**TAU BLUE REVOLUTION**

*Tau* neurofibrillary pathology represents the main hallmark of AD and human neurodegenerative tauopathies. In the last decade, therapeutic strategies targeting misfolded tau protein have been gaining momentum. An increasing body of evidence shows that modulation of *tau* cascade has a beneficial effect in the animal models in preclinical studies. *Tau* therapy is becoming the leading therapeutic approach in AD.

10 **reasons** why Axon Neuroscience believes that tau is the proper target for AD-modifying therapy

1 *Tau* neurofibrillary pathology is the major correlate of clinical symptoms in Alzheimer’s disease.

2 Distribution of neurofibrillary pathology defines subtypes of Alzheimer’s disease with distinct clinical characteristics.

3 Decline in memory that occurs around 12 years before clinically diagnosed Alzheimer’s disease may correlate with the presence of neurofibrillary pathology in the temporal areas.

4 Neurofibrillary tangles precede amyloid beta pathology.

5 Cortical atrophy measured by MRI is associated with neurofibrillary pathology.

6 Neurofibrillary lesions and neuroinflammation display the same regional distribution in Alzheimer’s disease and other human tauopathies.

7 There is strong regional, inversely proportional relationship between the number of neurons and the number of neurofibrillary tangles.

8 *Tau* pathology in the absence of amyloid pathology strongly correlates with clinical features in human tauopathies such as progressive supranuclear palsy, corticobasal degeneration, tangle-only dementia and argyrophilic grain disease.

9 *Tau* gene mutations are pathogenic for frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17).

10 *Tau* animal models reproduce neuronal and glial tau pathology leading to the progressive cognitive and/or motor impairment.
Selected Scientific Papers


