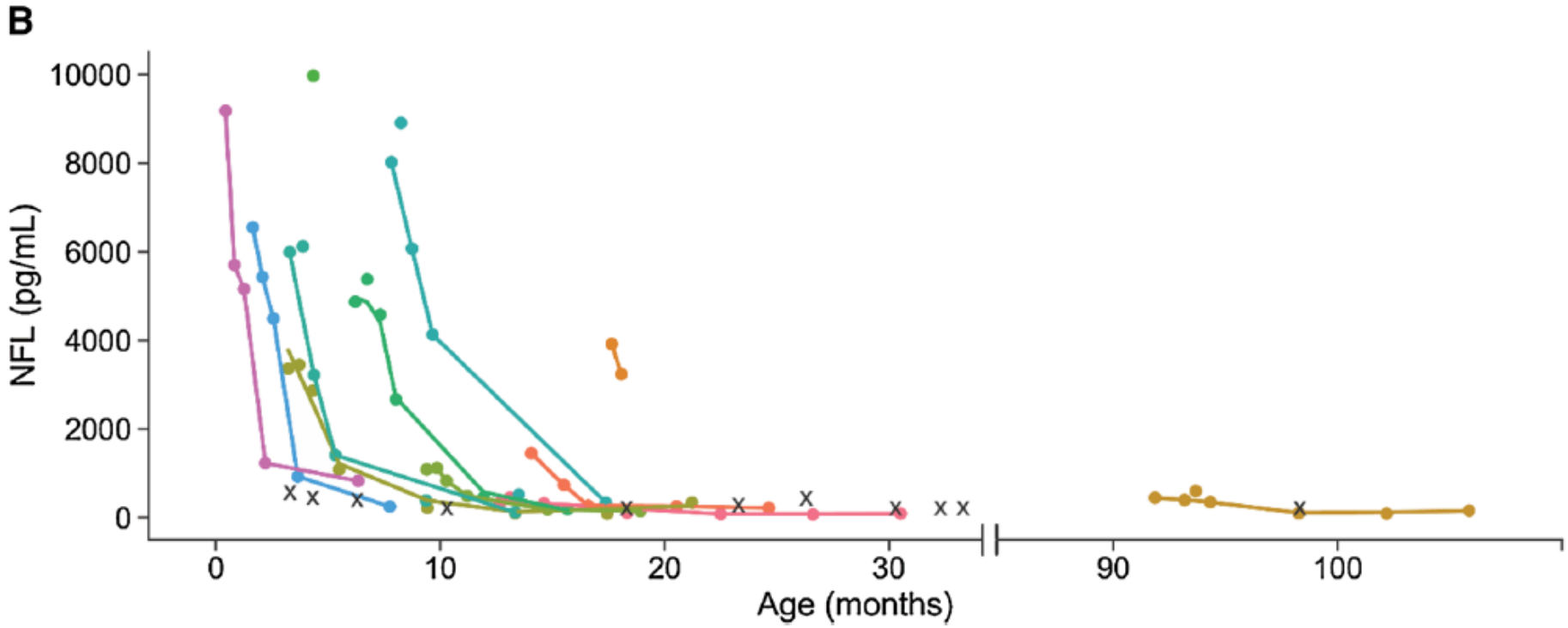
Bergman *et al*., Neurol Neuroimmunol Neuroinflamm. 2016

CSF NfL dynamics – normalization in response to successful treatment



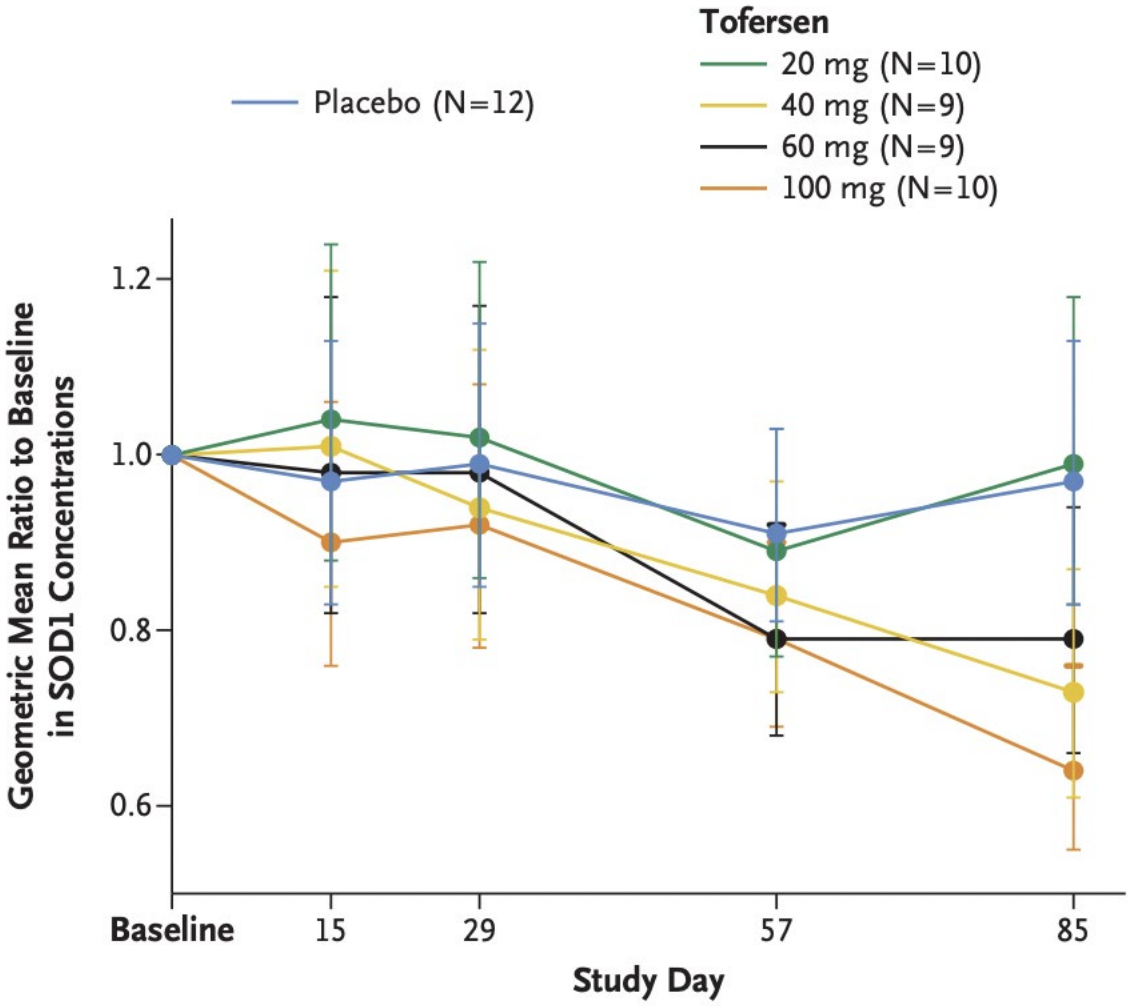
Gunnarsson M et al. Axonal damage in relapsing multiple sclerosis is markedly reduced by natalizumab. *Ann Neurol* 2011; 69: 83–89.

CSF NfL dynamics – before and during treatment with Spinraza (nusinersen) in spinal muscular atrophy



Olsson B *et al.*, J Neurol 2019

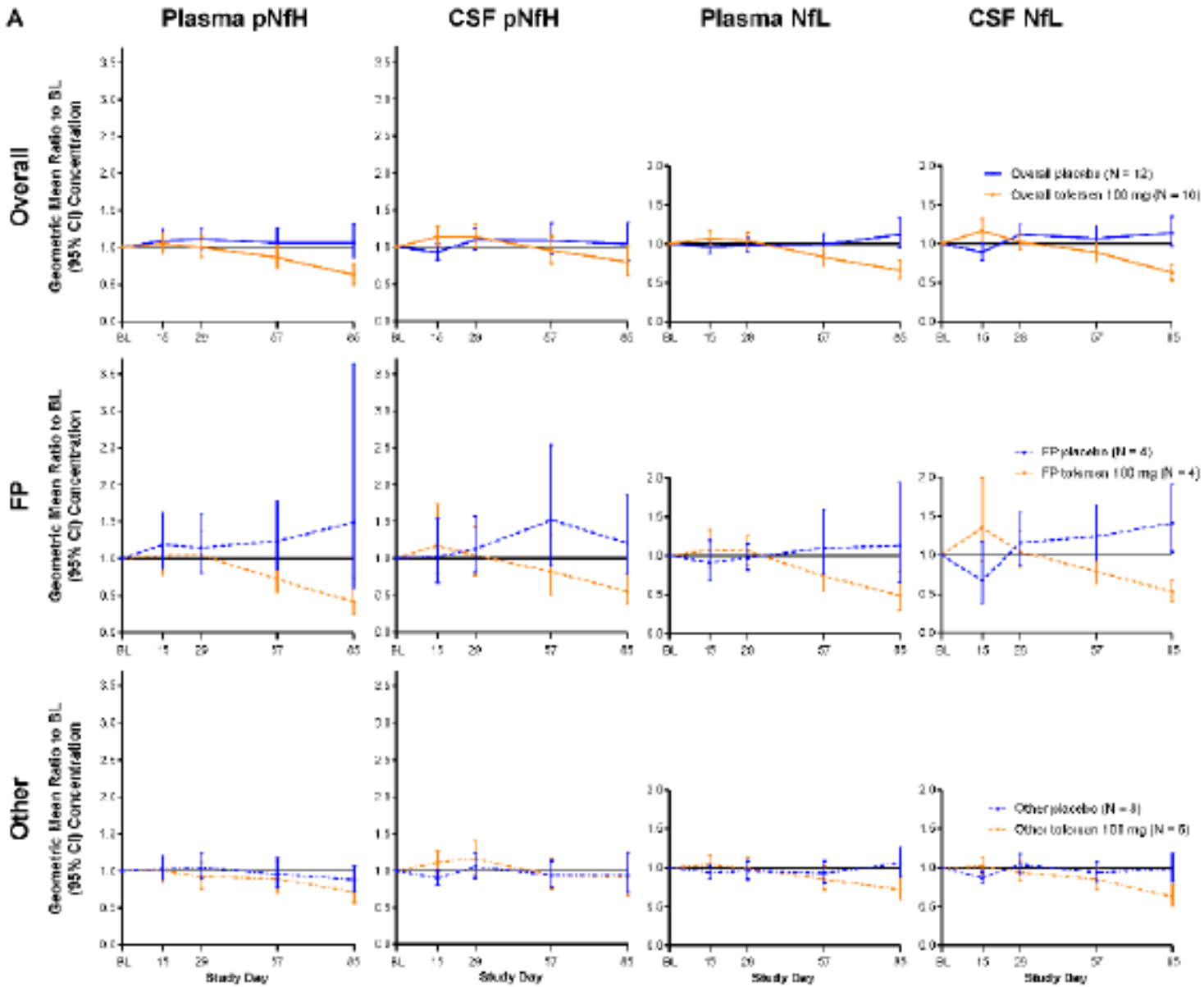
NfL as a pharmacodynamic marker in ALS



The NEW ENGLAND JOURNAL of MEDICINE

Phase 1–2 Trial of Antisense Oligonucleotide Tofersen for *SOD1* ALS

T. Miller, M. Cudkowicz, P.J. Shaw, P.M. Andersen, N. Atassi, R.C. Bucelli, A. Genge, J. Glass, S. Ladha, A.L. Ludolph, N.J. Maragakis, C.J. McDermott, A. Pestronk, J. Ravits, F. Salachas, R. Trudell, P. Van Damme, L. Zinman, C.F. Bennett, R. Lane, A. Sandrock, H. Runz, D. Graham, H. Houshyar, A. McCampbell, I. Nestorov, I. Chang, M. McNeill, L. Fanning, S. Fradette, and T.A. Ferguson



