Expanded View Figures

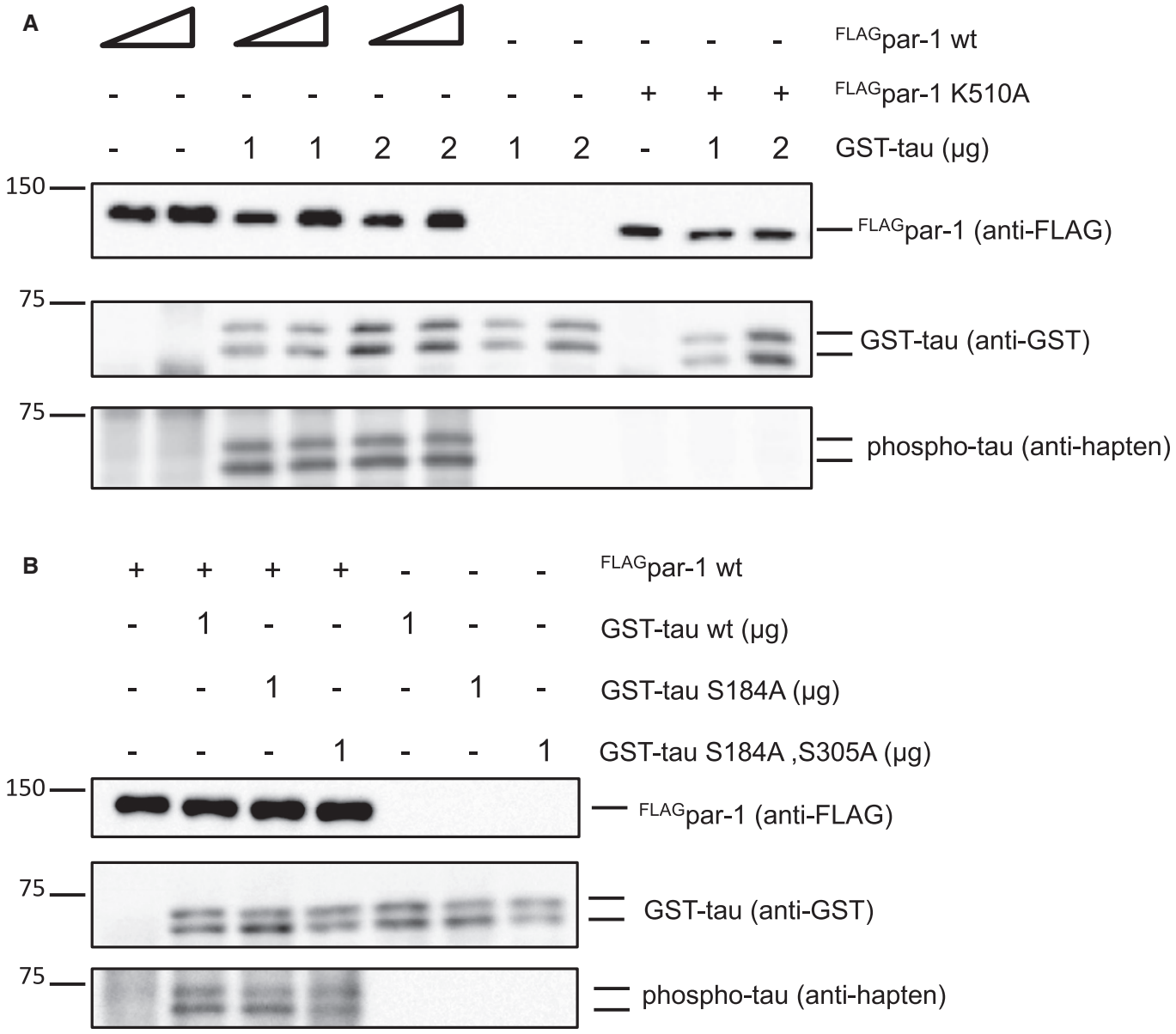

Figure EV1. PAR-1 phosphorylates *Drosophila tau*.

A The indicated combinations of recombinant ^{FLAG}PAR-1 (isoform RR, wild-type, or kinase-dead K510A) from S2 cells and bacterially expressed GST-tau were incubated with ATPγS, and thiophosphorylated proteins were detected after alkylation with an antibody against a semisynthetic thiophosphate epitope (anti-hapten) (Allen *et al*, 2007).

B Phosphoassay with wild-type ^{FLAG}PAR-1 and the indicated GST-tau phosphomutants.

