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Supplementary Data

CRMP2 Hyperphosphorylation is Characteristic of Alzheimer's Disease and not a Feature Common to Other Neurodegenerative Diseases

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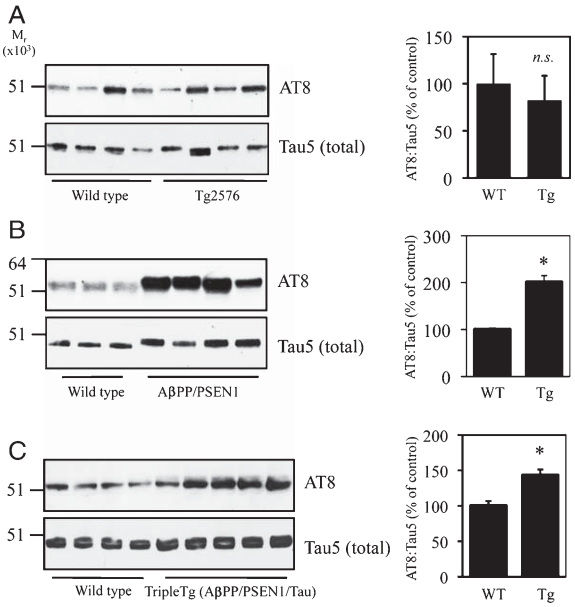
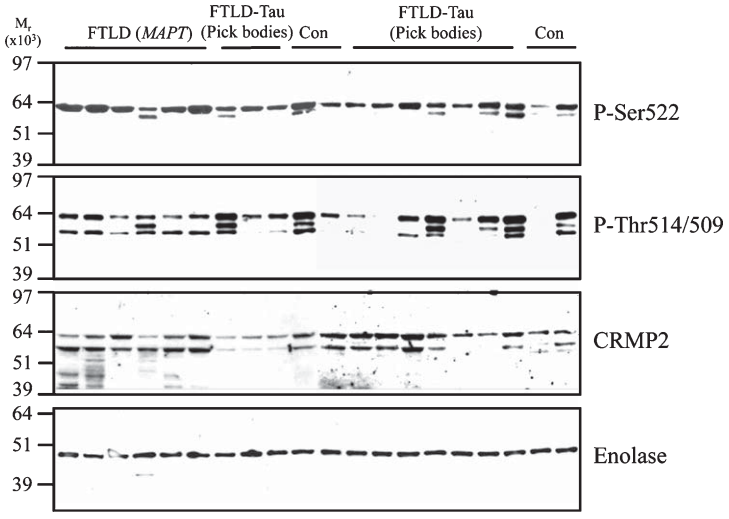


Supplementary Figure 1. *Tau phosphorylation in some mouse models of AD.* Cortical tissue from Tg2576 mice and age-matched controls were homogenized in 1% Triton X-100 lysis buffer, subjected to Western blot analysis and membranes probed with antibodies that recognize phosphorylated tau (AT8; upper panel) or total tau (tau-5; lower panel). The ratio of AT8:tau-5 in control and Tg2576 mouse cortex is presented as a graph. B) Same as A), except AβPP/PSEN-1 versus control mouse cortex. C) Same as A), except AβPP/PSEN1/tau triple-transgenic versus control mouse cortex. (average ± standard deviation; *n.s* = not significant, * = $p < 0.05$ (Students t-test).



Supplementary Figure 2. *CRMP2 phosphorylation is decreased in FLTD-tau diseases.* Frontal cortex tissue lysates from 6 cases of human FTLD-tau associated with exon 10 + 16 mutation in *MAPT* and 18 with Pick body pathology, as well as 8 age-matched controls, were subjected to Western blot analysis. Membranes were probed with antibodies that specifically recognize CRMP2 when phosphorylated at Ser522 (first panel), Thr514/509 (second panel), total CRMP2 (third panel) and NSE as a loading control (fourth panel). Representative blots of control, FLTD (MAPT) and FLTD-tau (Pick's Bodies) samples are shown.