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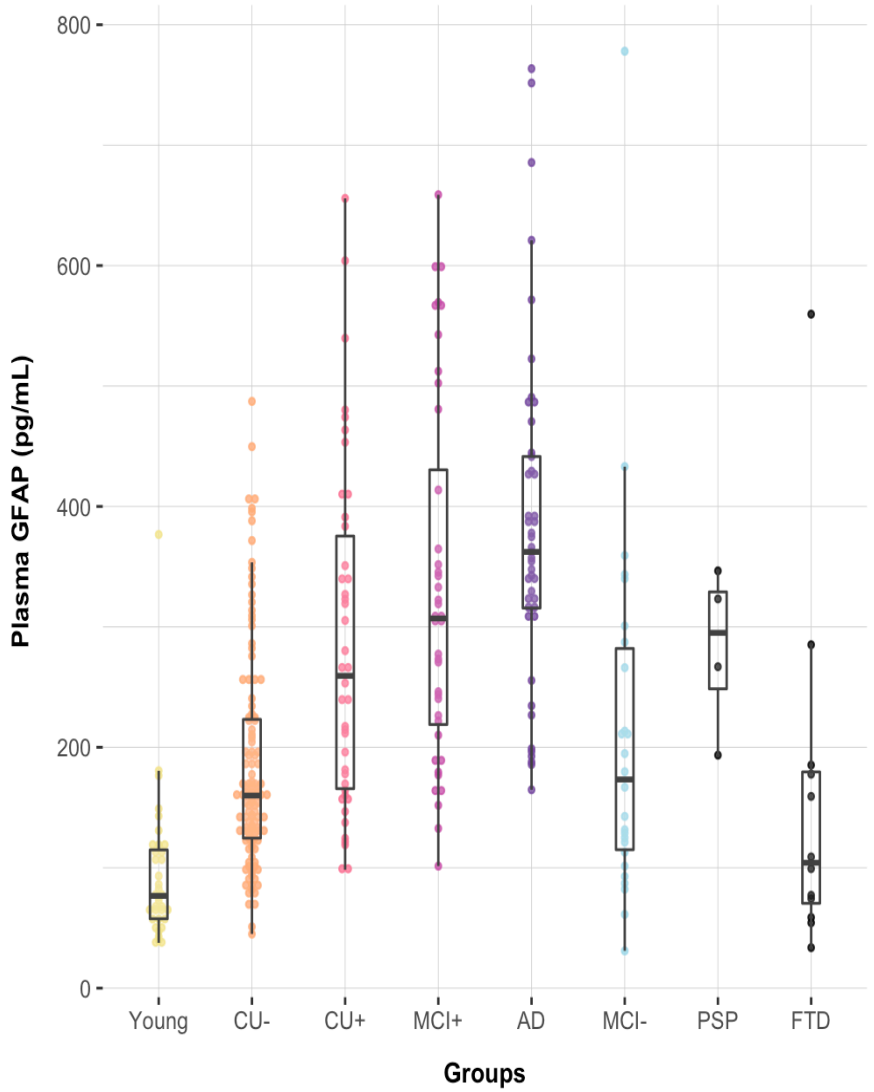
Diagnosing neurodegeneration with a blood sample: are we there yet?

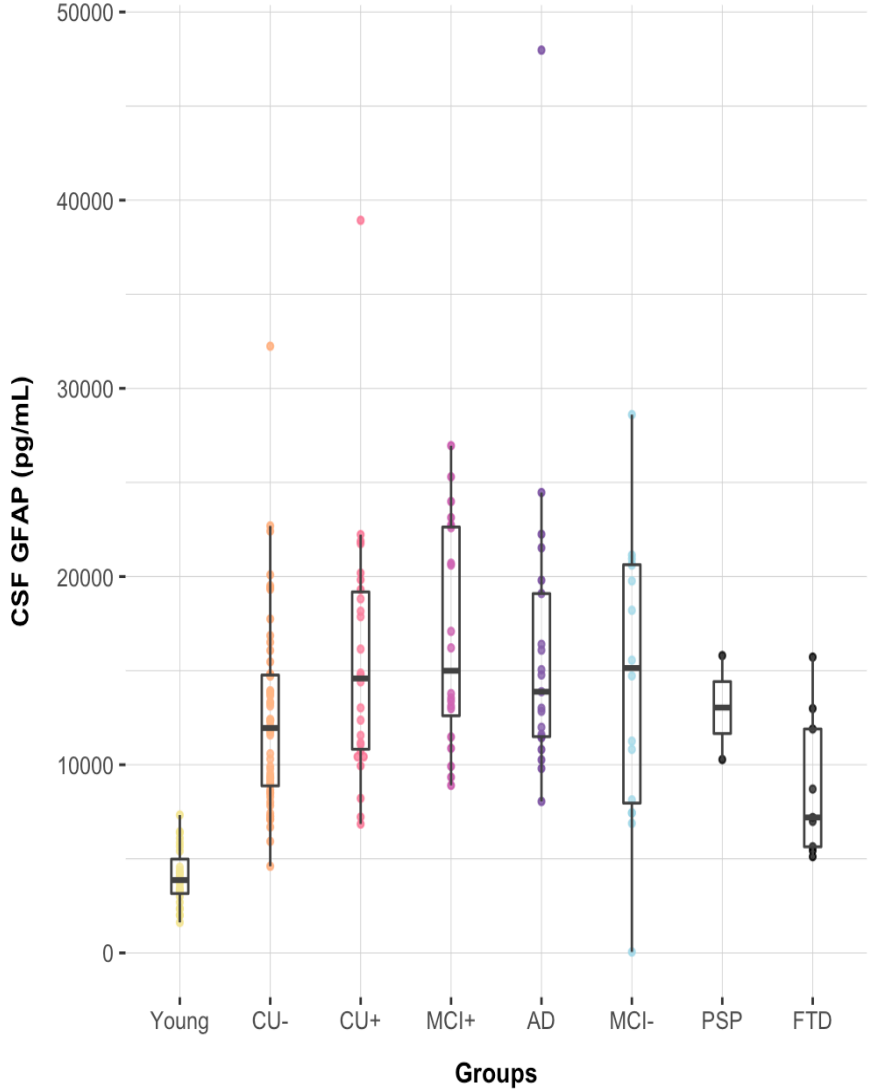
Group level enrichment/screening: Yes

Individual diagnostics: Yes, but CSF is probably a little bit more sensitive

G = glial activation

Glial fibrillary acidic protein (GFAP) – an astrocytic activation marker that works better in plasma than in CSF





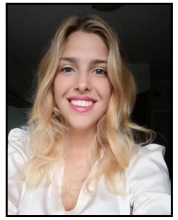
Benedet *et al.*, 2022

Several glial markers in CSF

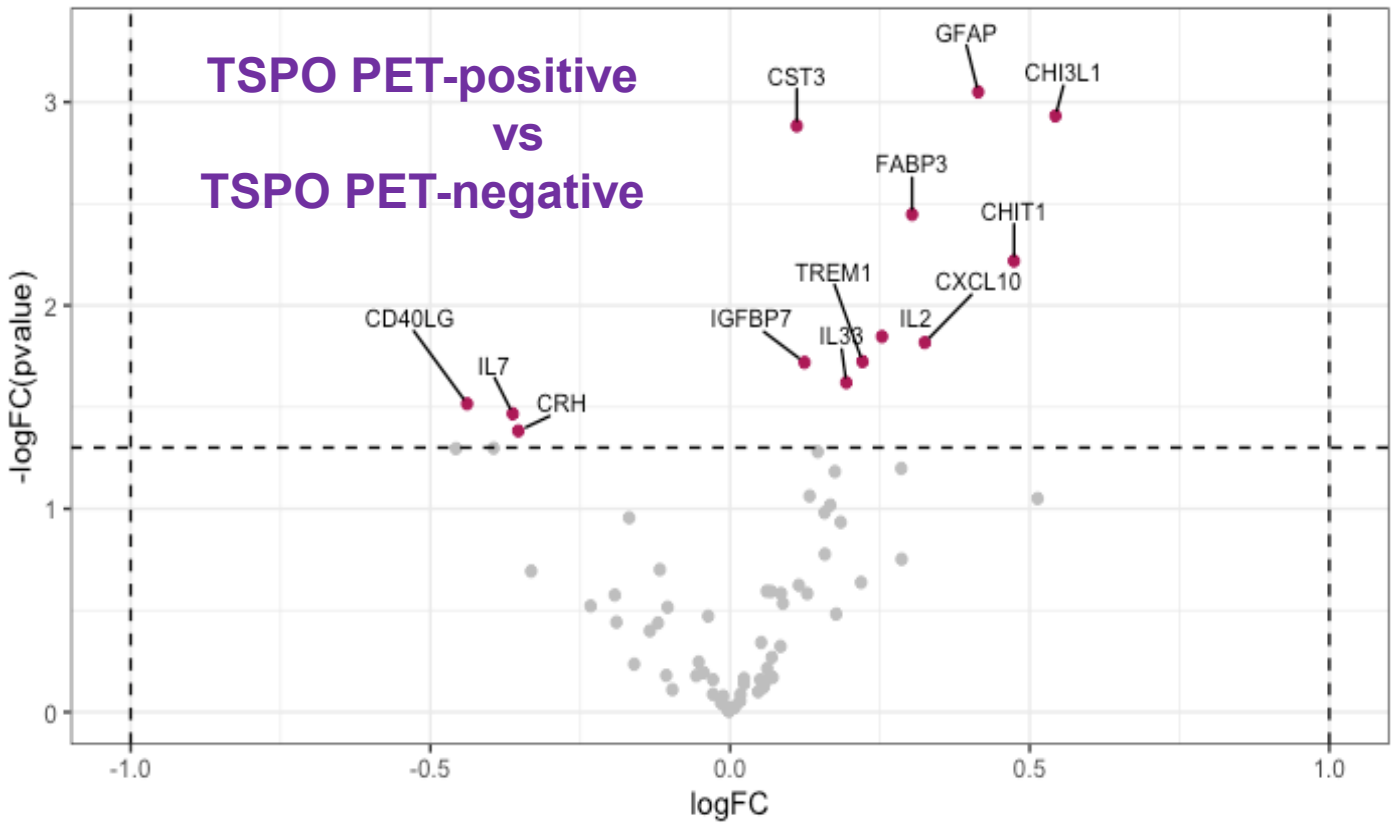
- Osteopontin (SPP1)
- sTREM2

...harder in blood (easy to measure but do not reflect the CNS)

