

Gene name	Allele name	Molecular lesion	Recessivity	Maternal effect
<i>sti-1</i>	<i>u1071</i>	R253*	recessive	Yes
<i>pph-5</i>	<i>u1072</i>	C381Y	recessive	Yes
<i>pph-5</i>	<i>u1073</i>	P448L	recessive	Yes
<i>pph-5</i>	<i>u1074</i>	Q325*	recessive	Yes
<i>pph-5</i>	<i>u1075</i>	Q192*	recessive	Yes
<i>dlk-1</i>	<i>u1105</i>	V844I	recessive	Yes
<i>dlk-1</i>	<i>u1138</i>	W394*	recessive	Yes
<i>maph-1.3</i>	<i>u1107</i>	E794K	dominant	N/A
?	<i>u1108</i>		dominant	N/A
?	<i>u1106</i>		semi-dominant	N/A
?	<i>u1142</i>		semi-dominant	N/A
?	<i>u1144</i>		semi-dominant	N/A

Table S1. Summary of the mutants isolated from the *mec-15(-)* suppressor screen. These alleles led to the production of a long ALM-PN in *mec-15(u1042); mec-7(u278); uIs115[mec-17p::TagRFP]* animals. * represents a stop codon; ? means the mutant allele was not mapped.

Gene name	Transcript name	Description	Long ALM-PN
<i>pph-5</i>	Y39B6A.2	Protein Phosphatase 5	Positive
<i>sti-1</i>	R09E12.3	Hsp90 co-chaperone; Hop/STI1	Positive
<i>hsp-1</i>	F26D10.3	Hsp70 family	Negative
<i>hsp-110</i>	C30C11.4	HSPA4; Hsp70 family	Positive
<i>hsp-3</i>	C15H9.6	HSPA5; Hsp70 family	Negative
<i>hsp-4</i>	F43E2.8	HSPA5; Hsp70 family	Negative
<i>hsp-6</i>	C37H5.8	HSPA9; Hsp70 family	Negative
<i>stc-1</i>	F54C9.2	HSPA13; Hsp70 family	Negative
<i>hsp-70</i>	C12C8.1	Hsp70 family	Negative
<i>F44E5.4</i>	F44E5.4	Hsp70 family	Negative
<i>F44E5.5</i>	F44E5.5	Hsp70 family	Negative
<i>T14G8.3</i>	T14G8.3	HYOU1; Hsp70 family	Negative
<i>hsp-75</i>	R151.7	Hsp90-related protein; Hsp75/TRAP1	Negative
<i>enpl-1</i>	T05E11.3	Molecular Chaperone; GRP94/GP96	Negative
<i>chn-1</i>	T09B4.10	CHIP; Hsp70-interacting protein	Negative
<i>daf-41</i>	ZC395.10	Hsp90 co-chaperone; p23	Positive
<i>aha-1</i>	C25A1.11	Hsp90 co-chaperone; Aha1	Negative
<i>aipr-1</i>	C56C10.10	Hsp90 co-chaperone; XAP2	Negative
<i>bag-1</i>	F57B10.11	Hsp90 co-chaperone; Bag-1	Negative
<i>fkf-6</i>	F31D4.3	FK506-Binding protein; Hsp90-interacting protein	Negative
<i>cdc-37</i>	W08F4.8	Hsp90 co-chaperone; cdc37	Negative
<i>trak-1</i>	T27A3.1	HAP1; Hsp90-interacting protein	Negative
<i>tomm-70</i>	ZK370.8	TPR repeat-containing mitochondrial protein	Negative
<i>sgt-1</i>	R05F9.10	PPH-5 homolog; TRP repeat-containing protein	Negative
<i>cct-1</i>	T05C12.7	Chaperonin Containing TCP-1	Negative
<i>hsb-1</i>	K08E7.2	Heat shock factor binding protein	Negative
<i>lit-1</i>	W06F12.1	Serine/threonine-protein kinase NLK	Negative

Table S2. Results of the candidate RNAi screen for *mec-15(-)* suppressors. *mec-15(u75); eri-1(mg366) uls115; mec-7(u278) lin-15B(n744)* animals were fed with RNAi bacteria. If 10% of the treated animals had ALM-PN longer than five cell body length, the treatment was considered to produce positive results.

