

Supplementary data

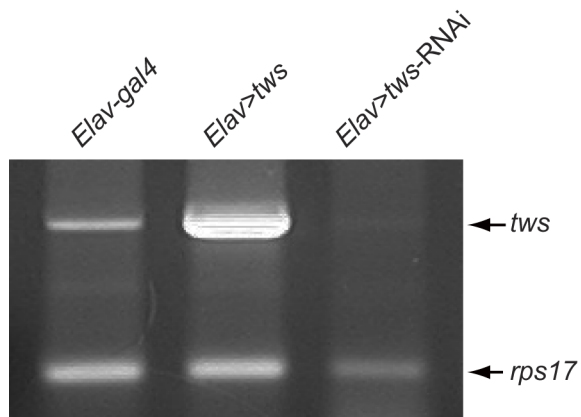


Fig. S1. Relative expression level of *tws*. UAS-*tws* and UAS-*tws*-RNAi transgenic constructs were driven by *Elav-gal4*. 10 μ g of purified total RNA was reverse transcribed using oligo(dT) primers and SuperScriptase (invitrogen). PCR was performed using *tws* specific primers (5'-GGAGCTAACAGAGGTGATC-3' and 5'-CACGCTGATCTCATCCTTCTTTCG-3') and *rps17* primers (5'-CGAACCAAGACGGTGAAGAAG-3' and 5'-CC TGCAACTTGATGGAGATACC-3'). PCR condition: at 94°C for 2 min, 30 cycles (at 94°C for 30 sec, at 40°C for 30 sec, at 72°C for 1 min), and at 72°C for 7 min. Samples were resolved in 1.5% agarose gel.

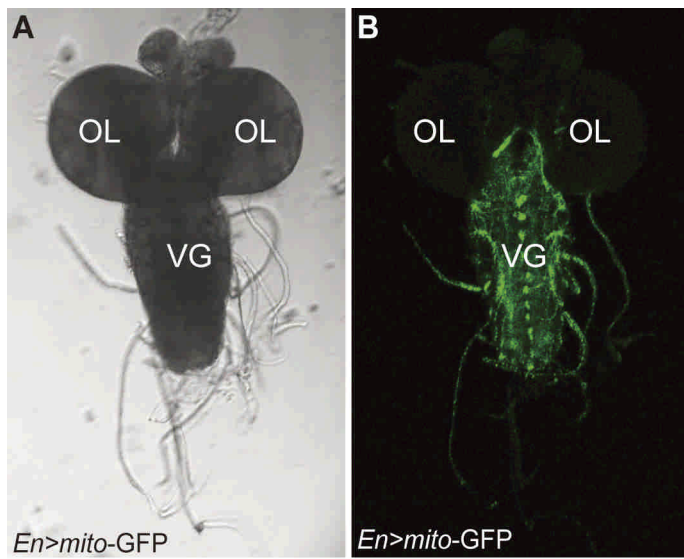


Fig. S2. *En-gal4* drives the expression of *mito-GFP* in neuritis of larva. (A) Larval brain lobe (optical view). (B) Mitochondria were labeled with *mito-GFP*. Expression of transgenic constructs in the neurites of ventral ganglia using *En-gal4*. OL, optical lobe; VG, ventral ganglion