Glial activation—additional plasma biomarker candidates with CNS relevance

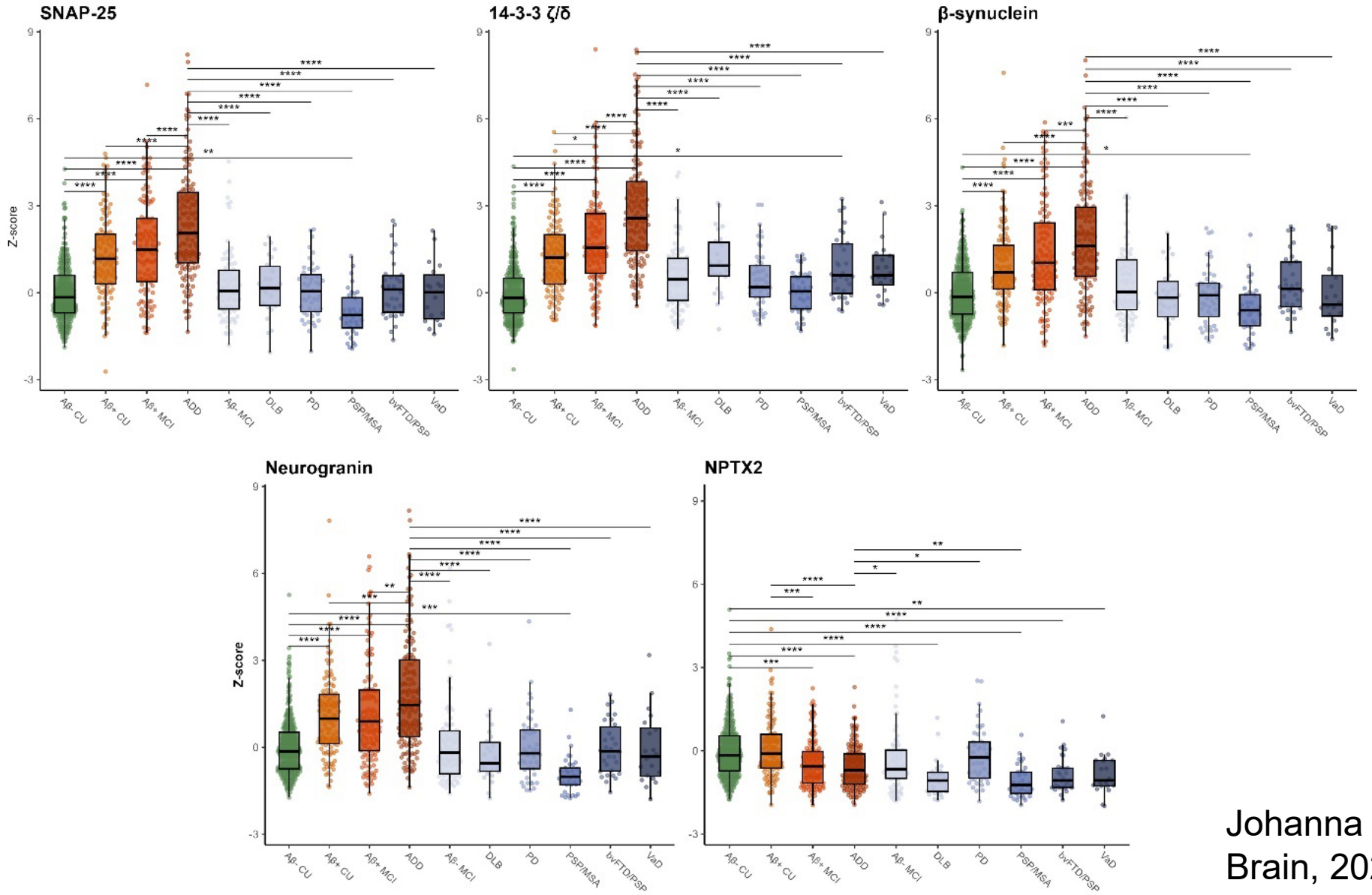


Ilaria Pola, Ph.D. student



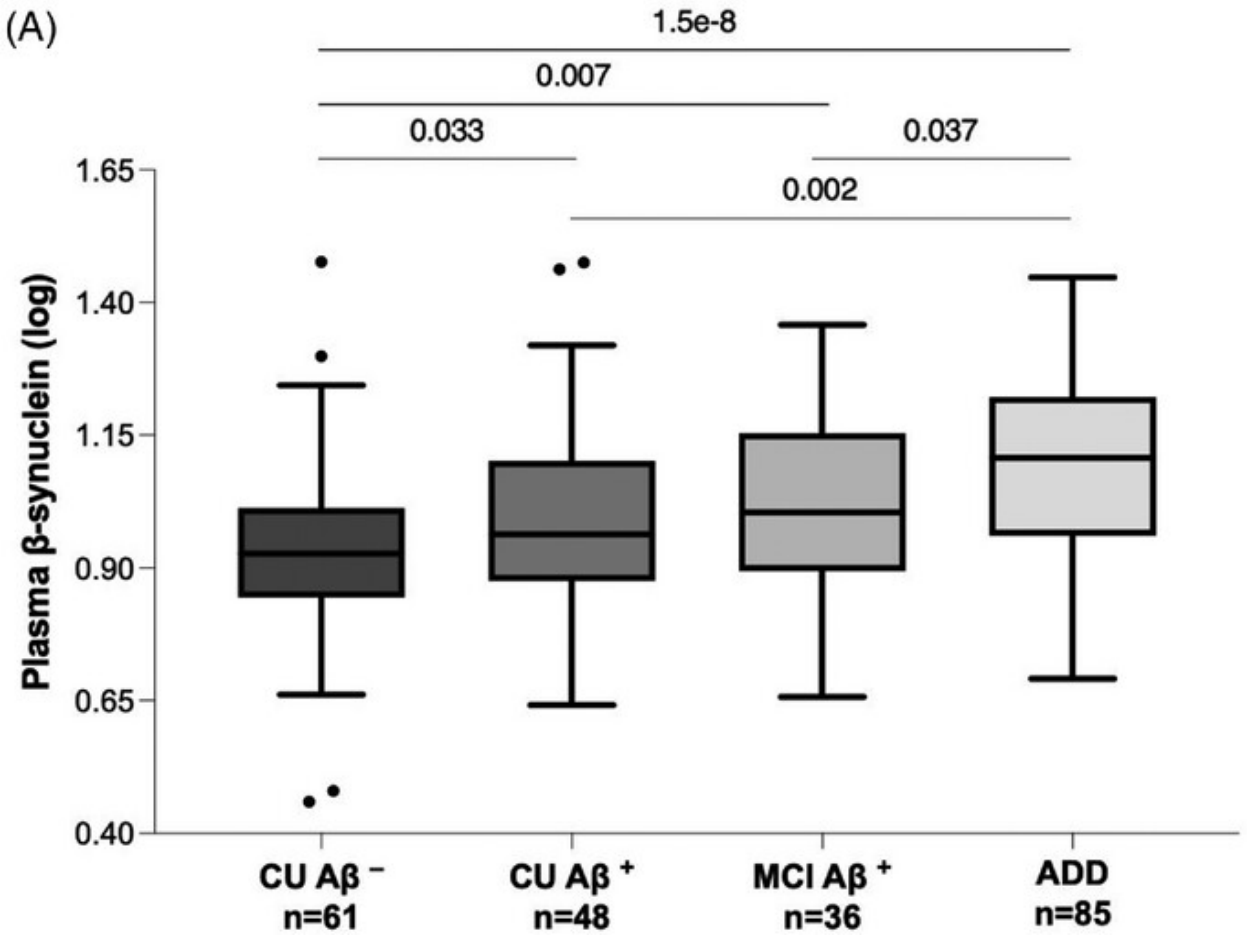
Synaptic pathology

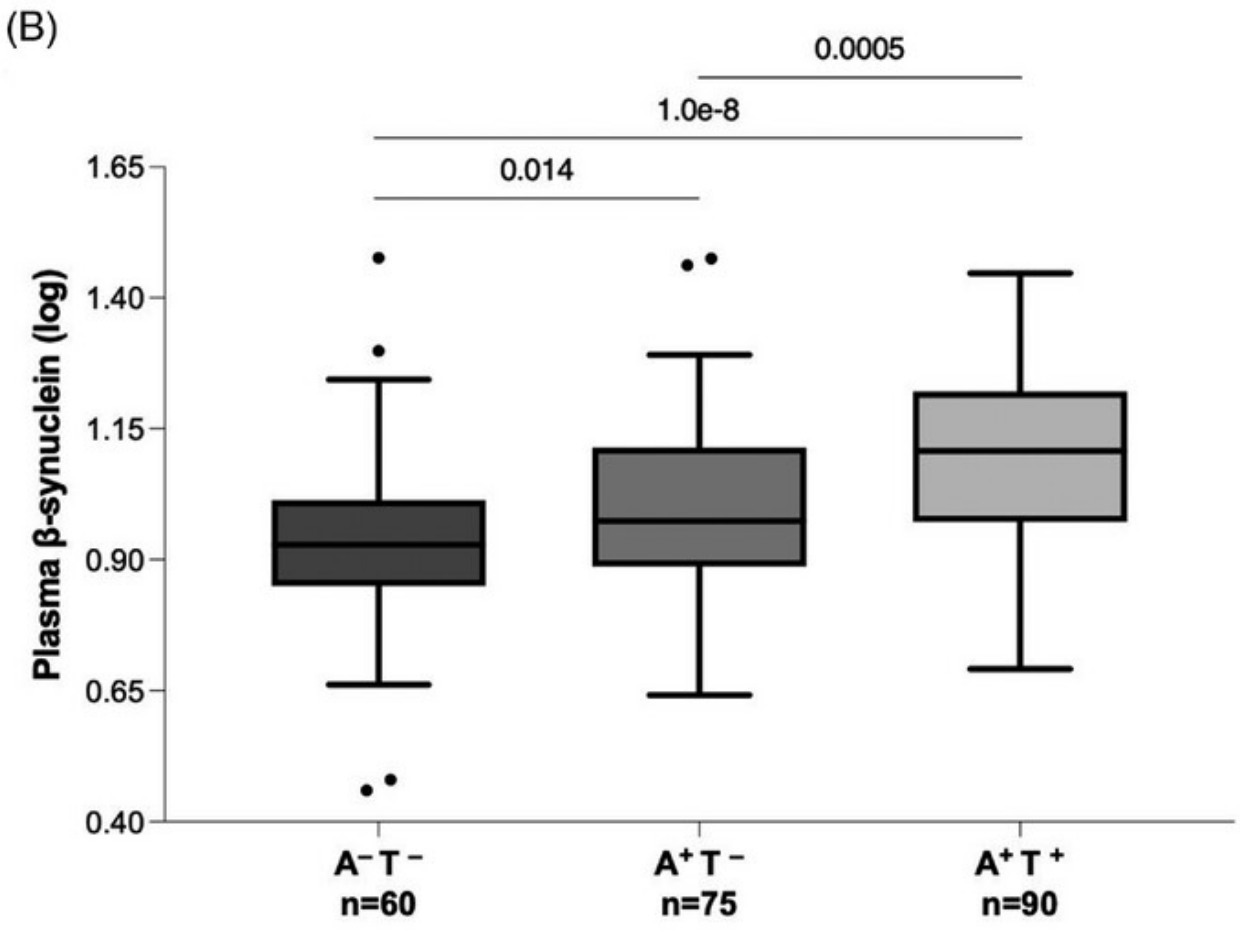
Synaptic markers in CSF



Johanna Nilsson *et al.*, Brain, 2024

