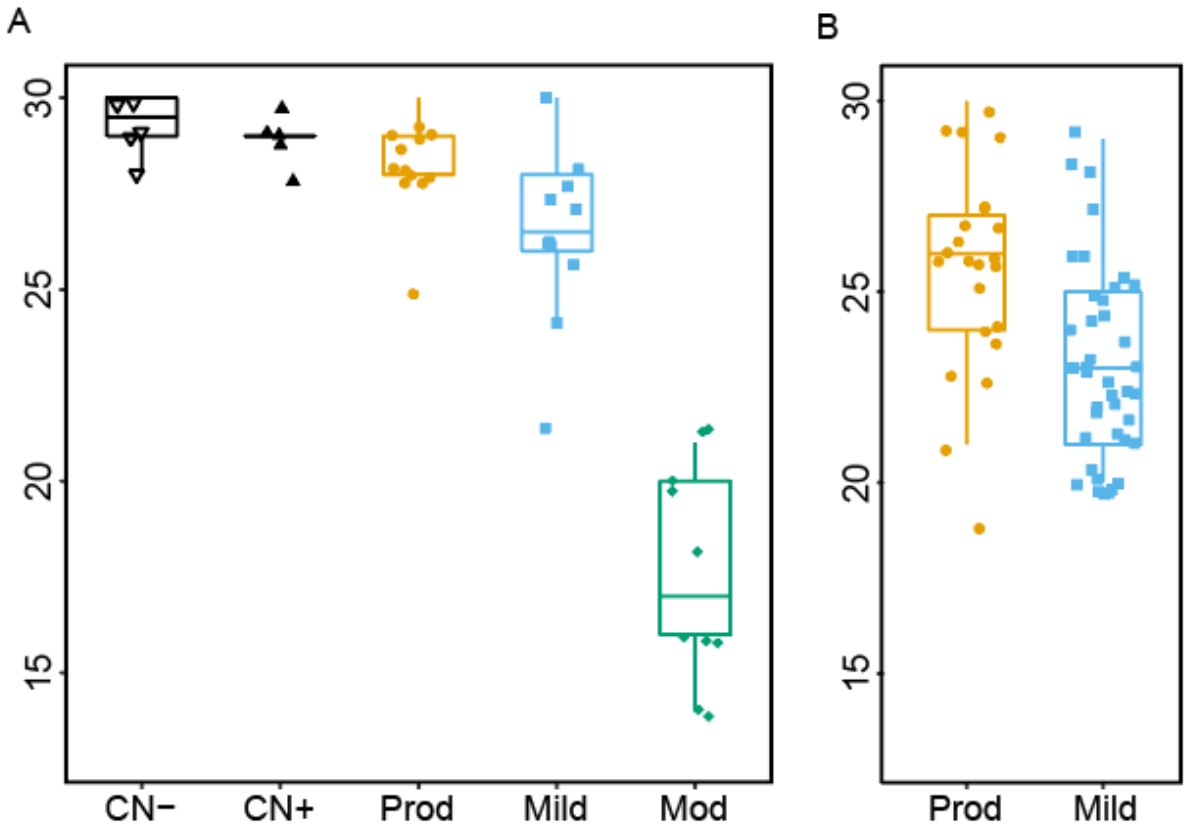
