site	antibody	reference
S202	CP13	Dr.Peter Davies
T231	AT180	Thermo
S262	pS262	Biosource

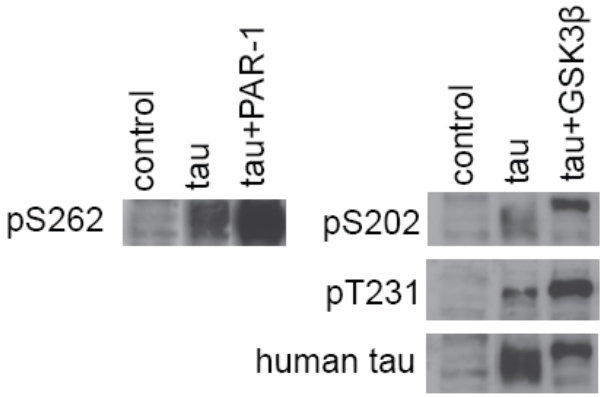

Figure S1. Characterization of the antibodies used to detect tau phosphorylation at Ser202, Thr231, and Ser262. (A) The antibodies and sources. (B) Western blot of fly head extracts. Control: flies carrying the pan-retinal gmr-Gal4 driver alone. (Left panel) Flies expressing human tau alone (tau), or co-expressing tau and GSK3β (tau+GSK3β). (Right panel) Flies expressing human tau alone (tau), or co-expressing tau and dMARK (tau+dMARK).

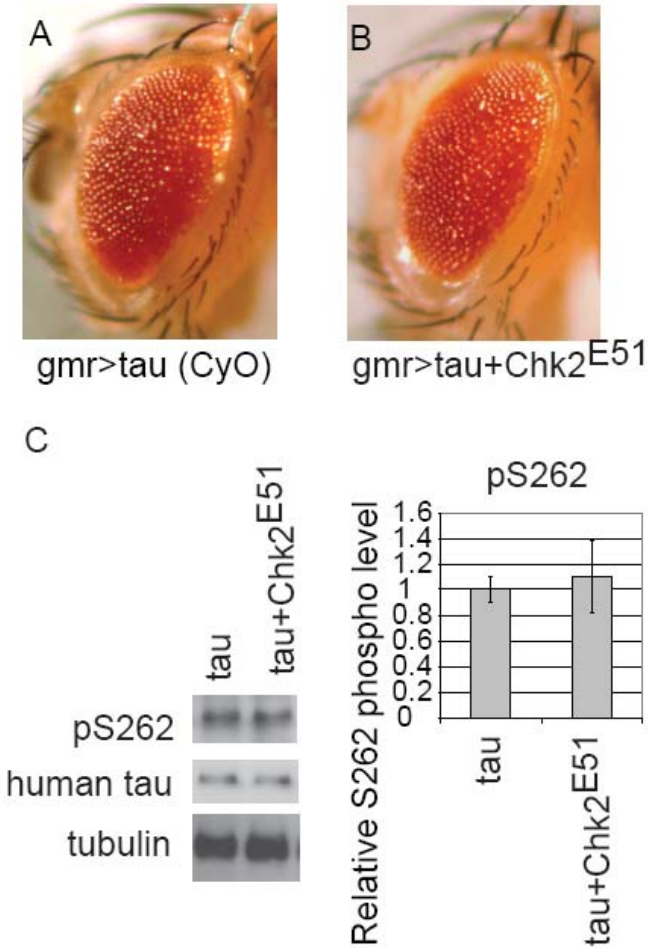

Figure S2. Reduction in endogenous Chk2 does not ameliorate human tau-induced eye degeneration. The external eyes of flies expressing human tau with the pan-retinal gmr-GAL4 in the control background (A), and of flies carrying one copy of a loss-of-function mutation in Chk2 (B), are shown. (C) One copy of a loss-of-function mutation in Chk2 does not reduce tau phosphorylation levels at Ser262. Fly heads expressing human tau in the control background (tau) or in the background with one copy of the loss-of-function mutation in Chk2 (tau+Chk2E51) were subjected to Western blotting with anti-tau (total tau) or anti-pSer262 (p-tau) antibodies. The phosphorylation levels of tau in head lysate from the animals carrying the Chk2 loss-of-function mutant were normalized to tubulin levels and shown as a ratio relative to fly heads expressing tau in the control background. No significant difference was detected (n=4 or 5, $p>0.05$, Student's t-test). Representative blots are shown.