

Figure S1. Independent assessment of Tn function in the prevention of DEOM1 muscle breakdown. (A-C) DEOM1 histolysis is blocked upon a complete loss of Tn at 12 h APF. (A) Histolysis is complete in *WT* DEOM1 muscles (yellow asterisk), while DEOM2 is still present at 12 h APF (white solid line) (B) All DEOM1 (yellow solid line) and DEOM 2 muscles have failed to degenerate in *m* mutants. (C) Bar

graph indicates the absence of DEOM1 histolysis in *tn* -/-. (D-F) Reduction of Tn with a second *tn RNAi* line (VDRC19290) shows a significant reduction in DEOM1 histolysis at 12 h APF. *mef2>tn RNAi* partially blocks DEOM1 muscle histolysis (E) compared to *mef2-Gal4/+* control muscles (D). (F) Quantification of DEOM1 histolysis in control or *tn RNAi* muscles. (G-I) The addition of an exogenous *UAS-GFP RNAi* (H) in a *mef2>tn RNAi* background (G) does not alter Tn-mediated muscle histolysis at 24 h APF. (I) The percentage of DEOM1 muscle histolysis in *tn RNAi* is comparable to the addition of GFP OE (*UAS-GFP* nnAi (*UAS-GFP RNAi*). Thus an additional UAS element does not titrate out the amount of Gal4 protein used to drive *tn RNAi*. Mean \pm SEM (n.s., not significant, **** p < 0.001, *** p < 0.005). Scale bar, 100µm.

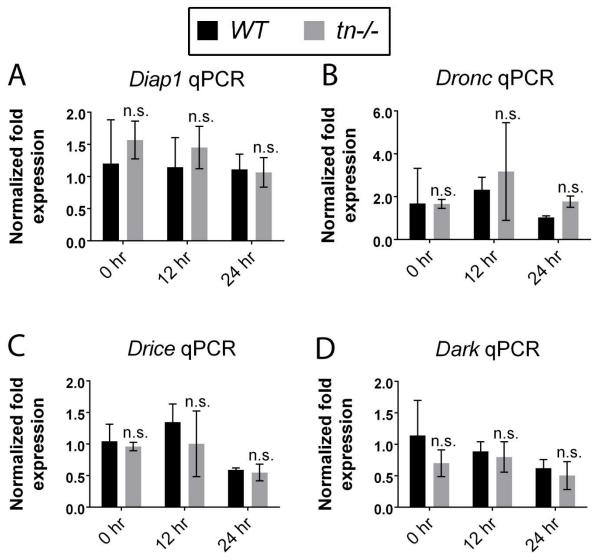


Figure S2. *tn* does not regulate the transcript levels of cell death components. (A-D) qPCR was used to assess mRNA levels from whole pupae in *WT* or *tn* mutants at the indicated time points before (0 h), during (12 h), or after (24 h) DEOM1 histolysis. There are no significant differences in the mRNA levels of *Diap1* (A), *Dronc* (B), *Dark* (C) or *Drice* (D) in *tn* -/- as compared to *WT* pupae at any stage of DEOM histolysis. Mean \pm SEM, (n.s., not significant).

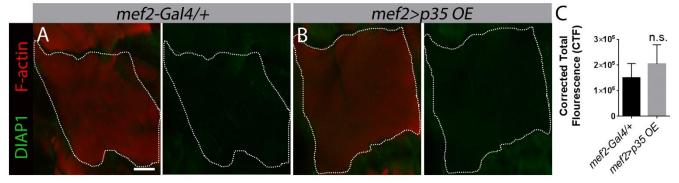


Figure S3. Overexpressing *p*35 does not affect DIAP1 levels in the DEOMs. Confocal micrographs of abdominal muscles co-labeled for F-actin (red) and DIAP1 (green). (A-B) There is no significant difference in DIAP1 levels between control *mef2-Gal4/+* (A) or *mef2>p35 OE* (B) muscles at 12 h APF. (C) A bar graph showing the corrected total fluorescence of DIAP1 immunostaining in the indicated genotypes. Mean \pm SEM, (n.s., not significant). Scale bar, 50µm.

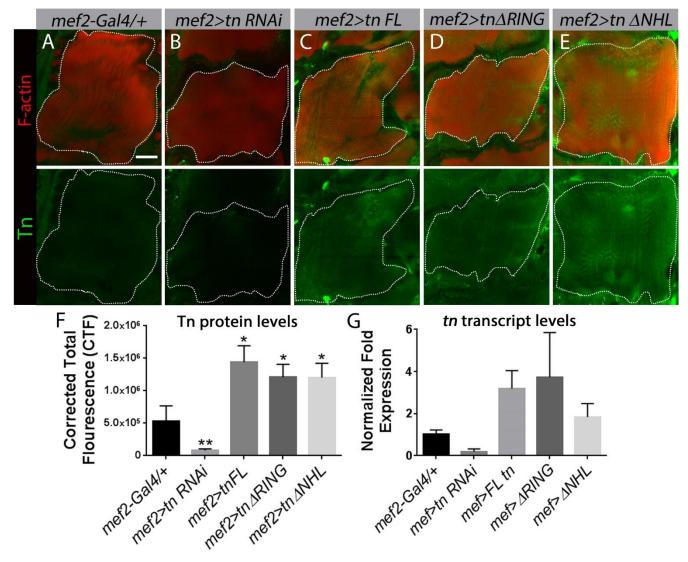


Figure S4. Expression levels of Tn protein and *tn* transcripts at 12 h APF. (A-E) Confocal micrographs of DEOMs to visualize F-actin (red) and Tn protein (green). (F) There is a significant upregulation of Tn protein levels in *mef2>tn FL*, *mef2>tn* $\Delta RING$ and *mef2>tn* ΔNHL DEOM muscles over *mef2-Gal4/+* control muscles or upon knockdown of *tn* using RNAi. (G) A bar graph showing upregulation of *tn* transcript levels in the indicated *UAS-tn* overexpression constructs. Mean ± SEM, (**, p<0.01, * p<0.05). Scale bar, 50µm.

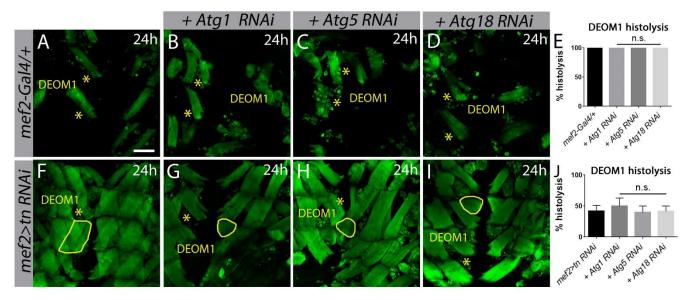


Figure S5. DEOM histolysis does not require autophagy. (A-D) There is no significant difference in DEOM1 breakdown between *mef2-Gal4/+* control (A) or *mef2>Atg1RNAi* (B), *mef2>Atg5 RNAi* (C) and *mef2>Atg 18a RNAi* (D) pupae. (E) A bar graph showing complete histolysis in all genotypes examined at 24 h APF. (F-I) DEOM degeneration is not significantly enhanced in upon a reduction in *Atg1* (G), *Atg5* (H), and *Atg18* (I) compared to *mef2;tn RNAi* alone (F). (J) Quantification of DEOM1 histolysis in (F-I). Mean \pm SEM, (n.s., not significant). Scale bar, 100µm.