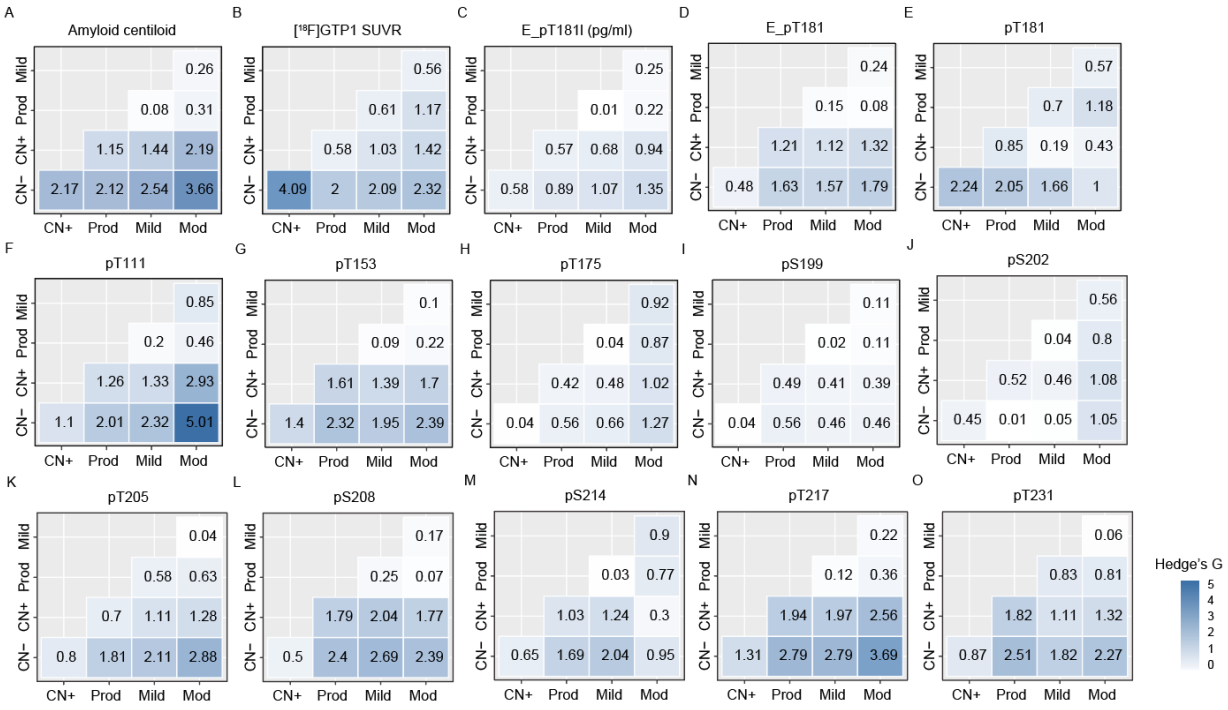
Supplementary Material

