



Figure S7

Fig S7| XBP-1 mediates ER-stress-induced autophagy activity

- (A-D) Expression of *Phsp-4::GFP* is very weak in *rpl-43(bp399)* (A,B) and *xbp-1(zc12)* (C,D) mutant animals. (A) and (C): DIC images of the animals shown in (B) and (D), respectively.
- (E-F) Expression of *Phsp-4::GFP* in wild-type animals is dramatically elevated upon tunicamycin treatment.
- (G-H) Tunicamycin treatment suppresses the accumulation of SQST-1::GFP aggregates in *rpl-43(bp399)* mutant animals.
- (I-L) Expression of *Phsp-4::GFP* is dramatically increased in *pep-2(RNAi)* (I,J), but is suppressed by simultaneous depletion of *xbp-1* activity (K,L).
- (M-N) Compared to *rpl-43(bp399)* mutants, accumulation of SQST-1::GFP aggregates in the intestine is not affected by loss of function of *xbp-1*.
- (O-P) The number of SQST-1::GFP aggregates is dramatically decreased in the intestine in *rpl-43(bp399); pep-2(RNAi)* mutant animals.
- (Q-R) Loss of function *xbp-1* restores the accumulation of SQST-1::GFP aggregates in *rpl-43(bp399); pep-2(RNAi)* mutants.
- (S-T) SQST-1::GFP aggregates are largely absent in *unc-89(RNAi); rpl-43(bp399)* mutant animals.
- (U) Loss of function of *xbp-1* does not restore the accumulation of SQST-1::GFP aggregates in *unc-89(RNAi); rpl-43(bp399)* mutant animals.
- (V) SQST-1::GFP aggregates are restored in *unc-89(RNAi); rpl-43(bp399); atf-6(ok551)* mutant animals.

(W-Z) Loss of function of *xbp-1* does not restore the accumulation of SQST-1::GFP

aggregates in *let-363(RNAi)*; *rpl-43(bp399)* (W,X) and *lin-35(RNAi)*;

rpl-43(bp399) (Y,Z) mutant animals.

(A2-B2) GFP::LGG-1 forms a large number of puncta in the intestine in *pep-2(RNAi)*

animals.

(A), (C), (E), (G), (I), (K), (M), (O), (Q), (S), (W), (Y) and (A2): DIC images of the

animals shown in (B), (D), (F), (H), (J), (L), (N), (P), (R) (T), (X), (Z) and

(B2), respectively. Scale bars: 100 μm (A-F,I-L); 20 μm (G,H,M-Z); 10 μm

(A2,B2).