

Plasma Amyloid and Tau Biomarkers in Relation to Amyloid PET Imaging in an Indian Alzheimer's Disease Cohort



P4-06

Sandhya M¹, Nivedita Mitra², Gokul krishnan Kuppan³, Deeksha Muralidhar¹, Siva Kumar PT⁴, Venkatesh Murthy¹, Tejasvi Ravi⁵, Pardeep Kumar¹.
¹NIIR, NIMHANS, Bangalore, India, ²Clinical and Technology Innovation, Siemens Healthcare Private Limited, Bangalore, Karnataka, India, ³Neurochemistry, NIMHANS, Bangalore, India, ⁴Psychiatry, NIMHANS, Bangalore, India, ⁵Public Health, Charles University, Second faculty of Medicine, Prague, Czech Republic

For educational and scientific exchange only. Not for sales or promotional use

Background: Alzheimer's disease (AD) is a neurodegenerative disorder characterized by progressive decline in memory, cognition, and daily functioning. Access to cerebrospinal fluid and amyloid PET imaging remain limited in many low- and middle-income settings. The AT(N) framework defines AD biologically using three core biomarker categories: amyloid- β (A), tau (T), and neurodegeneration (N). Blood-based biomarkers such as plasma p217, A β 42/40 ratio, GFAP and sNfL are being actively investigated for their association with neurodegenerative disease-related biological processes. Existing evidence from non-Indian cohorts suggests these biomarkers may show associations with amyloid PET measures and cognitive parameters, yet data from Indian populations remain sparse.

INTRODUCTION

The goal of this study is to evaluate the association between plasma amyloid- and tau-related biomarkers and amyloid PET-derived measures in an Indian research cohort with cognitive impairment phenotypes, consistent with Alzheimer-type and frontotemporal-type syndromes based on standard clinical assessment independent of the research assays .

The neuro blood biomarkers measured include, serum neurofilament light (sNfL), Glial fibrillary acidic protein (GFAP), phosphorylated Tau217 (pTau217), and beta-amyloid 42/40 ratio (A β 42/40).

METHODS

Cohort: 24 cognitively unimpaired (CU), average age 52.3 $\pm$ 4.2 yrs, and 34 cognitively impaired (CI), average age 69.1 $\pm$ 7.7 yrs, individuals (based on clinical assessment & ACE scores).

Amyloid PET: Amyloid PET acquired with Research use only (RUO*) radiopharmaceutical NAV4694 (flutafuranol F-18) on CI cohort only with the Siemens Healthineers Biograph mMR system. Centiloid values are used to define study grouping for research analysis.

Cognitive assessment: Done using Addenbrooke's Cognitive Examination (ACE-III) as part of the research protocol.

Blood Biomarkers: RUO* assays sNfL and pTau217 were measured on Siemens Healthineers Atellica IM Analyzer. GFAP, NFL and A β 42/40 were measured on the Quanterix Simoa HDX analyzer.

Statistical analysis: Spearman Rank correlation is used. ROC analysis was used for exploratory evaluation of discrimination between study-defined groups based on amyloid PET status.

