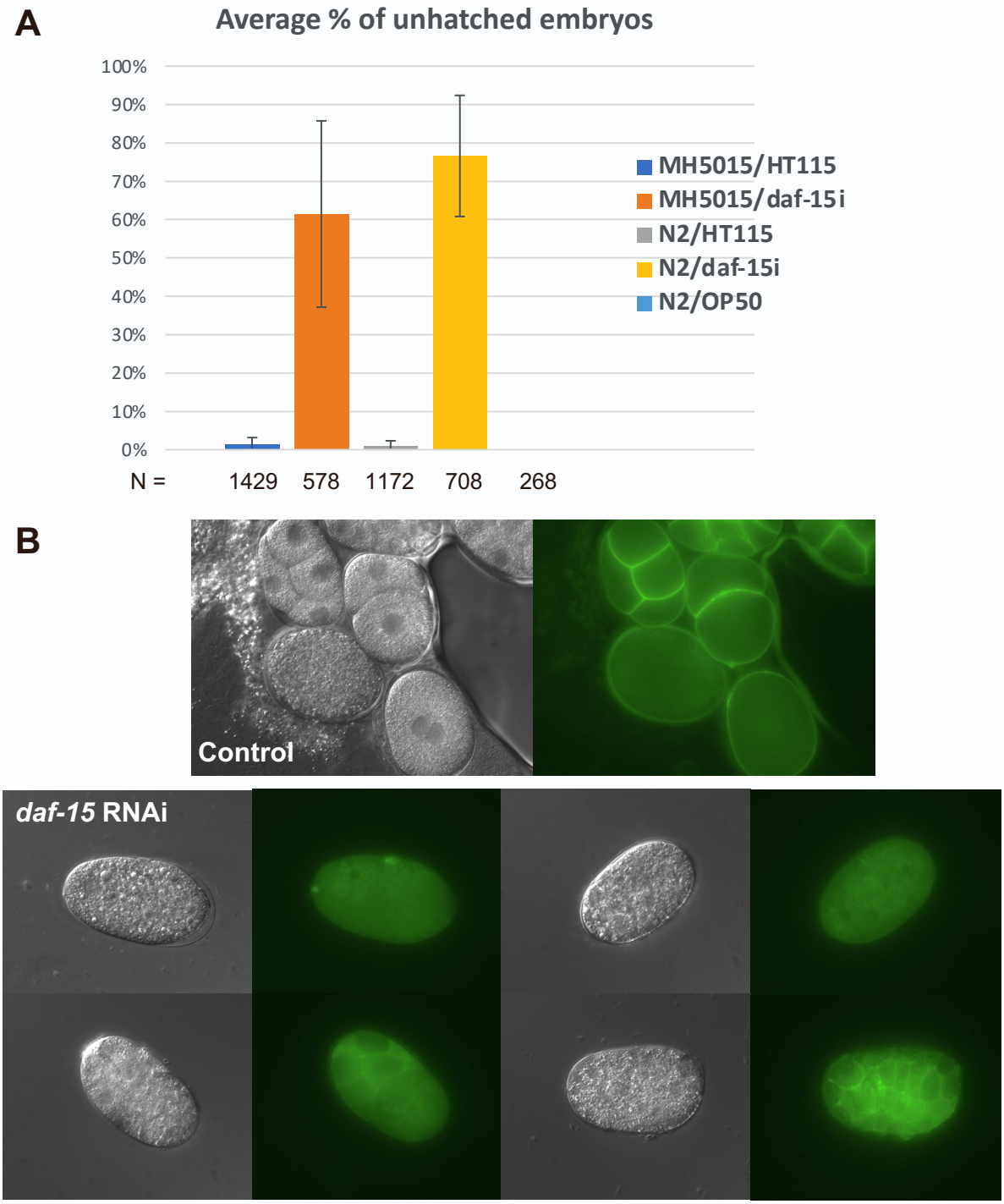
iScience, Volume *25*

Supplemental information

The TORC1 phosphoproteome in *C. elegans* reveals roles in transcription and autophagy

