Supplementary Material

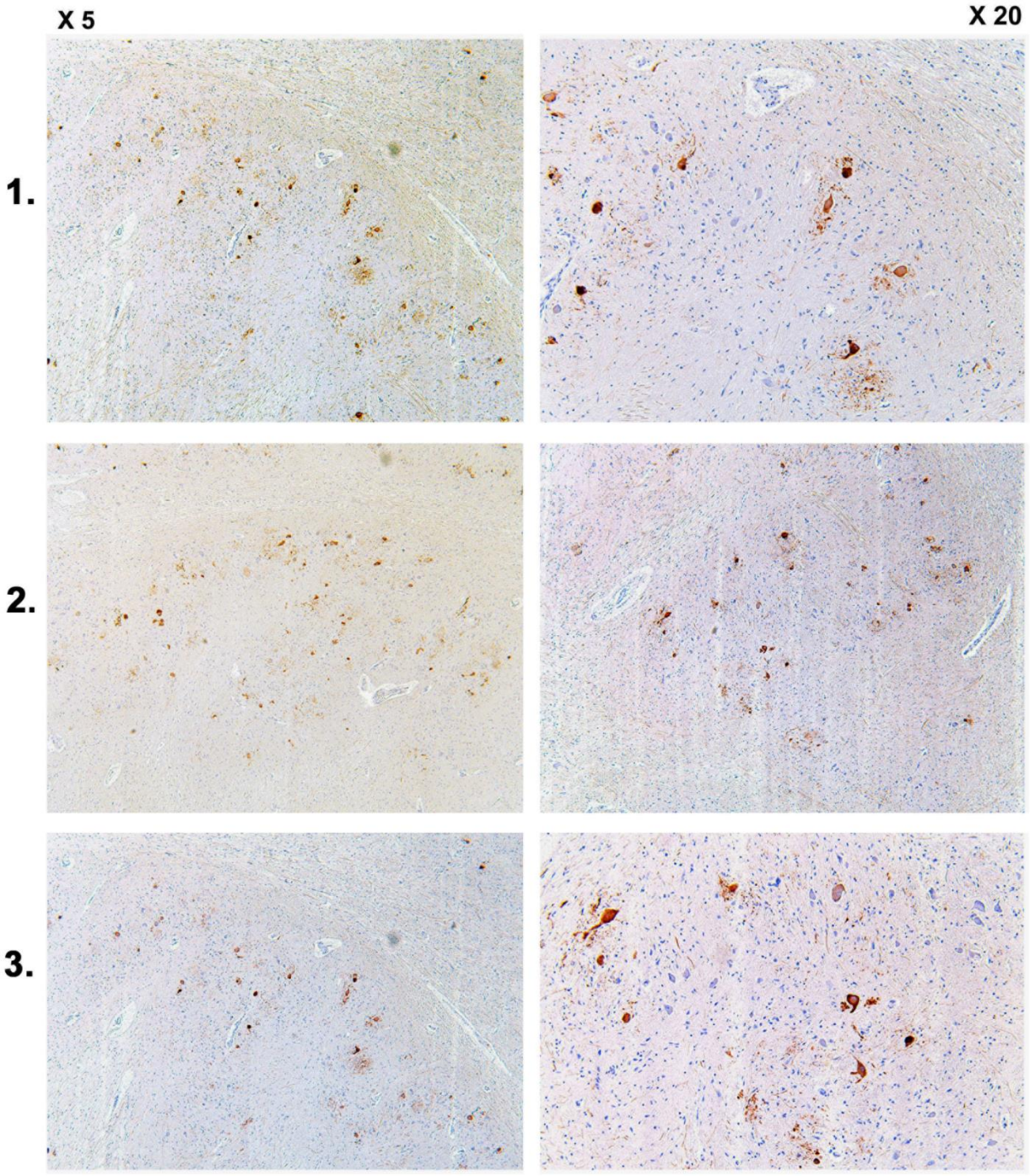
Supplementary Discussion

Both the clinical syndrome of SPT and the radiological and pathological changes in IO are known to vary depending on factors such as the type of injury (acute vs. chronic) to the DROP, the time since the injury[8,10], and other factors, such as coexisting lesions outside DROP. The presence or absence of vacuoles could be related to the rate of development of DROP lesions. It is plausible that the relative lack of vacuoles in IOH seen in PSP in our case suggests that relatively slow progressive DROP lesions will have a smaller potential to lead to vacuolation than lesions with abrupt onset. Alternatively, a cellular mechanism specific to neurodegenerative diseases such as PSP and PAPT could prevent vacuole formation, while allowing other transsynaptic degenerative changes to occur in the IOs. It was previously reported that vacuolization begins with neuronal hypertrophy in stage 3 of olivary