RESULTS

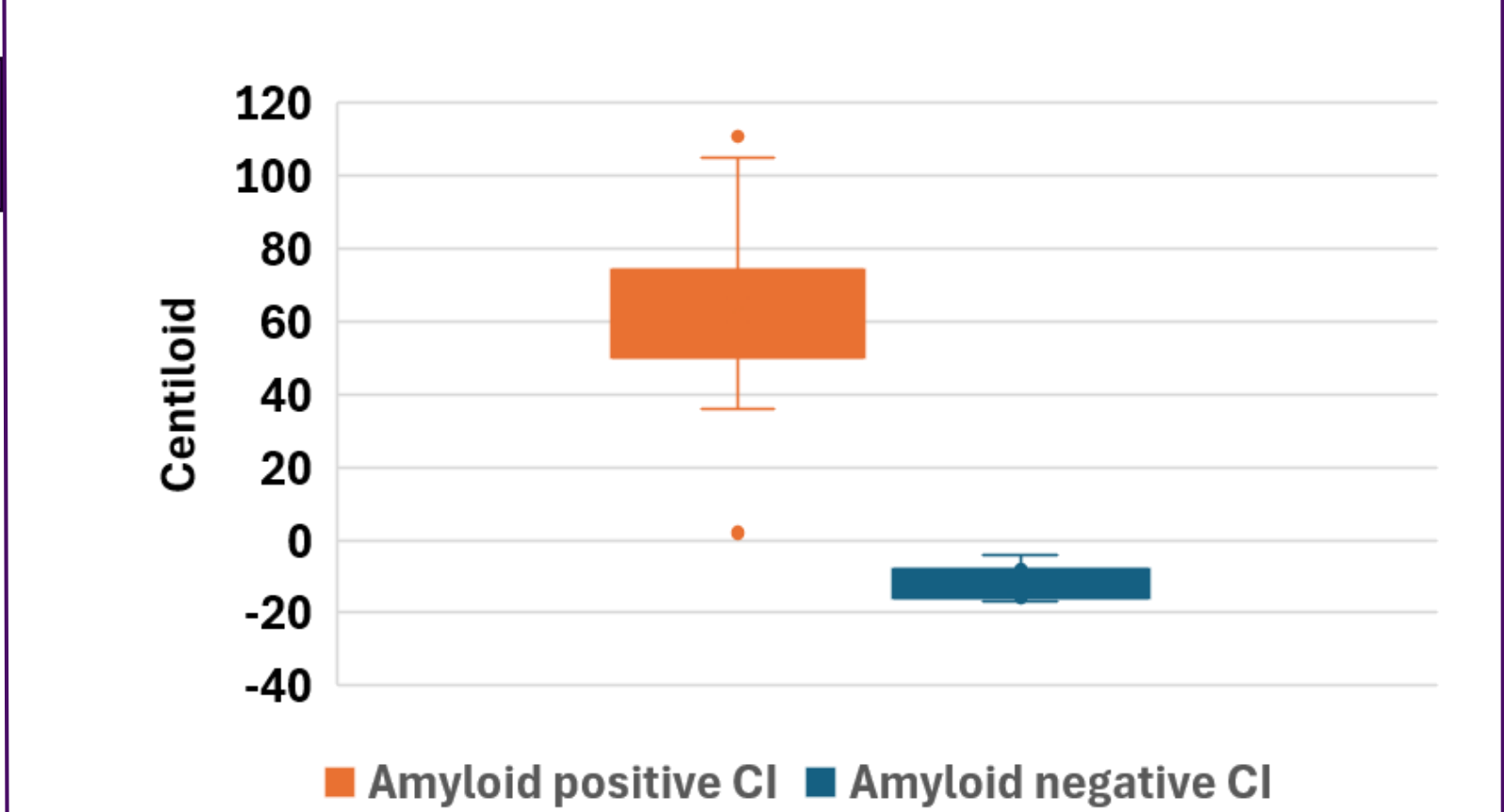


Fig 1. Box plot Centiloid values were used to classify CI patients into amyloid PET positive (amyloid positive CI) and negative (amyloid negative CI).

*For Research Use Only. Not for use in diagnostic procedures.