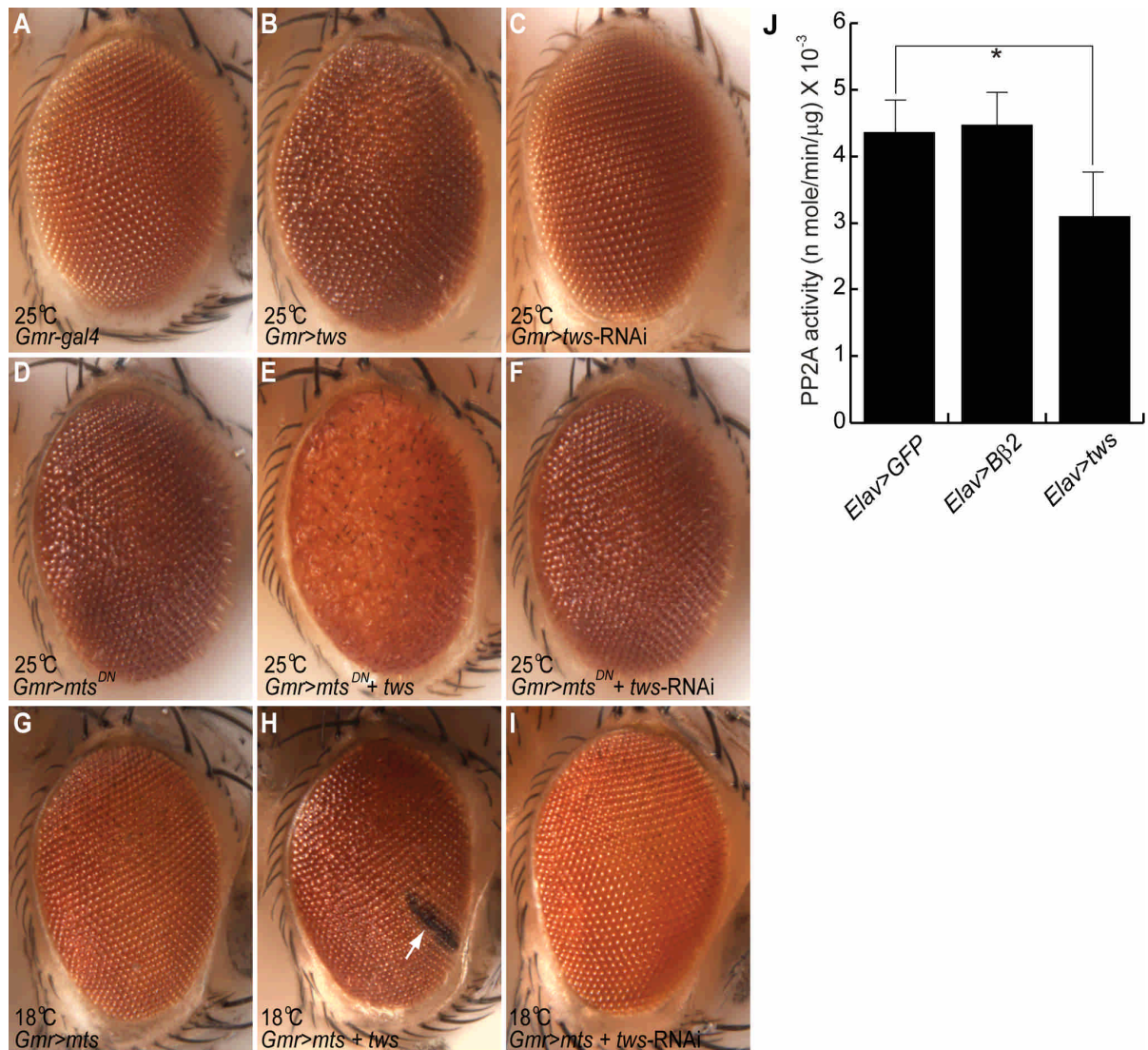


Fig. S3. *tws* induces apoptosis. Genetic crosses were conducted (A-F) at 25°C and (G-I) 18°C. (A) Control *Gmr-gal4* flies showed normal eye morphology. (B) Ectopic *tws* induce mild rough eye phenotype. (C) No obvious eye defect in *Gmr>tws-RNAi* flies. (D) Ectopic overexpression of dormant negative *mts* (*mts^{DN}*) caused very mild rough eye phenotype. (E). Depigmentation of ommatidia in flies co-expressing *mts^{DN}* and *tws*. (F) Very mild rough eye phenotype was observed in flies co-expressing *mts^{DN}* and *tws-RNAi*. (G) Targeted overexpression of *mts* under the control of *Gmr-gal4* caused lethality at 25°C. However, the flies were survival with no retinal defect at 18°C. (H). Ectopic *mts* and *tws* synergistically caused eye degeneration. Necrotic spots were consistently observed (white arrow). (I) Normal eye morphology in flies co-expressing UAS-*mts* and UAS-*tws-RNAi* transgenes. (J) PP2A activity was quantified using a PP2A activity assay kit (Promega, V631A). PP2A activity was not significantly increased in the heads of transgenic flies expressing Bβ2 flies, but its activity was significantly reduced in *tws*-expressing flies. Data were expressed as mean ± sd values and analyzed by Student's *t*-test; * indicates $p < 0.01$. (n = 3).