β-synuclein promising in blood

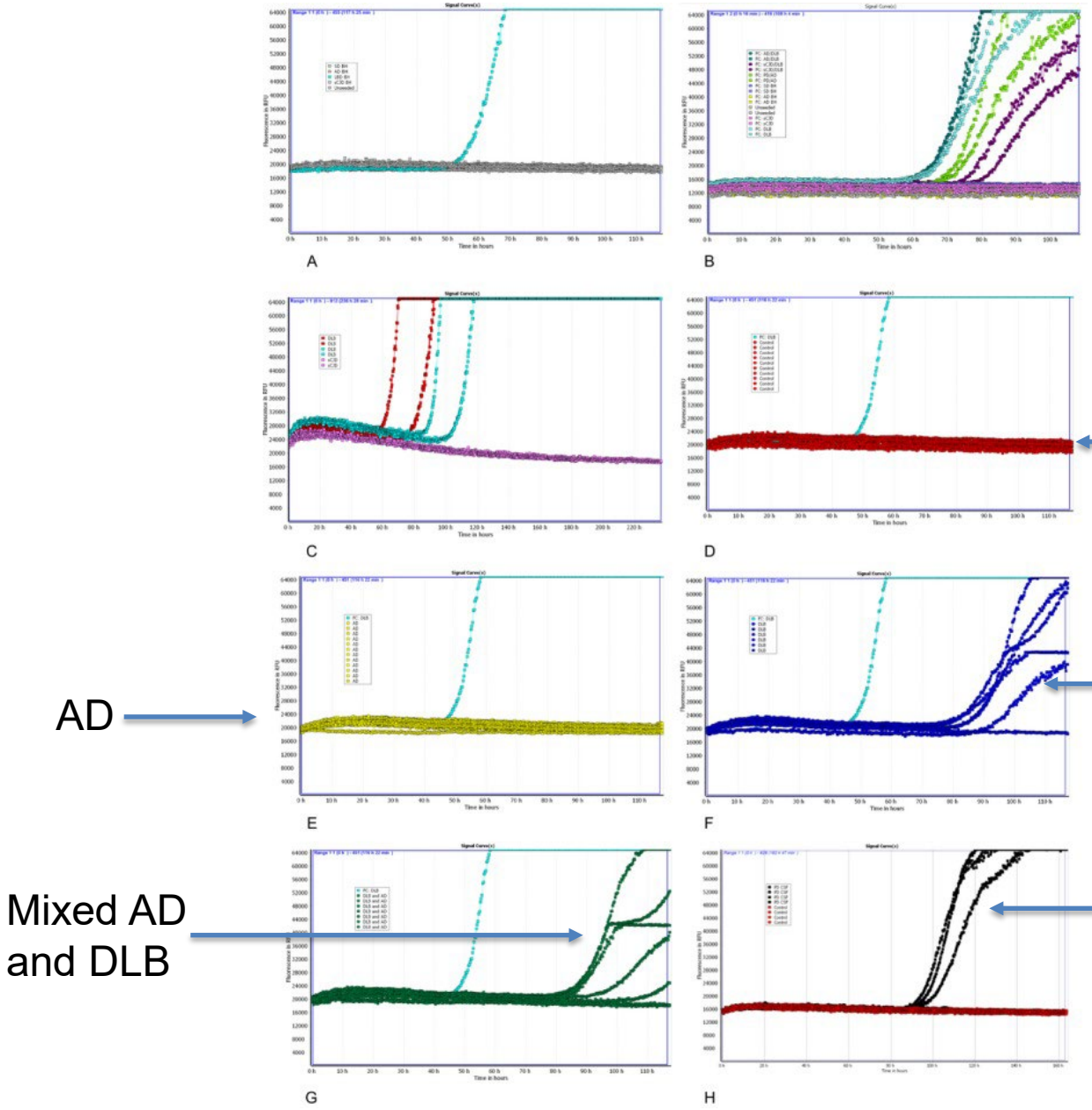




Oeckl *et al*.. A&D 2023

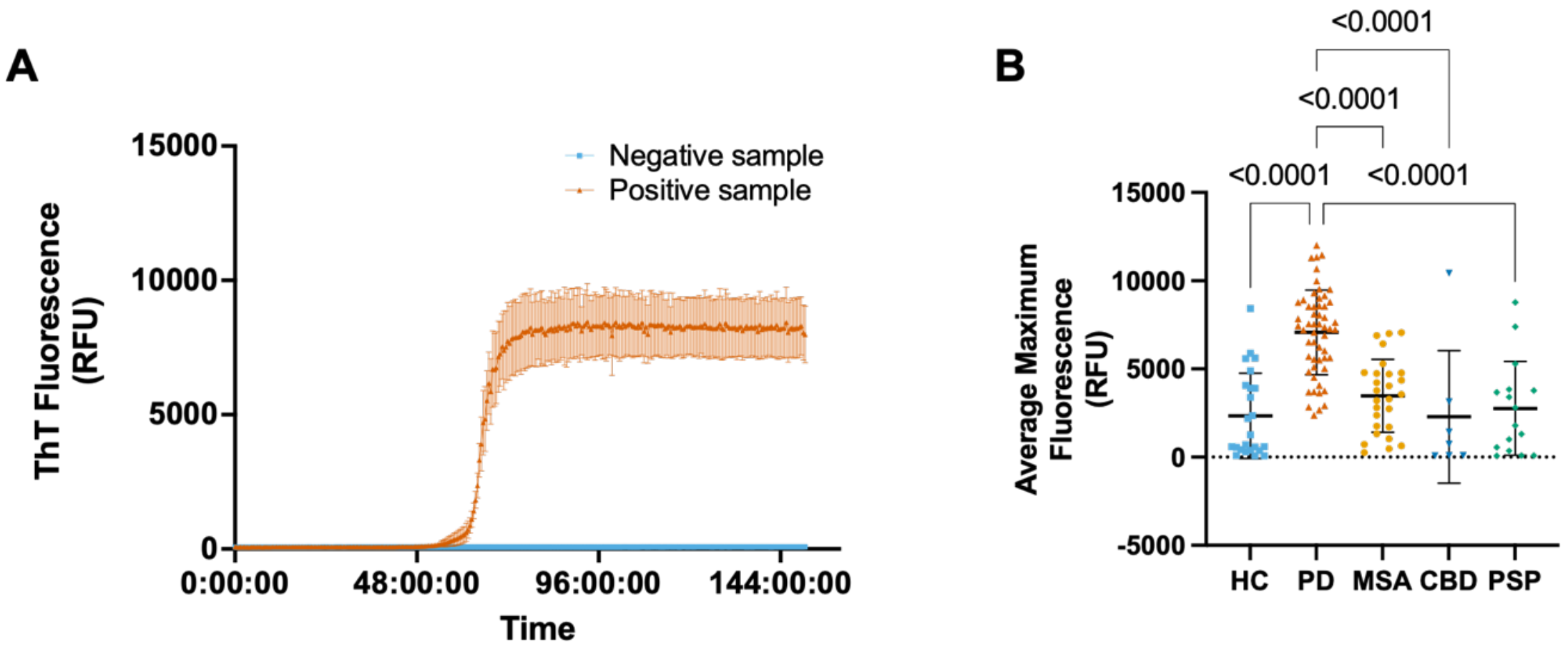
Synuclein pathology

CSF α-synuclein seeds across neurodegenerative diseases



Fairfoul et al., 2016

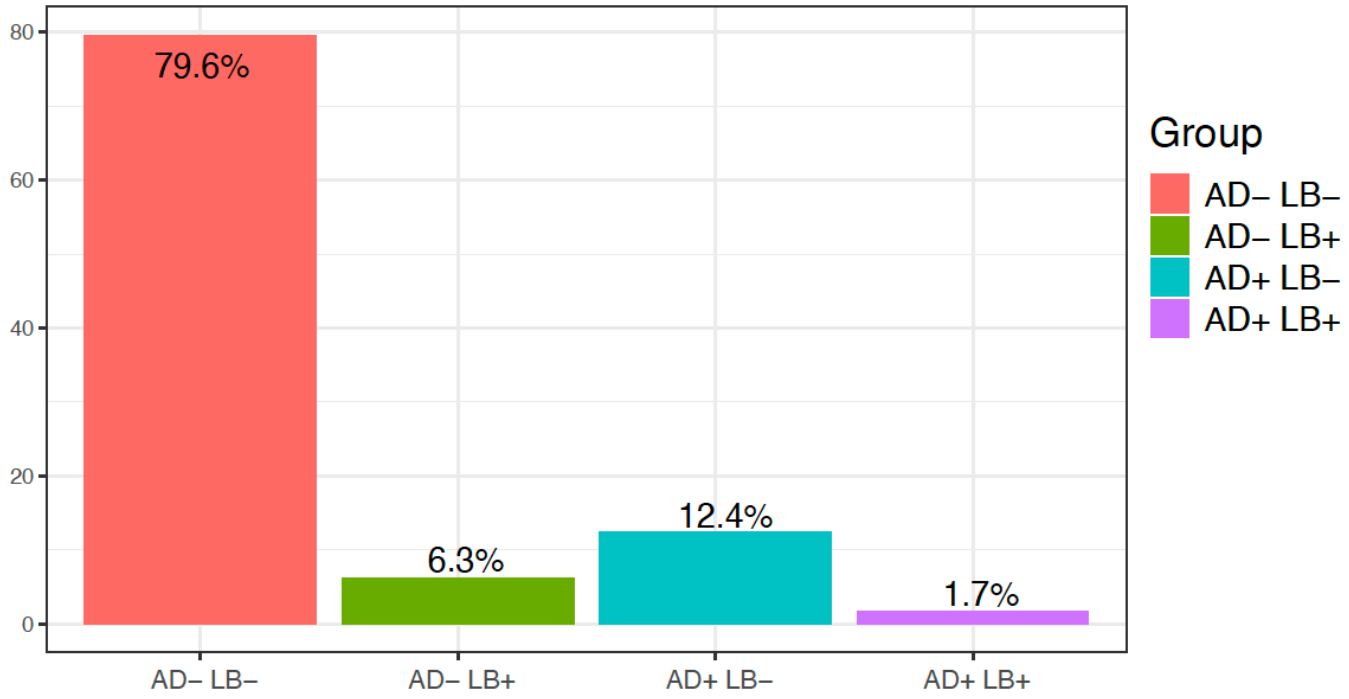
CSF α-synuclein seeds across neurodegenerative diseases