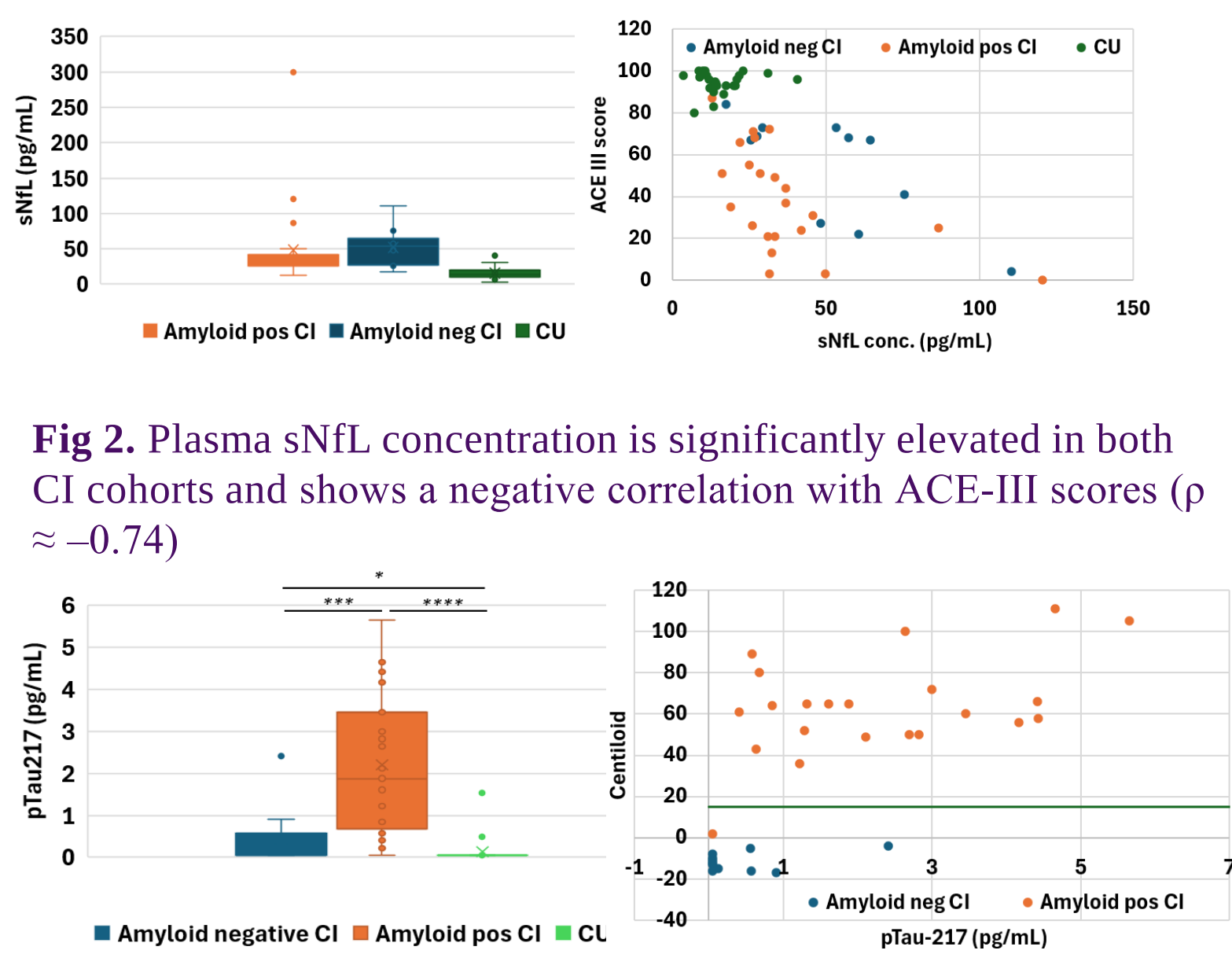


Fig 2. Plasma sNfL concentration is significantly elevated in both CI cohorts and shows a negative correlation with ACE-III scores ($\rho \approx -0.74$)