Site-Specific Cerebrospinal Fluid Tau Hyperphosphorylation in Response to Alzheimer's Disease Brain Pathology: Not All Tau Phospho-Sites Are Hyperphosphorylated

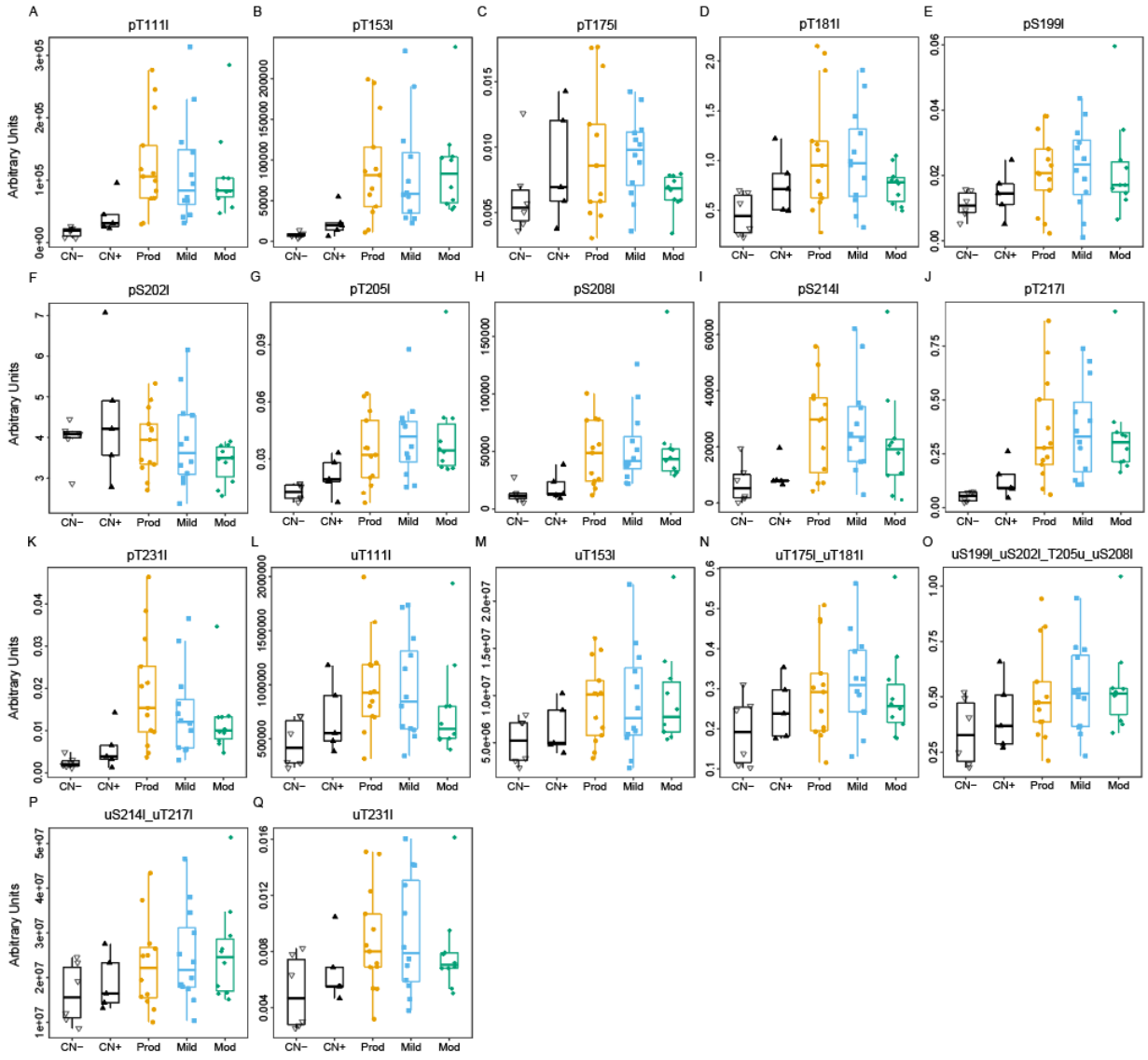
Supplementary Figure 1. Mini-Mental State Examination in (A) Cohort A and in (B) Cohort B. CN-, cognitively normal, amyloid-low control; CN+, amyloid-high control; prod, prodromal; mod moderate.



Supplementary Figure 2. Hedges' g ratios between the cohorts in Cohort A comparing brain amyloid deposition measured by (A) Amyloid centiloid, (B) [^{18}F]GTP1 uptake measuring brain tau aggregates, (C, D) p-tau measures by Elecsys Immunoassay on p-tau181, (E) tau phosphorylation occupancy on 181, and on the (F-O) 10 others phosphorylated residues monitored by MS. CN-, cognitively normal, amyloid-low control; CN+, amyloid-high control; prod, prodromal; mod moderate.



Supplementary Figure 3. Levels of phosphorylated (pX###l) and unphosphorylated peptides (uX###l) in Cohort A. CN-, cognitively normal, amyloid-low control; CN+, amyloid-high control; prod, prodromal; mod moderate. Y axis units are not comparable between the various peptides.



Supplementary Figure 4. Comparison of Elecsys immunoassay and IP-LCMS/MS assay coverage CSF tau phosphorylation on T181 (A). Association between phosphorylated to unphosphorylated peptide ratio (p-tau/u-tau) measured by MS and phosphorylated to total-tau ratio (p-tau/t-tau) by immunoassay in Cohort A and Cohort B, respectively (B, C).