

Erratum

Modeling Tauopathy in the Fruit Fly *Drosophila melanogaster* By Vikram Khurana

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On p. 543, figure 1 is incorrect. The correct figure is:

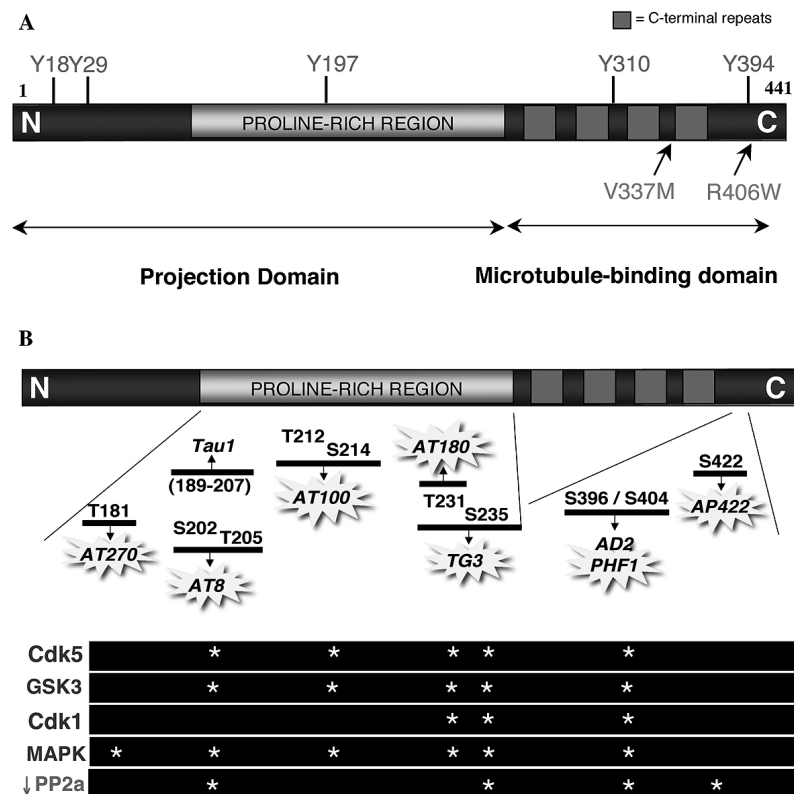


Fig. 1. A: Schematic diagram of 4R tau (adapted in part from [12]). Tau consists of projection and microtubule-binding domains separated by a proline-rich region. Tau has five tyrosines (Y) that are known targets for tyrosine kinases including Src family kinases. More than 25 mutations in tau are known to cause frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17). Two that have been used in *Drosophila* models are marked: Arg406→ Trp (R406W) and Val337→ Met (V337M). B: Schematic representation of commonly studied tau Serine/Proline (SP) or Threonine/Proline (TP) phosphoepitopes and the proline-directed kinases/phosphatases that can target these sites *in vitro* and *in vivo* [4,6]. Abbreviations: Cdk, cyclin-dependent kinase; MAPK, mitogen-activated protein kinase; GSK, glycogen synthase kinase; PP2a, protein phosphatase-2A. Note that phosphorylation within the microtubule-binding domain, not shown, is mediated by microtubule affinity-regulating kinase (MARK) and related kinases, targeting S262. Adapted from a figure generously provided by M. Steinhilb.

On p. 544, figure 2 is incorrect. The correct figure is:

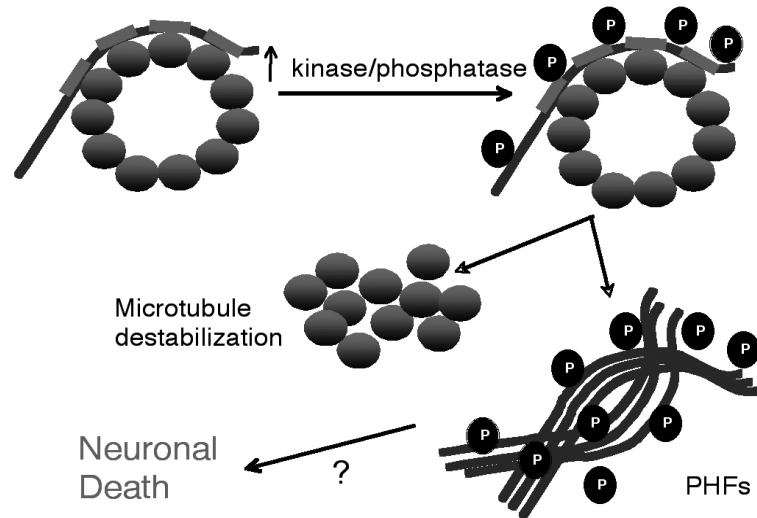


Fig. 2. **A commonly accepted general model for tauopathy pathogenesis.** Hyperphosphorylation (P) of tau (blue) bound to microtubules (green), perhaps as a result of a disturbed kinase/phosphatase balance, leads to detachment and aggregation of tau into paired helical filaments (PHFs) and microtubule destabilization. This is thought to lead to neuronal dysfunction and death, although the mechanisms through which this occurs are unclear.