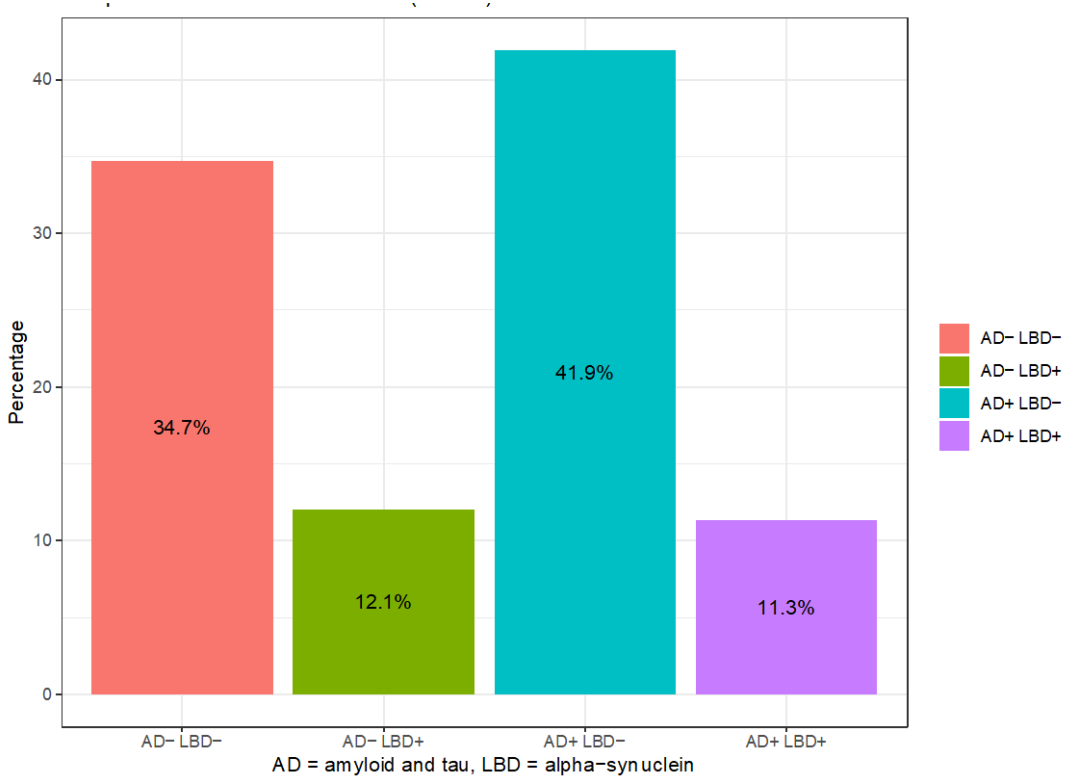
Gomes *et al.*, manuscript in preparation

Prevalence of Lewy body pathology in >1900 individuals in the Swedish BioFINDER studies

LB and AD pathology in CU

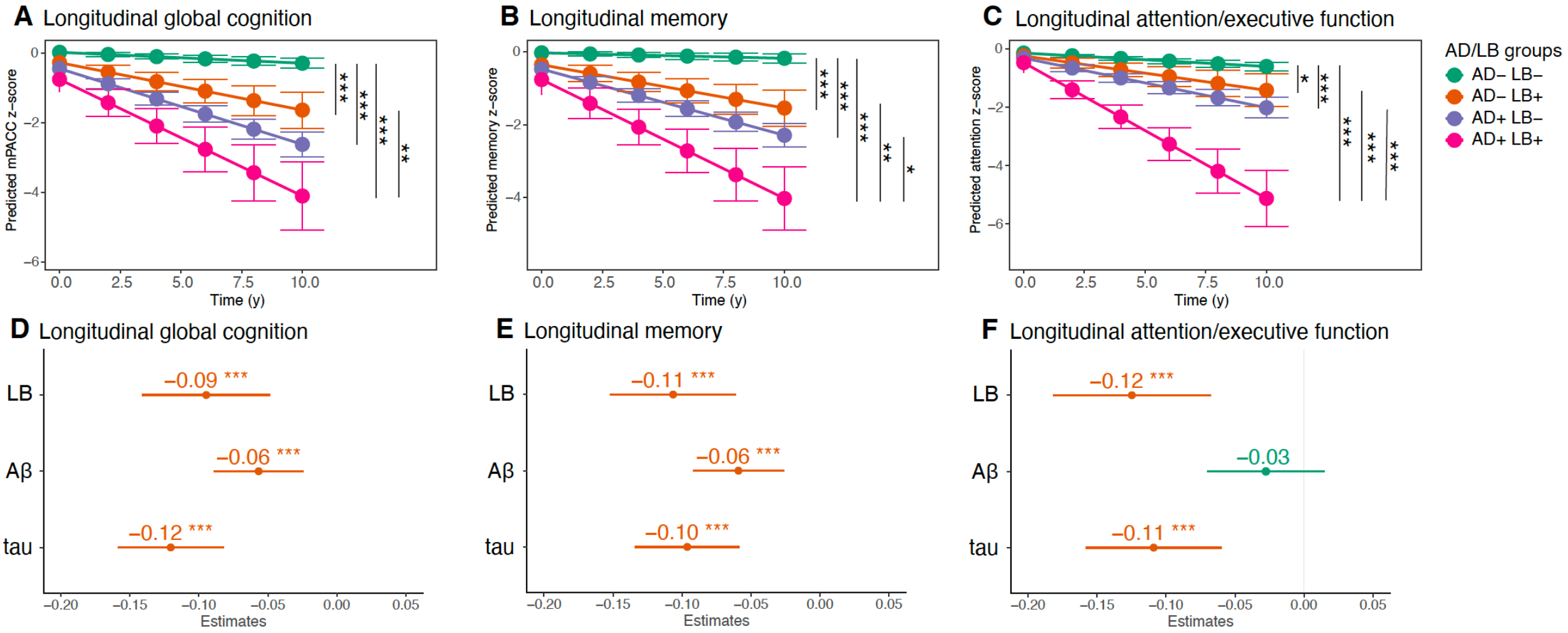


LB and AD pathology in CI



Hansson O *et al*., Nat Med 2023

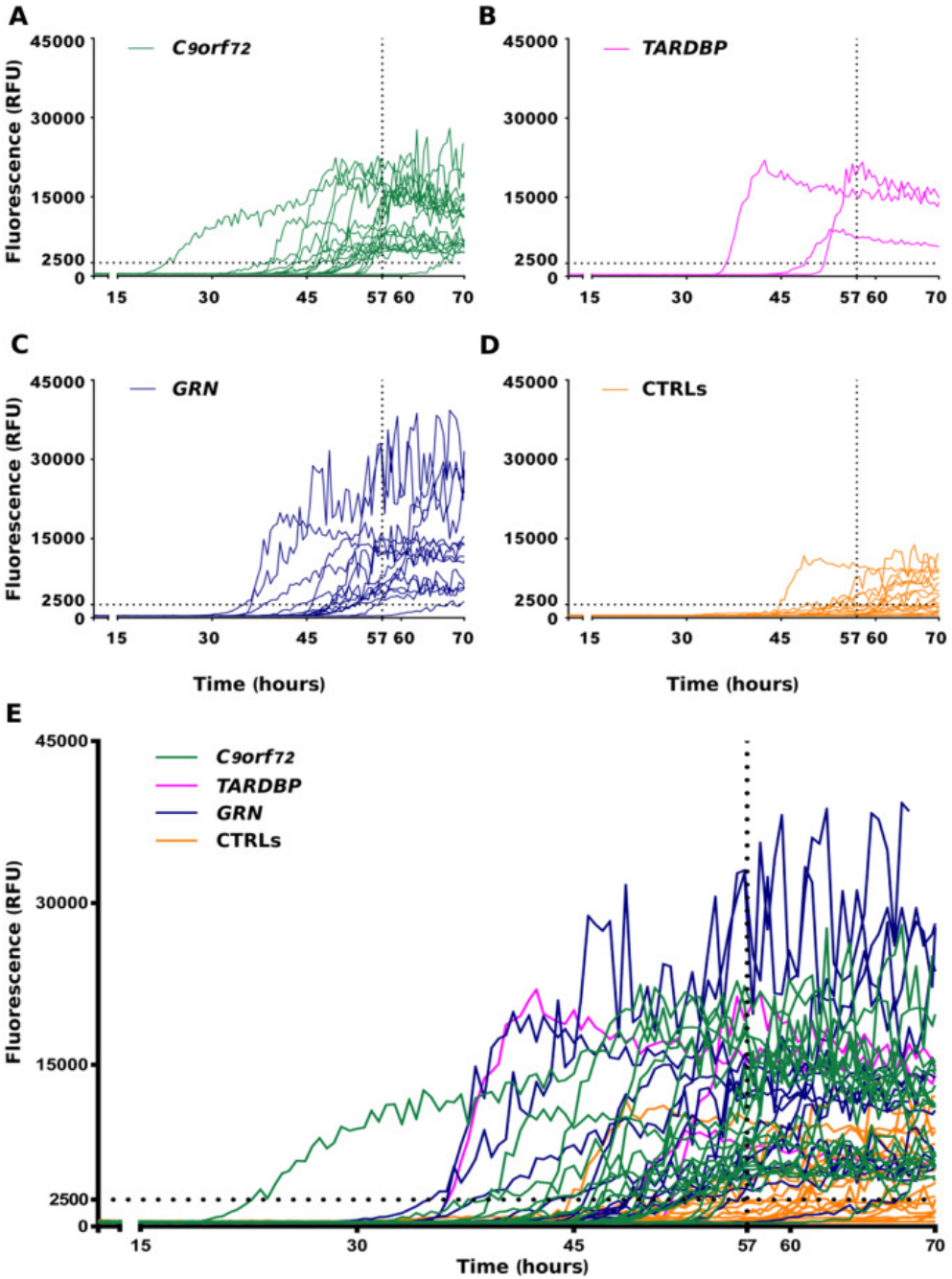
Longitudinal associations of LB pathology with cognitive change over time in clinically normal individuals