degeneration, about 3 weeks after DROP lesions[8]. Additionally, vacuole formation was a typical feature in DROP lesion cases with long survival times, whereas acute cases showed no vacuolation.[48] This would argue against a time/rate related mechanism to explain the lack of vacuoles in our long-surviving **c**ase. Loss of synaptic vesicles and some stained vesicles being invaginated into the neuronal cytoplasm were seen using anti-synaptophysin antibody in an autopsy case approximately 2 years after cerebrovascular injurthe DROP.[49] Similar features were found in our case, suggesting that the lack of vacuoles in IOH in PSP and PAPT may be related to a disease-specific mechanism and not to the rate of IOH development.

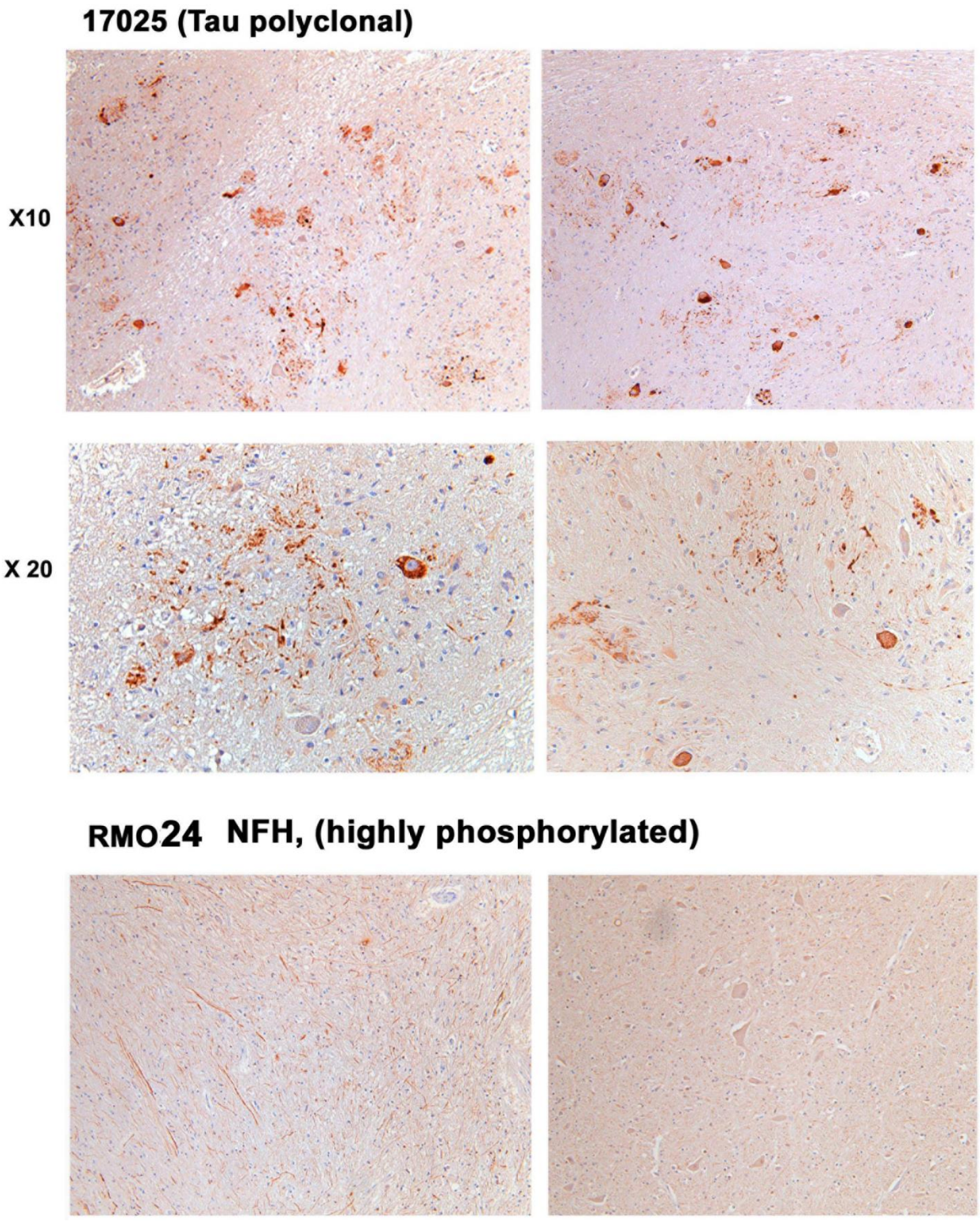
---

[48] Takamine K, Okamoto K, Fujita Y, Sakurai A, Takatama M, Gonatas NK. The involvement of the neuronal Golgi apparatus and trans-Golgi network in the human olivary hypertrophy. *J Neurol Sci* 2000; 182: 45-50.