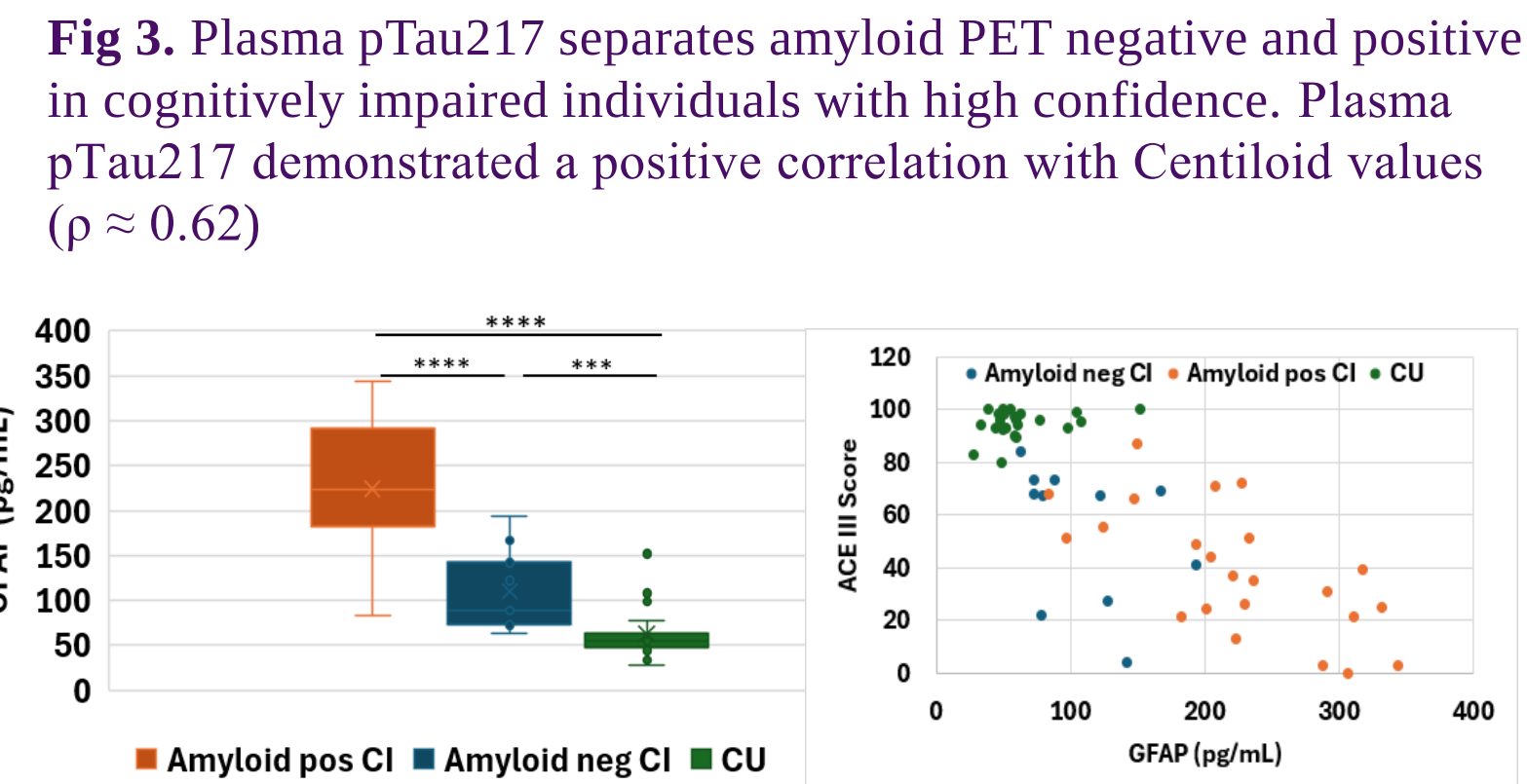


Fig 4. GFAP plasma concentration is positively correlated with centiloid ($\rho = 0.60$) and negatively correlated with ACEIII score ($\rho = -0.76$).

