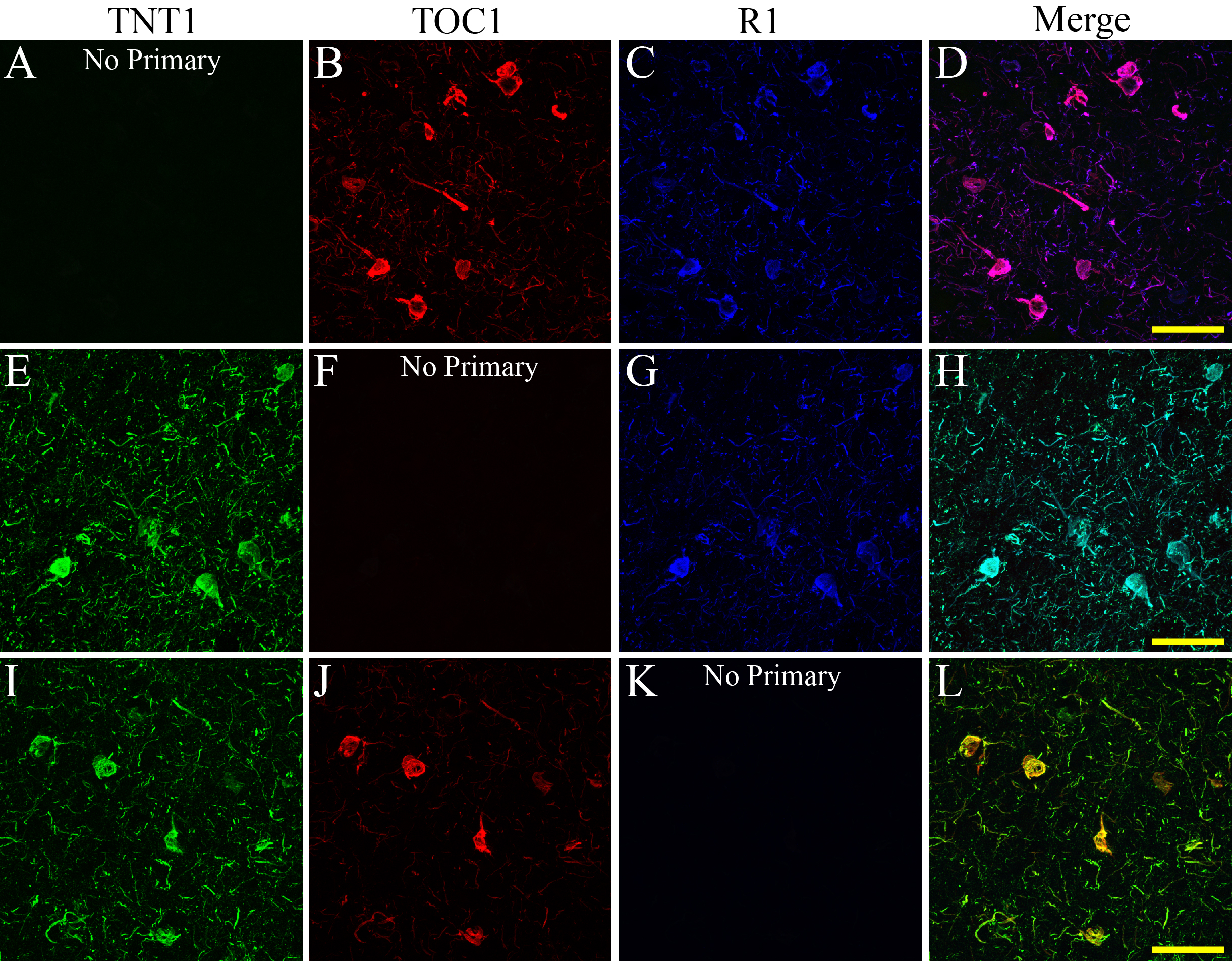
**Supplemental Materials**

**Supplementary Table 1.** Detailed information on human subjects used for fresh frozen and fixed tissue samples.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Diagnosis** | **Age (yrs)** | **Gender** | **PMI (h)** | **Tau Staging** | **Region** | **Type** |
| Control | 80 | M | 5 | Braak II | Ft Ctx | Fresh Frozen |
| Control | 75 | F | 17 | Braak II |
| Control | 88 | M | 27 | Braak II |
| Control | 76 | F | 13 | Braak II |
| Severe AD | 69 | M | 8 | Braak VI | Ft Ctx |
| Severe AD | 90 | M | 19 | Braak V |
| Severe AD | 93 | M | 10 | Braak V |
| Severe AD | 83 | M | 12 | Braak VI |
| PiD | 67 | M | 21 | --- | Ft Ctx |
| PiD | 55 | M | 9 | --- |
| PiD | 78 | F | 25 | --- |
| PiD | 82 | F | 9 | --- |
| CBD | 75 | F | 11 | --- | Ft Ctx |
| CBD | 81 | F | 17 | --- |
| CBD | 54 | M | 24 | --- |
| CBD | 62 | F | 5 | --- |
| Control | 83 | F | unk | Braak I | HP | Fixed Sections |
| Control | 77 | M | 17 | Braak II |
| Control | 82 | F | 5 | Braak II |
| Severe AD | 68 | F | 7 | Braak VI | HP |
| Severe AD | 71 | M | 6 | Braak VI |
| Severe AD | 91 | M | 4 | Braak VI |
| PiD | 74 | F | 4 | --- | Ft Ctx |
| PiD | 66 | M | 16 | --- |
| PiD | 75 | M | 0 | --- |
| PiD | 55 | M | 4 | --- |
| CBD | 57 | M | 2 | --- | Ft Ctx |
| CBD | 60 | F | 0 | --- |
| CBD | 64 | F | 13 | --- |
| CBD | 61 | M | 0 | --- |