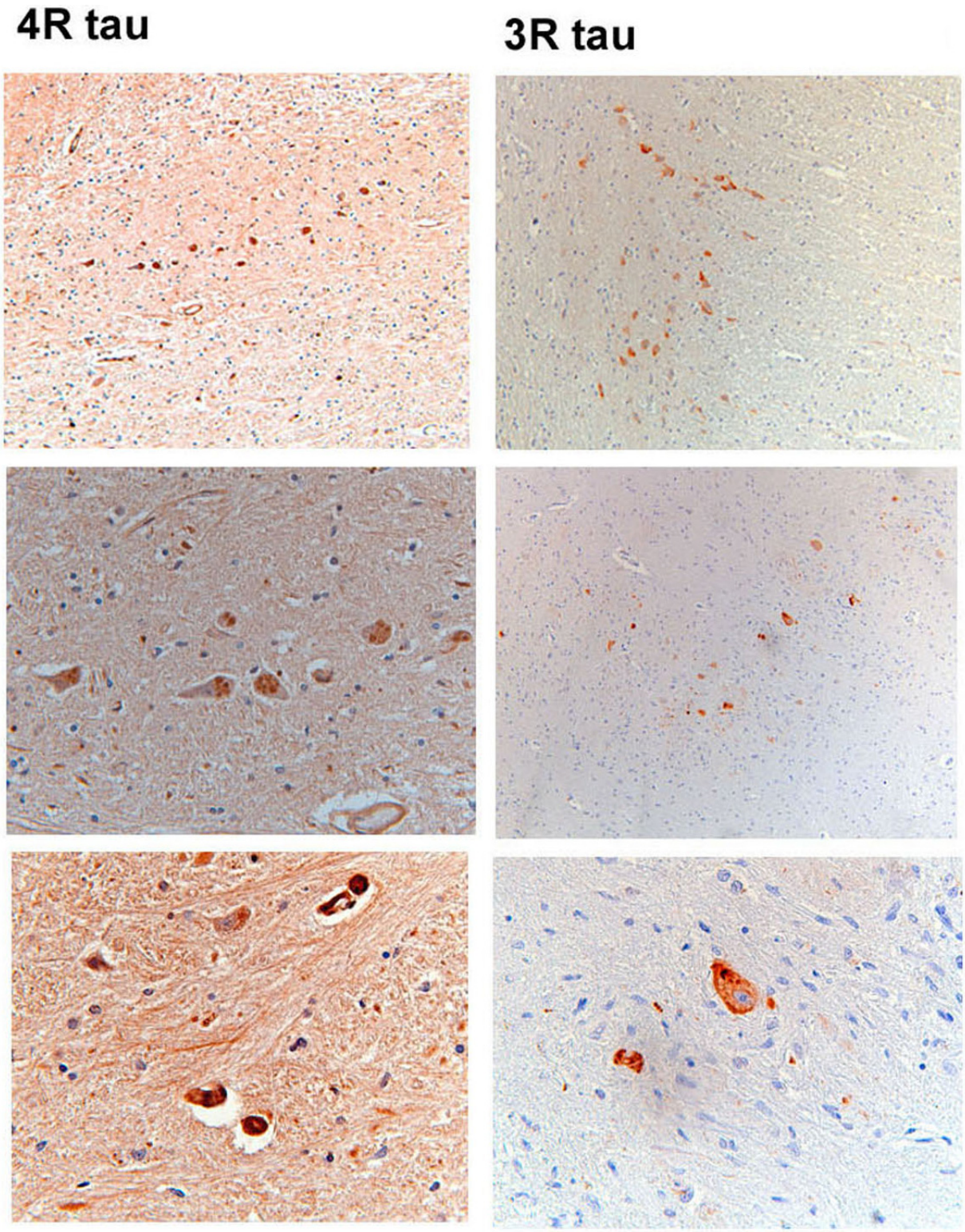
[49] Nishie M, Yoshida Y, Hirata Y, Matsunaga M. Generation of symptomatic palatal tremor is not correlated with inferior olivary hypertrophy. *Brain : a journal of neurology* 2002;125(Pt 6):1348-57.