Hansson O *et al*., Nat Med 2023

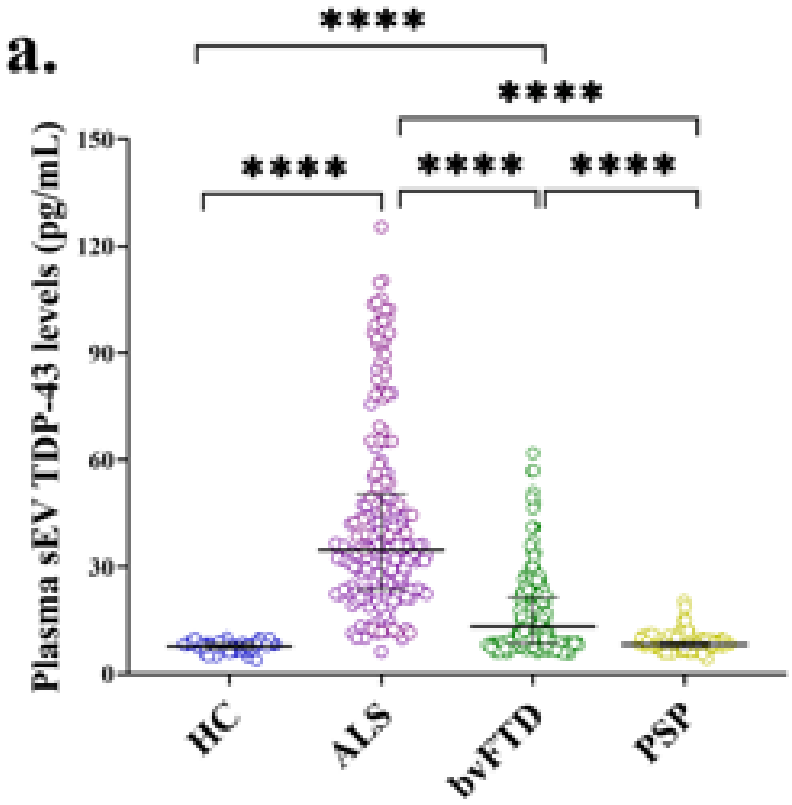
TDP-43 pathology

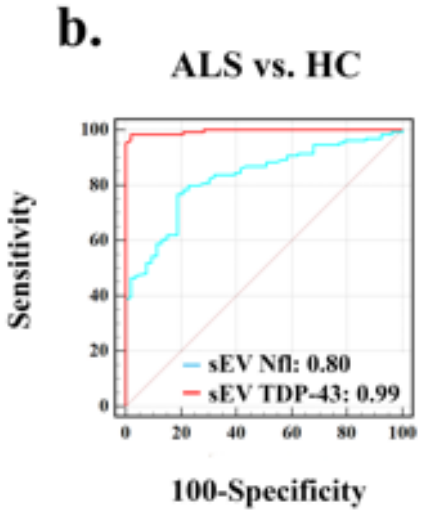
TDP-43 seeds in CSF?

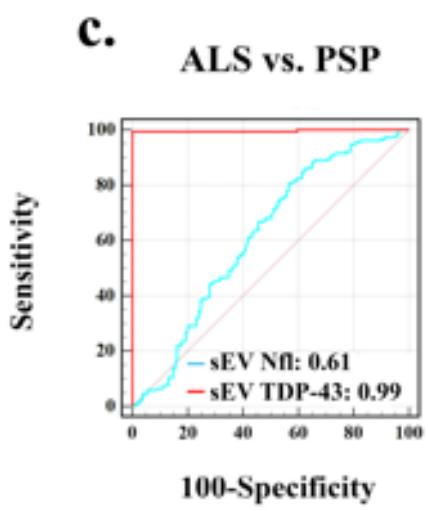


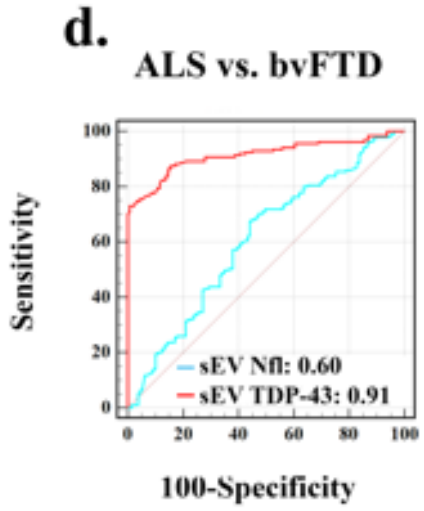
Scialò et al.
Brain Comm 2020

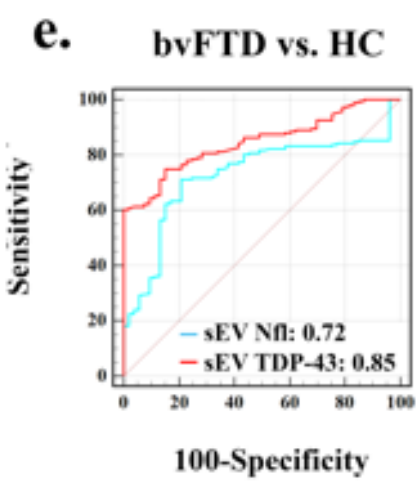
Plasma EV TDP-43 is increased in ALS and a subset of bvFTD cases

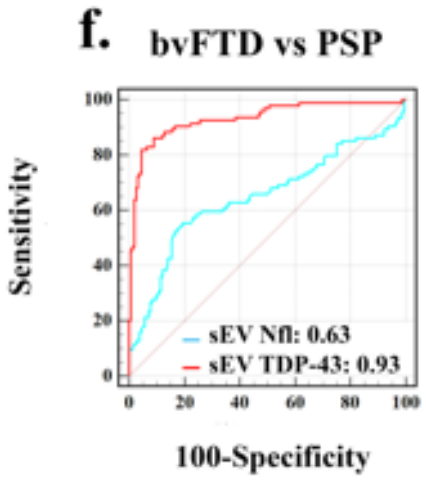








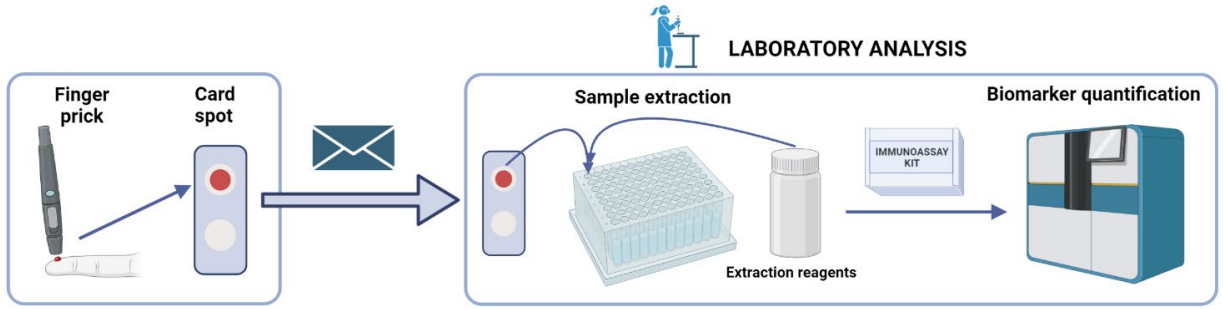


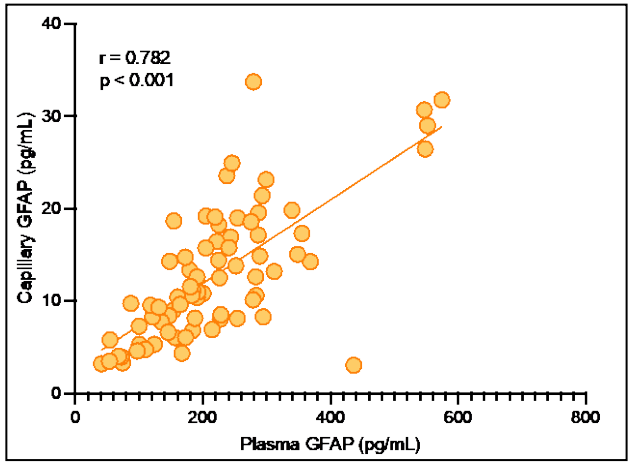


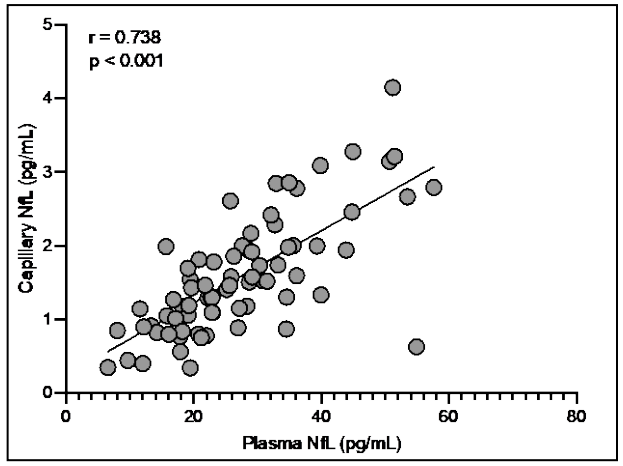
Chatterjee *et al*., Nature Med 2024

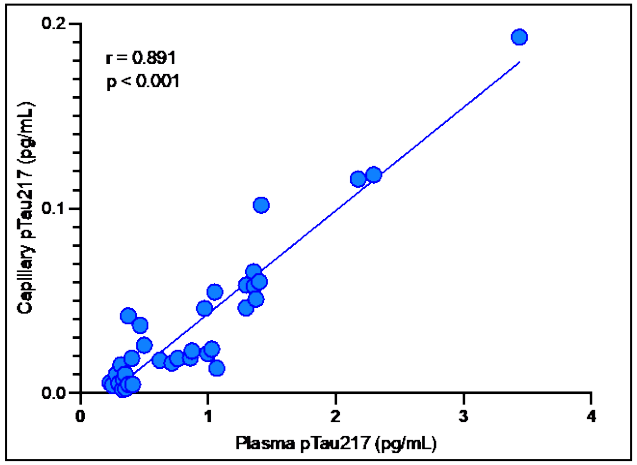
Simplified testing:
DROP-AD—detecting AD blood biomarkers using a finger-prick

- Current blood processing protocol require a strict procedures – useful in primary care?
- How do we monitor people overtime (including those on DMT) for personalised management?
- Detecting pre-clinical changes – if/when that it is required?