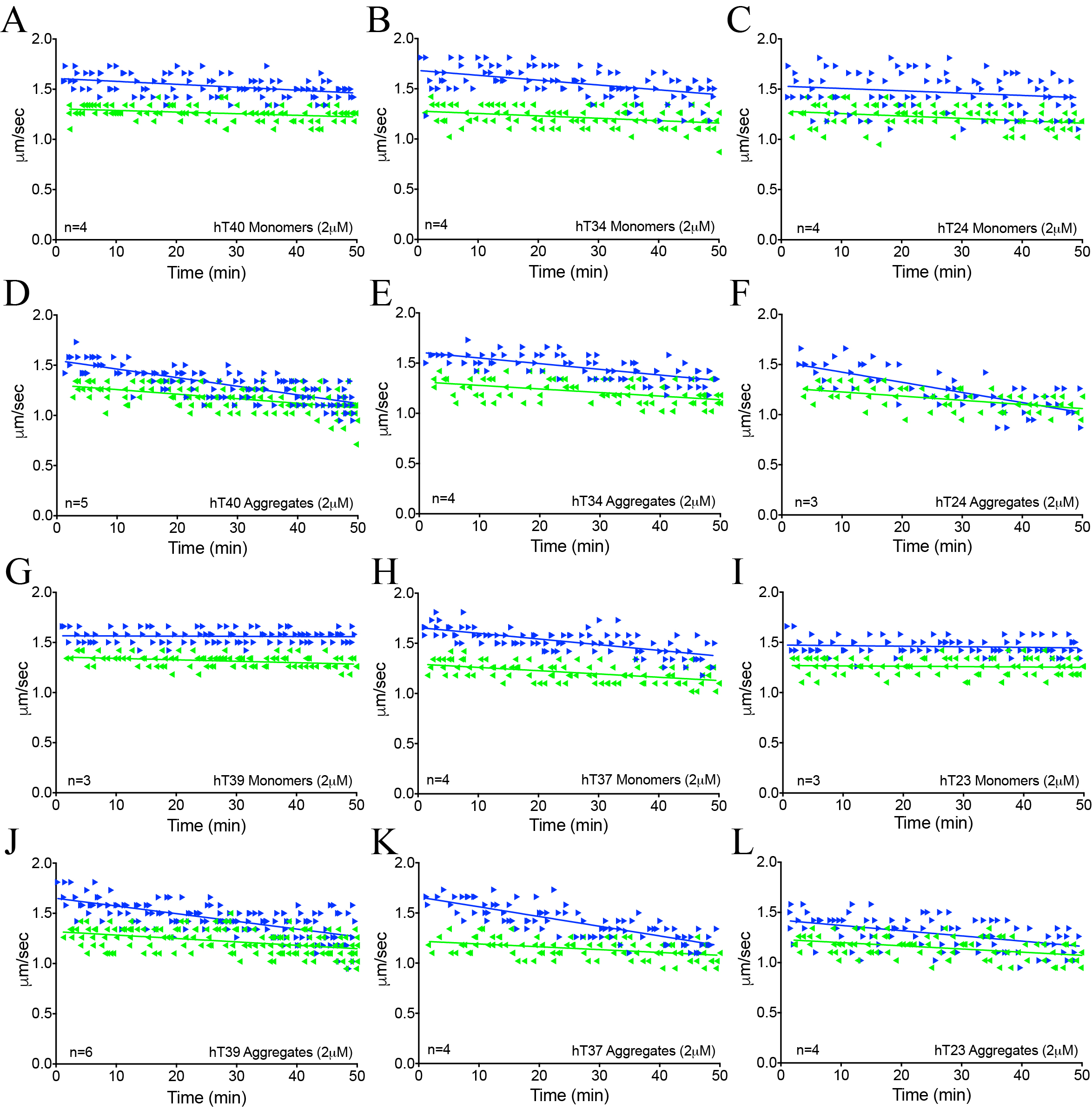
AD-Alzheimer’s disease; PiD-Pick’s disease; CBD-corticobasal degeneration; M-male, F-female; PMI- postmortem interval; unk-unknown; PMI-post-mortem interval Ft Ctx-frontal cortex; HP-hippocampus. All tissue was obtained from the Brain Bank of the Cognitive Neurology and Alzheimer’s Disease Center at Northwestern University.

**Supplementary Figure 1.**

****

**Supplementary Fig. 1.** Control immunofluorescence images confirm staining specificity. (A-L) Tissue sections were processed following the triple-label immunofluorescence protocol for TNT1, TOC1 and R1 tau staining with the exception that the (A-D) TNT1 (green), (E-H) TOC1 (red), or (I-L) R1 (blue) primary antibody was omitted. In all conditions, the lack of fluorescent signal for the omitted primary antibody channel confirmed that the signals for each primary-secondary antibody combination were specific and did not produce erroneous signal. All scale bars are 50 μm.

**Supplementary Figure 2.**

****

**Supplementary Fig. 2.** Plots of transport velocity over time in squid axoplasms exposed to each tau isoform monomer and aggregate samples. (A-L) Vesicle motility assays in isolated squid axoplasm were run and individual velocity (μm/sec) measurements of anterograde (blue arrowheads) and retrograde (green arrowheads) are plotted as a function of time (min). (A-C) Perfusion of hT40 (A), hT34 (B) and hT24 (C) monomers did not alter anterograde or retrograde FAT. (D-F) Perfusion of hT40 (D), hT34 (E) and hT24 (F) aggregates selectively inhibited anterograde but not retrograde FAT. (G-I) Perfusion of hT39 (G), hT37 (H) and hT23 (I) monomers did not alter anterograde or retrograde FAT. (J-L) Perfusion of hT39 (J), hT34 (K) and hT24 (L) aggregates selectively inhibited anterograde but did not appear to dramatically affect retrograde FAT. It is noteworthy that a small but significant reduction (~12%) in retrograde transport in axoplasms treated with hT39 aggregates was observed when the average velocity over the last 20 minutes was analyzed (see Fig. 4B).