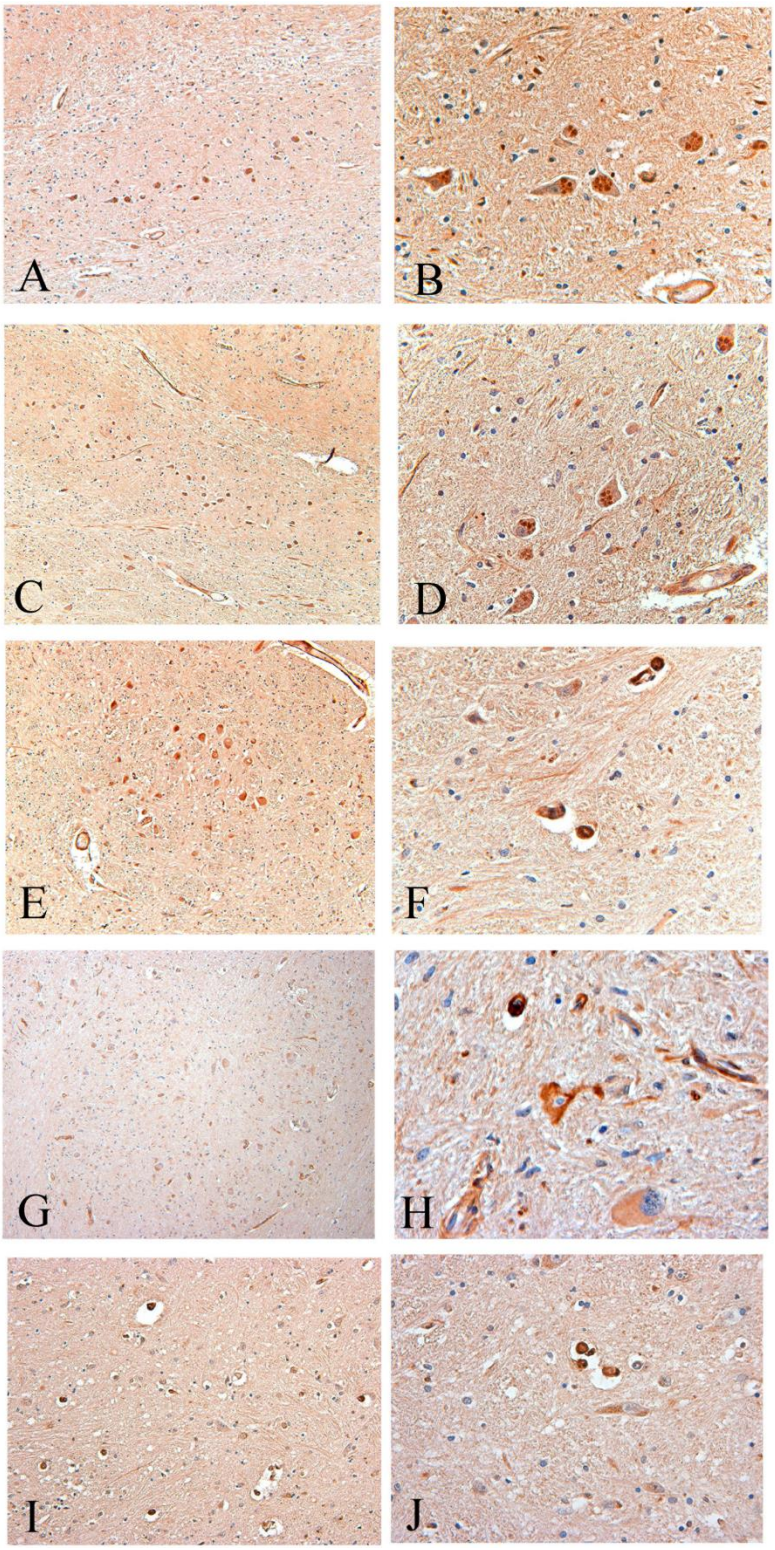
Supplementary Figure 1.
PHF1 (ser262; Pd) immunoreactivity in IOs. Three fields (1, 2, 3) shown in X5 (first column) and X20 magnification (second column).