All analyses presented here represent exploratory statistical evaluations and are not intended to establish clinical performance

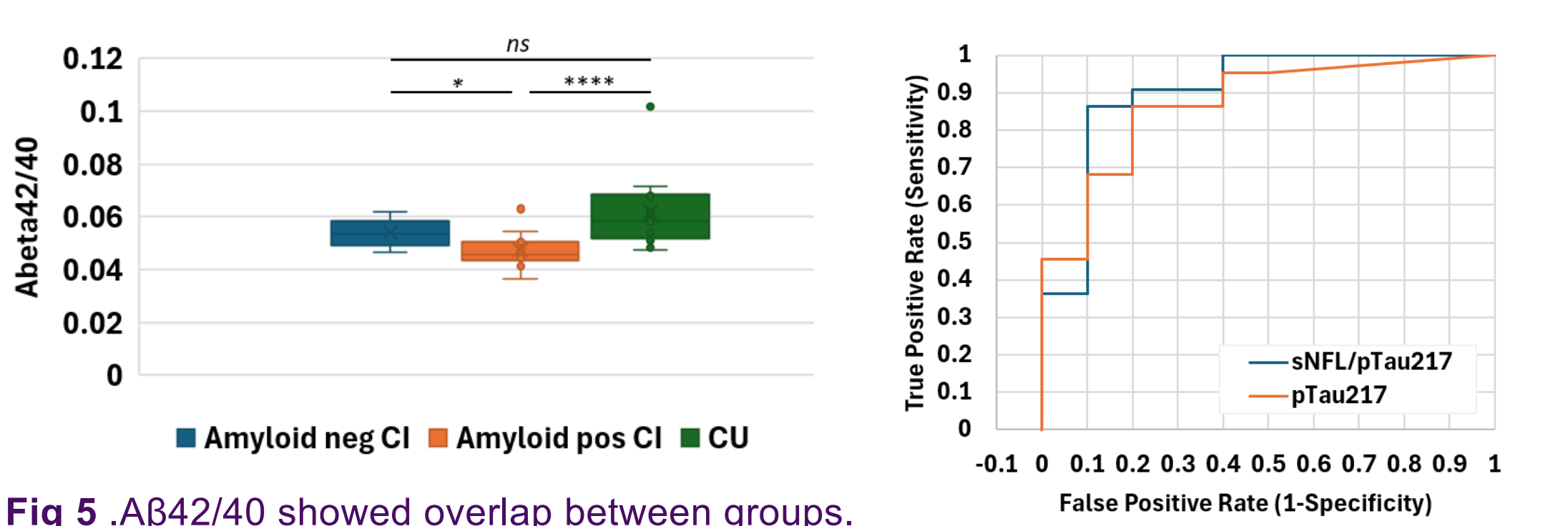


Fig 5 . A β 42/40 showed overlap between groups.

Fig 6: Exploratory Discrimination Analysis showed an AUROC of 0.87 (pTau217, threshold 0.58, Sens 0.86 & Spec 0.80) and 0.905 (sNfL/pTau217 ratio, threshold 72.8, Sens 0.86 & Spec 0.90) when classifying amyloid PET positive and negative individuals.

CONCLUSIONS

In this Indian research cohort, plasma pTau217, sNfL, GFAP, and A β 42/40 demonstrated associations with amyloid PET-derived measures under research conditions. These findings support continued research into blood-based biomarkers and their relationship to neurodegenerative disease processes. Further studies are required to better understand their behavior across populations and over time.

References: Benussi et. al., Alzheimer's Dement. 2025;21:e14482; Jack Jr et. al., Alzheimer's Dement. 2024;20:5143–5169; Ravindranath et. Al., Nat Rev Neurol, 2021; 17, 747–75.

Acknowledgements: Sarada Subramanian, Professor and Head, Neurochemistry Department, NIMHANS for Quanterix Support; Ruchi Chauhan and Suparna Ghanvatkar for data analysis and Sudipa Bhattacharya for Atellica support (Siemens Healthcare Private Limited)

Contact: Professor Dr. Sandhya M
drsandym@gmail.com;
sandhya.e00205@nimhans.net



Scan me!