Conclusions

Blood biomarkers for AD are (almost) as good as CSF and PET

AD and neurodegeneration biomarkers can be measured in dried plasma spots

Promising new results for α-synuclein and TDP-43 pathologies

More work needed on non-AD tau pathology

More work needed on biomarkers for the neurovascular unit

More explorative cross-disease biomarker work needed

Thanks!

h.zetterberg@ucl.ac.uk; henrik.zetterberg@gu.se

mAbs for Alzheimer's Disease Biomarkers

2024-08-22

Emilia Galli, PhD, R&D Manager

Medix Biochemica

Medix Biochemica

How Can We Help?

- We provide high quality, high sensitivity mAbs produced in vitro by scalable procedures, ideal for assay development and manufacture
- Currently in catalog or in pipeline mAbs against Amyloid beta, p-Tau, t-Tau, CNS-Tau, NfL, GFAP
- We listen to our customers and are happy to collaborate in mAb development projects

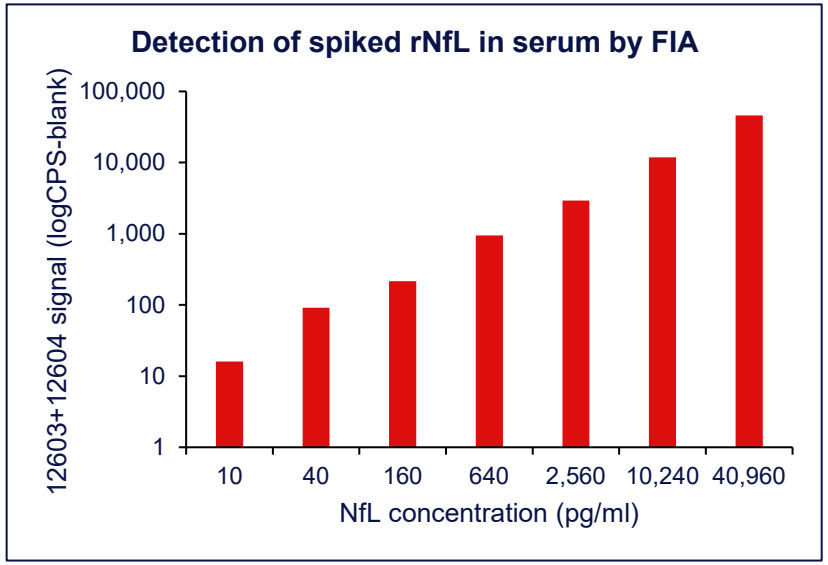
Medix Biochemica

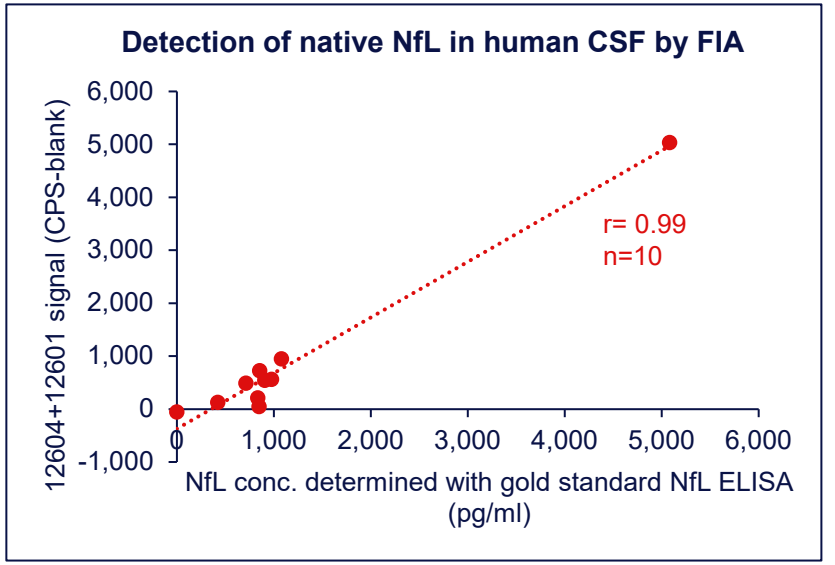
Neurodegeneration – NfL

- Three mAbs that work in pair for sensitive detection of NfL
- High sensitivity, reaches to detection of blood levels (10 pg/ml) with unoptimized FIA
- Kinetics measured by BLI (Octet) with free rNfL (LA666, Medix Biochemica) show strong binding
- Good correlation of CSF measurements to results with “gold standard” mAb pair
- No cross-reactivity with NfM, NfH, S100B and NSE

12601 – Anti-h NfL (cat. 100984)
12603 – Anti-h NfL (cat. 100985)
12604 – Anti-h NfL (cat. 100986)

N





mAb	K_a (1/Ms)	K_d (1/s)	KD (M)
12601 (NfL)	$2.3x10^5$	Does not dissociate	N/A
12603 (NfL)	$3.8x10^5$	Does not dissociate	N/A
12604 (NfL)	$1.7x10^5$	Does not dissociate	N/A

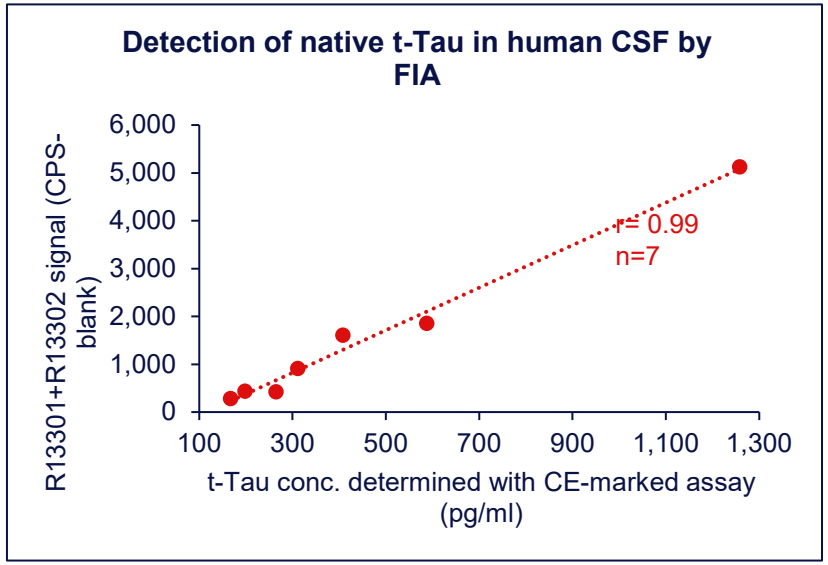
Medix Biochemica

Neurodegeneration – t-Tau

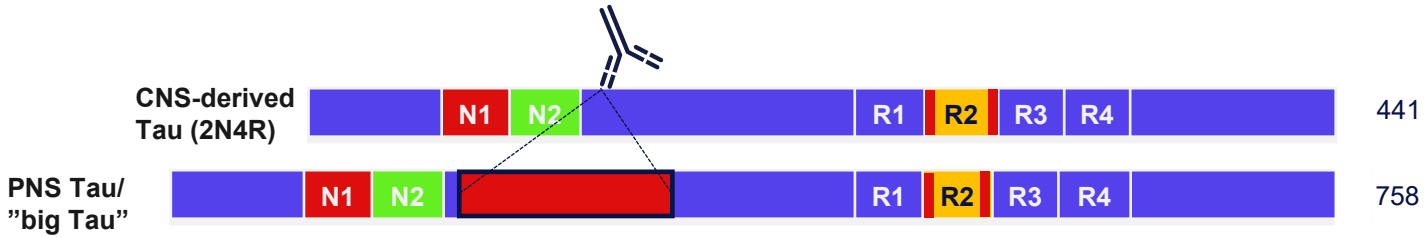
- Three mAbs that work in pair for t-Tau detection in CSF
- Good correlation of CSF measurements to CE-marked assay
- Kinetics measured by BLI (Octet) with sensor-bound antigen show strong binding to rTau
- The mAbs most likely detect also the peripheral ”big” Tau, and hence not recommended for blood-based detection of t-Tau
  - Tested with peptides spanning over the differing sequence between these variants
  - Project for generating mAbs for the specific detection of CNS-Tau is on-going

R13301 – Anti-h t-Tau (cat. 140038)
R13302 – Anti-h t-Tau (cat. 140039)
R13303 – Anti-h t-Tau (cat. 140040)

N



mAb	K_a (1/Ms)	K_d (1/s)	KD (M)
R13301 (Tau)	$5.1x10^5$	$3.1x10^{-5}$	$5.6x10^{-11}$
R13302 (Tau)	$8.9x10^5$	$2.9x10^{-5}$	$4.4x10^{-11}$
R13303 (Tau)	$2.1x10^6$	$8.0x10^{-5}$	$4.0x10^{-11}$