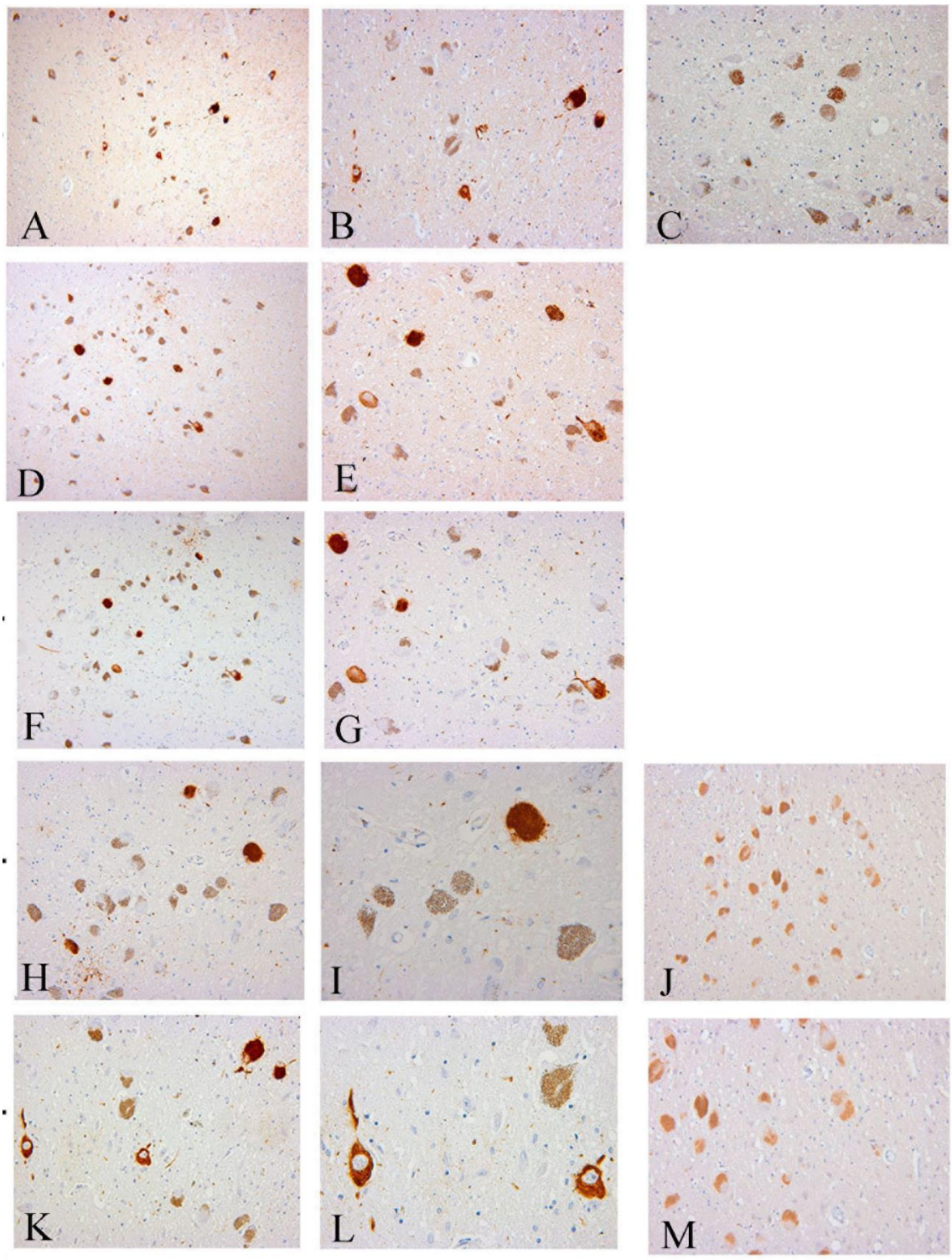
Supplementary Figure 2A.
Rabbit polyclonal α-Tau 17025 and neurofilament specific RMO24 (NFH-p+++) immunoreactivity in IOs.



Supplementary Figure 2B.
Medullar 4R tau and 3R tau immunoreactivity.



Supplementary Figure 3.
Medullar 4R tau (E10, 4R specific MAb, 1:1000) immunoreactivity. Five fields shown in X10 (first column: A,C, E, G, I) and X40 magnification (second column: B, D, F, H, J) from IO.



Supplementary Figure 4.
Locus ceruleus PHF1 (A, B, D, and E), AT8 (F, G, H, I, K, and L) tau, 3R tau (J and M), and ubiquitin (C) immunoreactivity (no other stains done for these sections). Magnification is X10 for A, D, F, H, and K, otherwise X40.