Aileen K. Sewell, Zachary C. Poss, Christopher C. Ebmeier, Jeremy R. Jacobsen, William M. Old, and Min Han

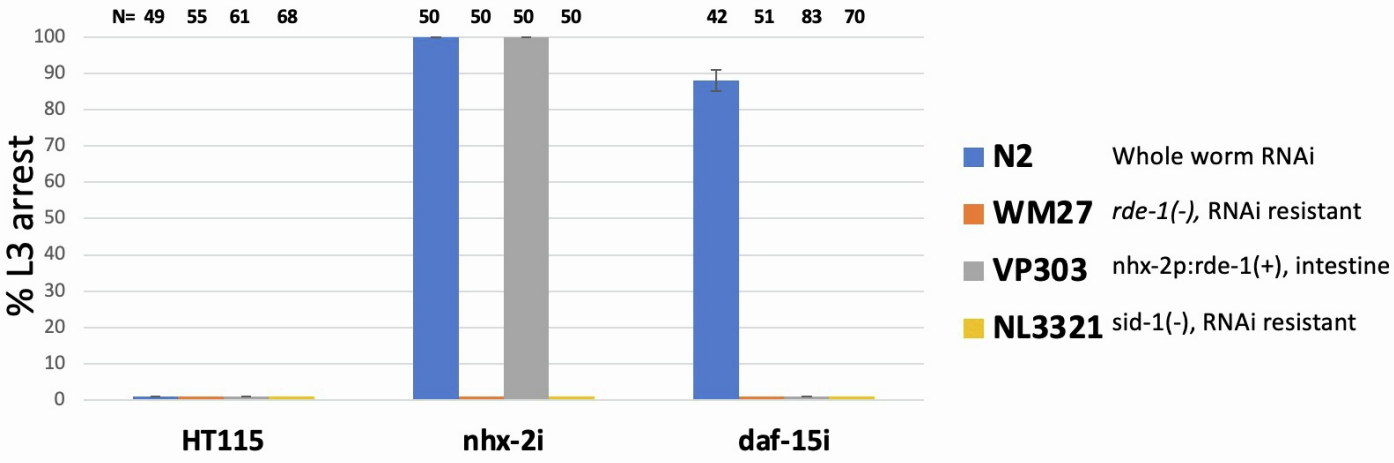
Supplemental Figure 1



Supplemental Figure 1, related to Figure 2. *daf-15(RNAi)* causes an embryonic hatching defect.

A. Bar graph quantification of the % of unhatched embryos found within a population with the noted treatment [HT115 empty vector control strain, *daf-15(RNAi)* or OP50 *E. coli*]. This embryonic phenotype was seen in both treated wild-type (N2) and cas9 DAF-15::mCherry (MH5015) worms. Graph represents the average of 4 or more replicates. N = number embryos scored. Error bars are StdDev. **B**. Representative microscopy images of OD95 embryos from mothers fed either the RNAi control strain (upper panel) or *daf-15(RNAi)* (lower panels). The GFP plasma membrane marker PH(PLCdelta1) highlights the lack of organized structures in the *daf-15(RNAi)*-treated sample.

Supplemental Figure 2



Supplemental Figure 2, related to Figure 3. *daf-15(RNAi)* in multiple tissues is required to cause larval arrest.
Bar graph quantification of feeding RNAi results [HT115 control, *nhx-2(RNAi),* and *daf-15*(*RNAi)]* in wild type (N2) and tissue-specific RNAi mutant worms. Data are represented as percentage of population arrested at L3 stage. N = number embryos scored. Error bars are StdDev. Only whole worm RNAi in N2 was sufficient to cause larval arrest with *daf-15(RNAi).* Intestine-specific *daf-15(RNAi)* had no impact on development. *nhx-2(RNAi)* is provided as an intestine-specific target and control.