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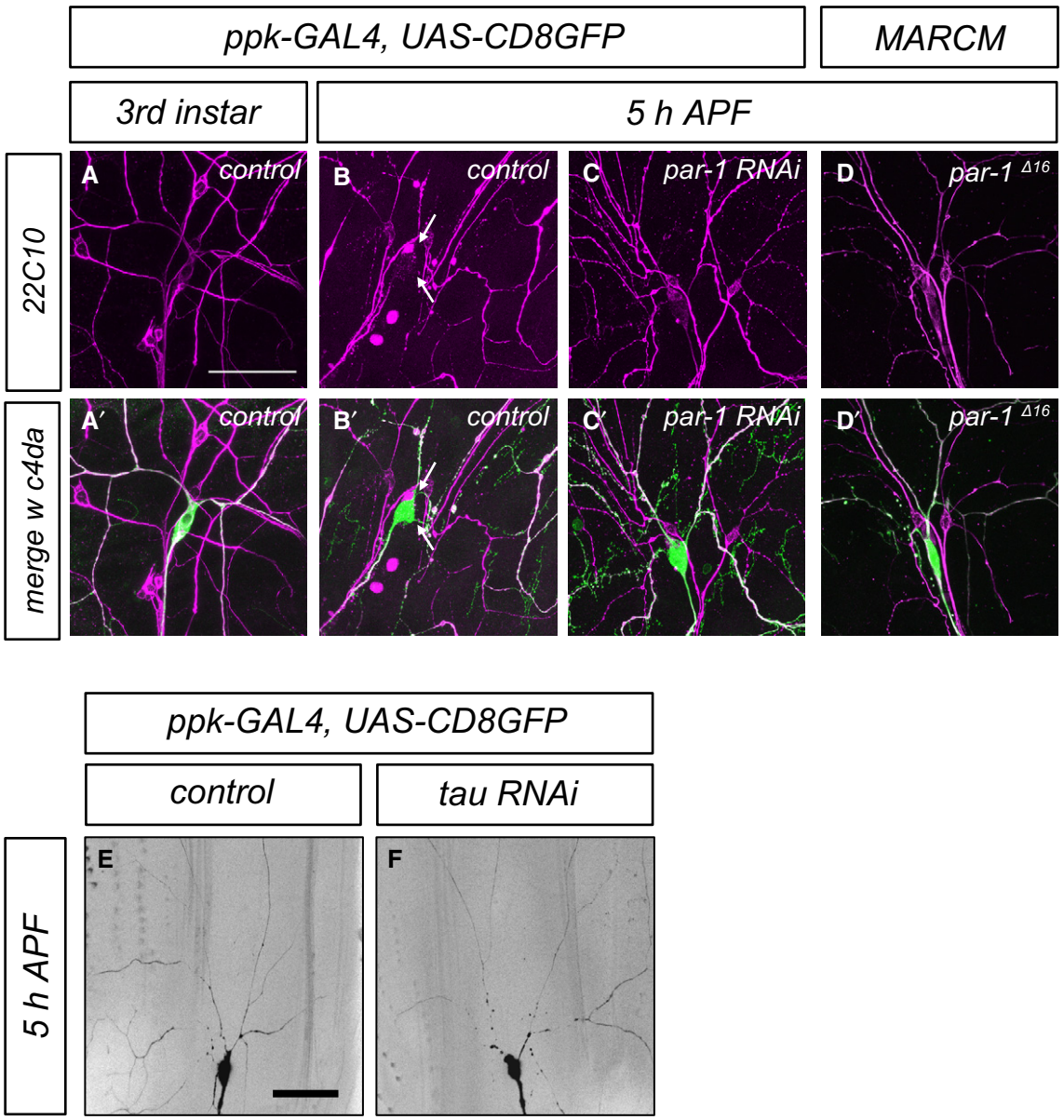

Figure EV2. Futsch/MAP1B distribution during c4da neuron dendrite pruning and effects of *tau* depletion on dendrite pruning.

A–D′ Futsch/22C10 distribution in c4da neurons. Panels (A–D) show Futsch/22C10 staining, and panels (A′–D′) show the merge with the c4da neuron marker (*ppk>CD8GFP* in A–C, *tdtomato* in D). (A, A′) Control c4da neuron at the third-instar larval stage. (B, B′) Control c4da neuron at 5 h APF. Futsch/22C10 is lost from proximal dendrites (arrows). (C, C′) A c4da neuron expressing *par-1* RNAi at 5 h APF still shows strong dendritic Futsch/22C10. (D, D′) A *par-1*$^{\Delta 16}$ mutant MARCM c4da neuron at 5 h APF still shows strong dendritic Futsch/22C10.

E, F *tau RNAi* does not trigger precocious dendrite pruning at 5 h APF. Representative images of control c4da neurons (E) or c4da neurons expressing *tau RNAi* under the control of *ppk-GAL4* (F) are shown.

Data information: Scale bars are 50 μm.

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