A

```

Bβ2 MKCFSRYLPIYIFR-----PENTILSSS-----
Tws MGRWGRQSPVLEPPDPQMOTPPPPTLPPRTFMRQSSITKIGNMLNTAININGAKK
Bβ2 -----CHTEADIISTVEFNHTGELLATGDKGGRVVIHQREIQ
Tws PASNGEASWCFSQIKGALDDDDVTADAILSCVEFNHDGELLATGDKGGRVVIHQREIQ
Bβ2 ESKNQVHRERGEYNVYSTFQSHEPEFDYLSLEIEEKINKIRWLPCQNAAYFLLSTN
Tws ASKAANPRERGEYNVYSTFQSHEPEFDYLSLEIEEKINKIRWLQCKNPVHLLSTN
Bβ2 DKTVKLWKVSRDKRPEGYNLKDEECRLRDPATITTLRVPLRPMDLMEATPRRV
Tws DKTVKLWKVSRDKSFGGYNTKEENGLIRDPQNVTLRVPSVKQIPLLVEASPRRT
Bβ2 FANAHTYHINSISVNSDYETYSADDLRINLWNFELTNQSFNIVDIKPANMEELTE
Tws FANAHTYHINSISVNSDQETELSADDLRLNHLWLEVVNOSYNIVDIKPTNMEELTE
Bβ2 VITAAEFHPPHCNTEFVYSSSKGTIRLCDMRASALCDRHTEFEEPELFSNRFFSE
Tws VITAAEFHPTFCNVEFVYSSSKGTIRLCDMRSAALCDRHSEKQEEPEPEINRFFSE
Bβ2 IISSISDVKFS SGRYIMTRDYLTIVKVDLNMENRPIETYQVHDYLRSKLCSLYEN
Tws IISSISDVKLS SGRYIMSRDYLSTIKVVDLHMETKPIETYPVHEYLRAKLCSLYEN
Bβ2 DCIFDKFECVWNGSDSVIMTGSYNNFFRMFDRNTKRDVTLASRENSKPRAILKPR
Tws DCIFDKFECVWNGKSSIMTGSYNNFFRVFDRNSKKDVTLEASRDIIKPKTVLKPR
Bβ2 KVCVGGKRRKDEISVDLSLDFSKKILHTAWHPSENTIAVAATNNLYIFQDKVN
Tws KVCTGGKRRKDEISVDCLDFNKKILHTAWHPENI IAVAATNNLFI FQDKF

```

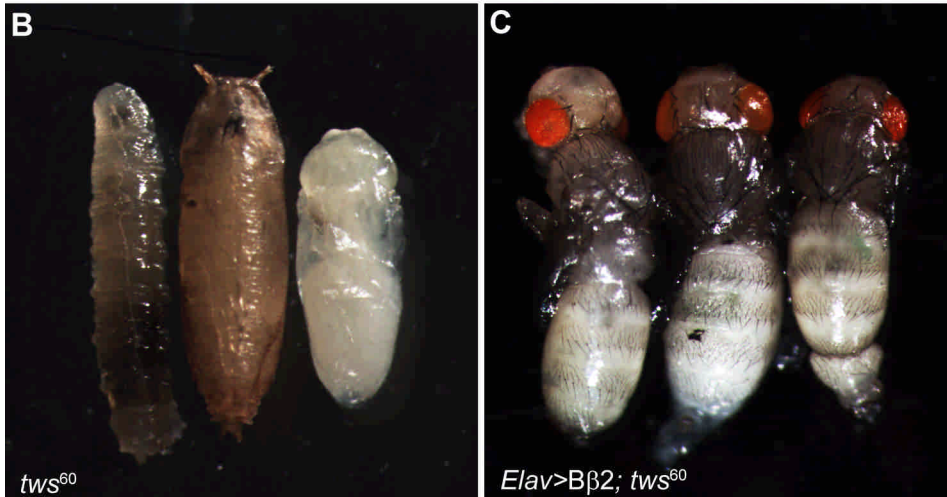


Fig. S4. *Tws* and Bβ2 are functionally equivalent. (A) Amino acid sequence alignment of Bβ2 and *Tws*. The entire sequence of *Tws* is highly homologous to the human Bβ2 except that the N termini of both proteins are less conserved. Identical and conserved amino acid residues are shaded in black and grey respectively. (B) Majority of homozygous *tws*⁶⁰ mutants die at larval stages. Only few animals would survive to early pupal stages (3.58%). (C) Neuronal overexpression of Bβ2 driven by *Elav-gal4*, were able to rescue the survival of *tws*⁶⁰ to the pharate adult stages.