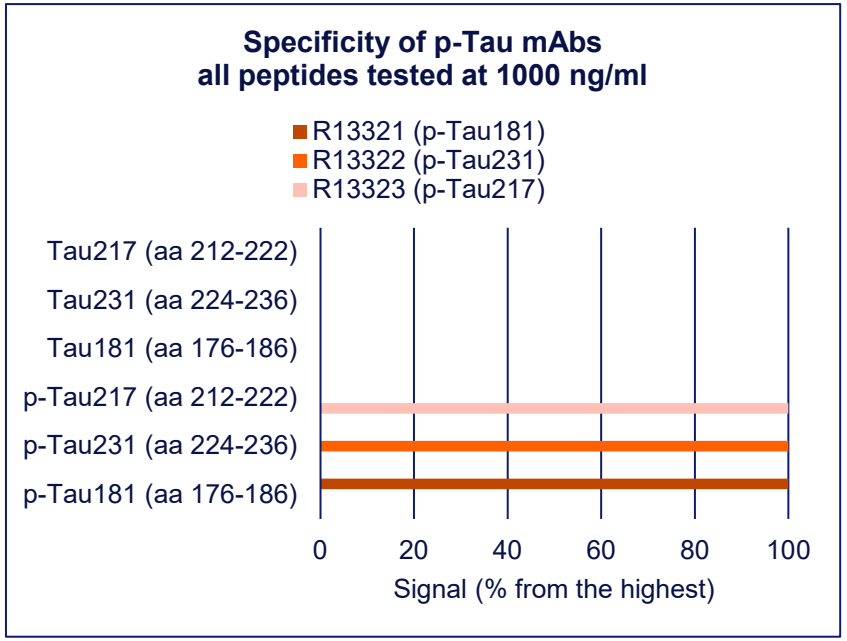


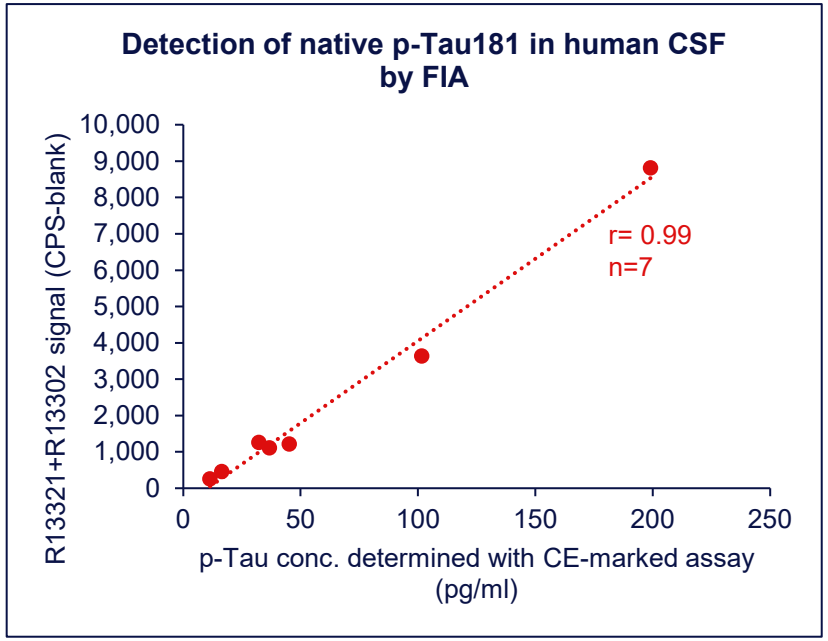
Medix Biochemica

Phosphorylated Tau

- Three phosphorylation-specific mAbs that work in pair with t-Tau mAbs R13301-R13303 for detectetion of p-Tau
- Highly specific to the annotated phosphorylation site as tested with phosphorylated peptides
- For anti-h p-Tau181 R13321, detection of native protein in CSF has been verified with correlation to known concentration measured with CE-marked assay
- Kinetics measured by BLI (Octet) with sensor-bound antigen show strong binding

R13321 – Anti-h p-Tau181 (cat. 140037)
R13322 – Anti-h p-Tau231 (cat. 140036)
R13323 – Anti-h p-Tau217 (cat. 140050)





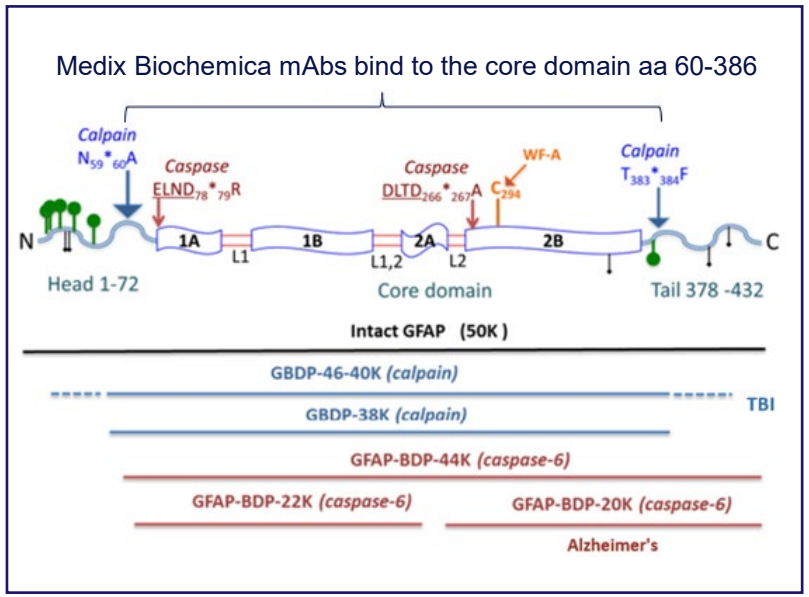
mAb	K_a (1/Ms)	K_d (1/s)	KD (M)
R13321 (p-Tau181)	$2.9x10^5$	Does not dissociate	N/A
R13322 (p-Tau231)	$2.9x10^6$	$7.7x10^{-4}$	$3.9x10^{-10}$
R13323 (p-Tau217)	$5.2x10^5$	$2.7x10^{-4}$	$6.3x10^{-10}$

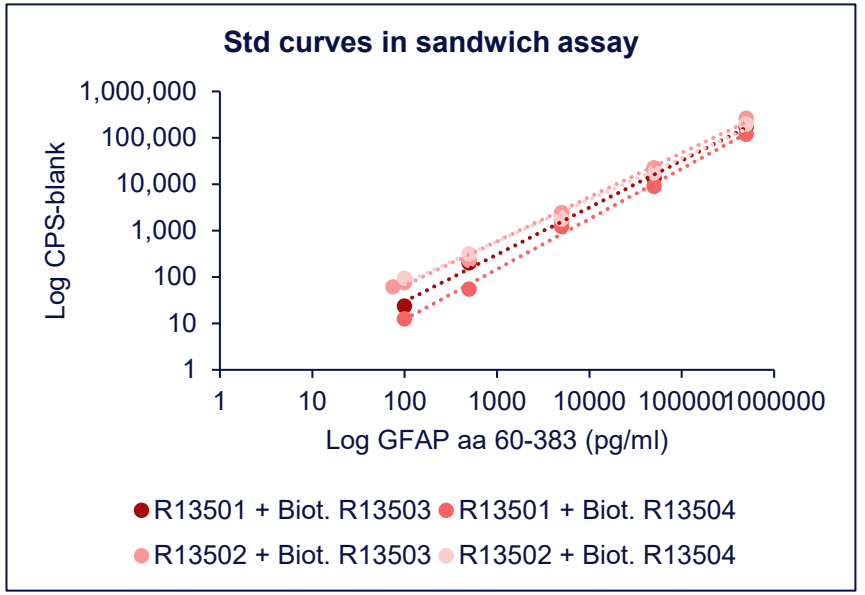
Medix Biochemica

Neuroinflammation – GFAP

- Four mAbs that work in pair for the detection of GFAP
- Detect the break-down product, epitope between amino acids 60-383 of human GFAP
  - Further internal studies indicate, that the epitope of at least R13501, R13503 and R13504 is in the region relevant to Alzheimer's disease diagnostics (2B)
- Sensitivity reaches to appr. 100 pg/ml with unoptimized FIA
- Kinetics measured by BLI (Octet) with free GFAP show strong binding
- No or minor cross-reactivity with homologs vimentin, desmin or pheripherin

R13501 – Anti-h GFAP (cat. 140046)
R13502 – Anti-h GFAP (cat. 140047)
R13503 – Anti-h GFAP (cat. 140048)
R13504 – Anti-h GFAP (cat. 140049)





mAb	K_a (1/Ms)	K_d (1/s)	KD (M)
R13501 (GFAP)	$2.0x10^5$	Does not dissociate	N/A
R13502 (GFAP)	$1.4x10^5$	Does not dissociate	N/A
R13503 (GFAP)	$1.3x10^5$	Does not dissociate	N/A
R13504 (GFAP)	$1.6x10^5$	Does not dissociate	N/A

Figure 1 adapted from Yang Z, Wang KK. Glial fibrillary acidic protein: from intermediate filament assembly and gliosis to neurobiomarker. Trends Neurosci. 2015 Jun;38(6):364-74. doi: 10.1016/j.tins.2015.04.003. Epub 2015 May 11. PMID: 25975510; PMCID: PMC4559283 and Yang Z et al., Characterization of Calpain and Caspase-6-Generated Glial Fibrillary Acidic Protein Breakdown Products Following Traumatic Brain Injury and Astroglial Cell Injury. Int J Mol Sci. 2022 Aug 11;23(16):8960. doi: 10.3390/ijms23168960. PMID: 36012232; PMCID: PMC9409281.

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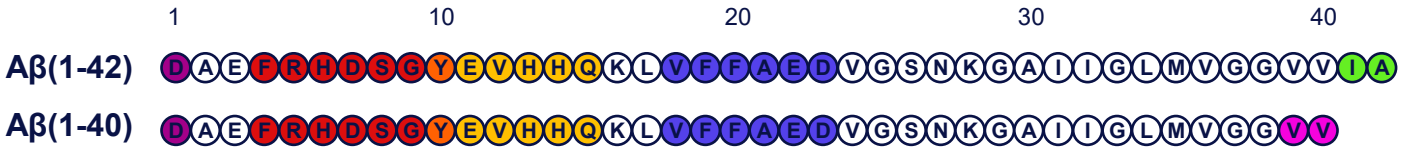
Amyloid beta – Aβ42 & 40

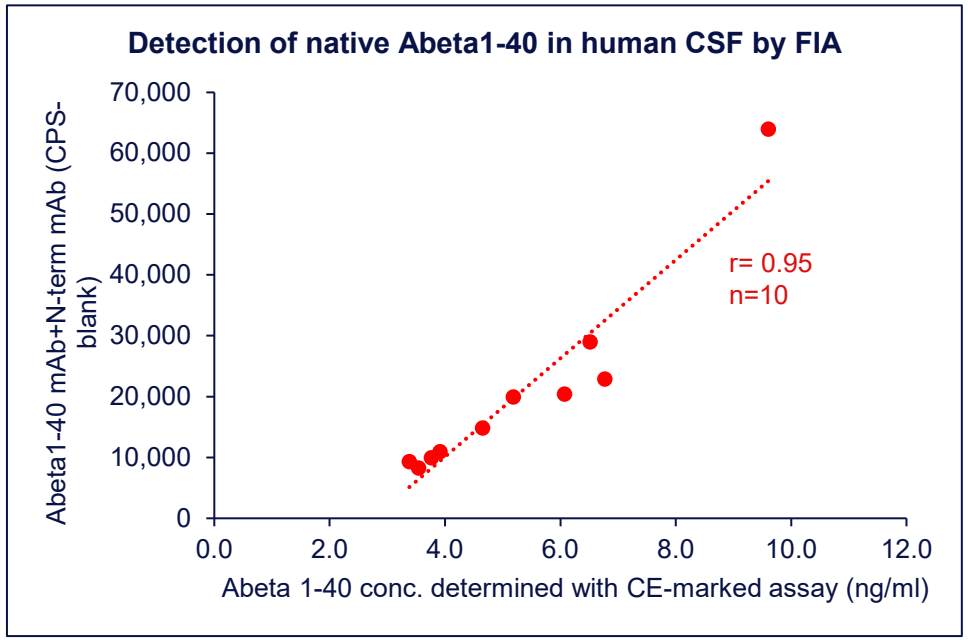
- mAbs for the specific detection of C-terminus in Aβ1-42 and Aβ1-40 and mAbs for N-terminus
- For Aβ1-40, detection of native protein in CSF has been verified with correlation to known concentration measured with CE-marked assay
- Detection of Aβ1-42 in CSF has been more challenging due to the analyte's sticky nature and lower levels
  - Effort for generating ever better 1-42 C-terminal candidates are ongoing

Upcoming!

Expected in Q4-2024

A





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