

Supporting Information

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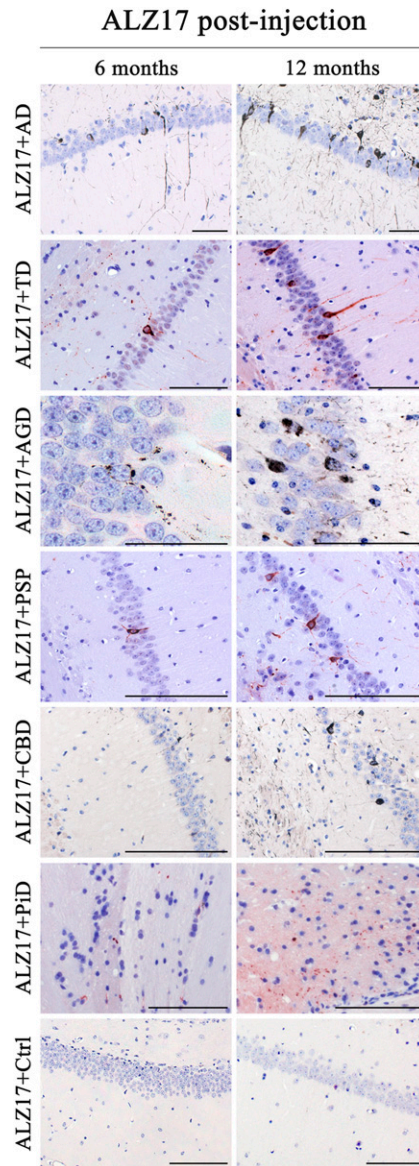


Fig. S1. Increase of the induced tau lesions over time in brains of mice transgenic for wild-type human tau (ALZ17 line) injected with human tau extracts 6 mo (*Left* column) and 12 mo (*Right* column) after injection. Gallyas-Braak silver impregnation in Alzheimer's disease (AD)-, argyrophilic grain disease (AGD)-, and corticobasal degeneration (CBD)-injected mice revealed a time-dependent increase in the number of tau lesions. The same phenomenon was observed after AT100 immunohistochemistry in tangle-only dementia (TD)-, progressive supranuclear palsy (PSP)-, and Pick's disease (PiD)-injected mice. Gallyas-Braak silver impregnation of an ALZ17 mouse injected with brain homogenate prepared from a control brain did not detect any tau pathology. (Scale bars, 100 μ m.) Sections were counterstained with hematoxylin.

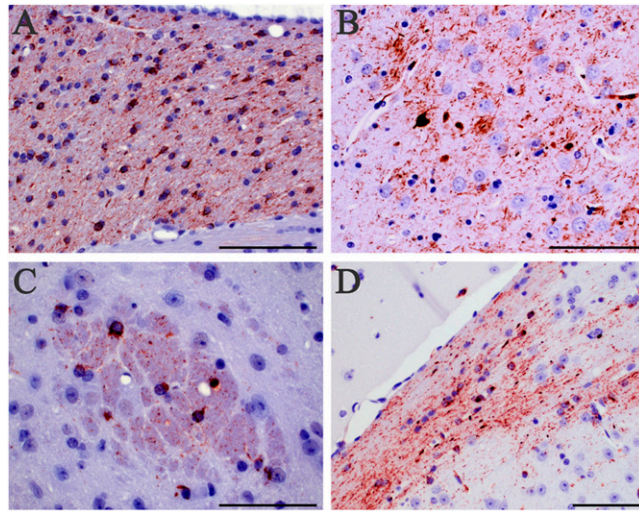


Fig. S2. Propagation of neuronal tau inclusions in ALZ17 mice following the intracerebral injection of brain homogenates from sporadic human tauopathies. AT100 antitau-immunostaining of (A) the fimbria, (B) the entorhinal cortex, and (C) the fornix 12 mo after the injection of brain homogenate prepared from an AGD case and (D) the optic tract 12 mo after the injection of brain homogenate prepared from an AD case. (Scale bars, 100 μm .) Sections were counterstained with hematoxylin.