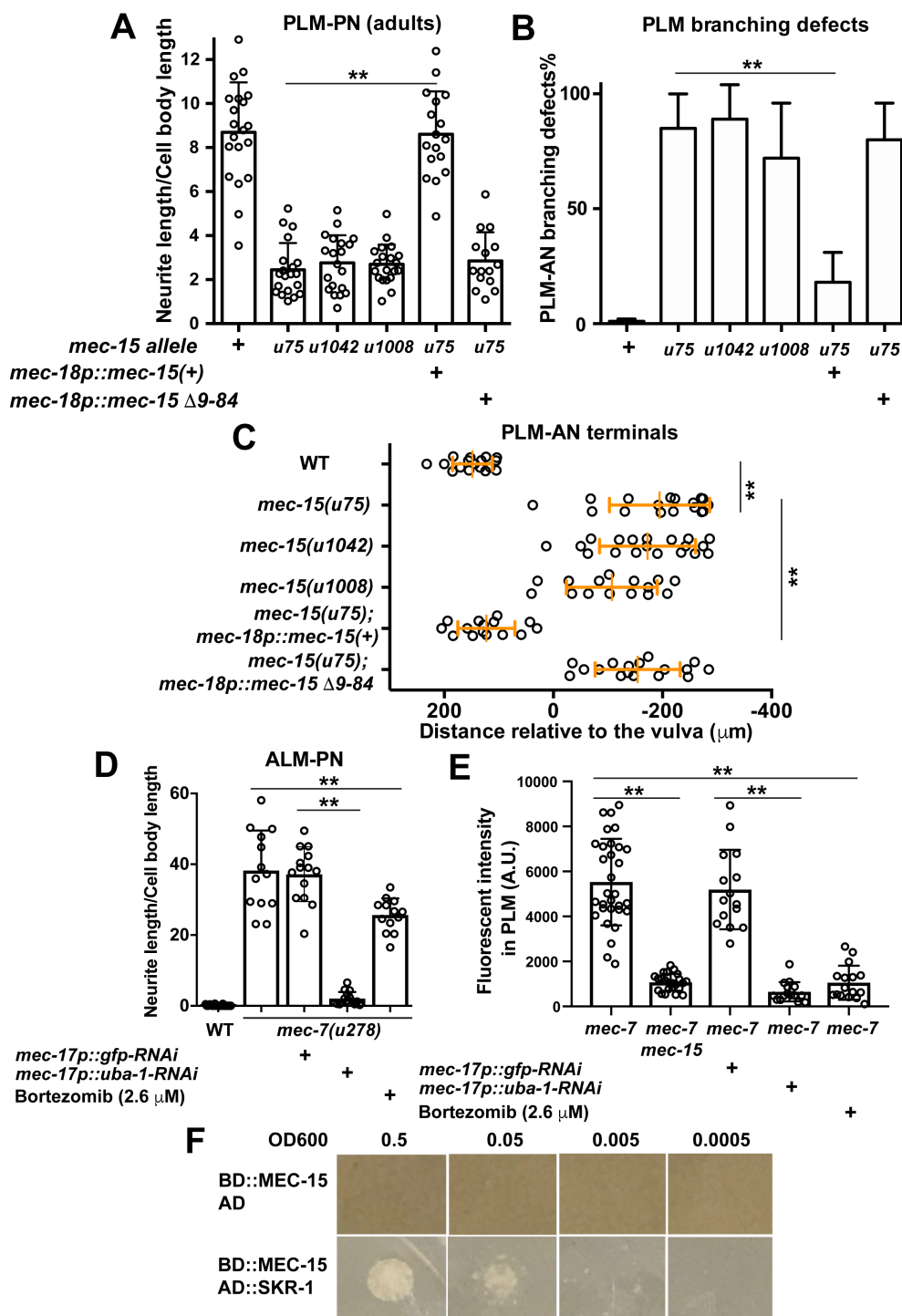


Figure S1. F-box protein MEC-15 is required for TRN neurite development. (A) The length of PLM-PN in animals carrying various *mec-15* *lf* alleles and *mec-15(u75)* mutants carrying the transgene expressing either wild-type MEC-15 (+) or MEC-15 Δ9-84 truncates from the TRN-specific *mec-18* promoter. (B) The percentage of PLM cells with PLM-AN branching defects in various strains. N > 40. (C) The distance of PLM-AN terminals to the vulva in the indicated strains. The distance is positive if PLM-AN grew pass the vulva towards the anterior and negative if PLM-AN did not reach the vulva.

(D) The length of ALM-PN in wild type (WT), *mec-7(u278)* mutants, and *mec-7* animals carrying the transgene expressing dsRNA against *uba-1* from the TRN-specific *mec-17* promoter, as well as in *mec-7* animals grown on plates containing 2.6 μ M bortezomib. (E) TagRFP fluorescent intensity in PLM neurons of *mec-7(u278)* mutants, *mec-7 mec-15(u1042)* double mutants, and *mec-7* animals with *uba-1* knockdown or bortezomib treatment. (F) Yeast two-hybrid assays for the interaction between MEC-15 and SKR-1. AD means the GAL4 activation domain and BD means the GAL4 DNA-binding domain.

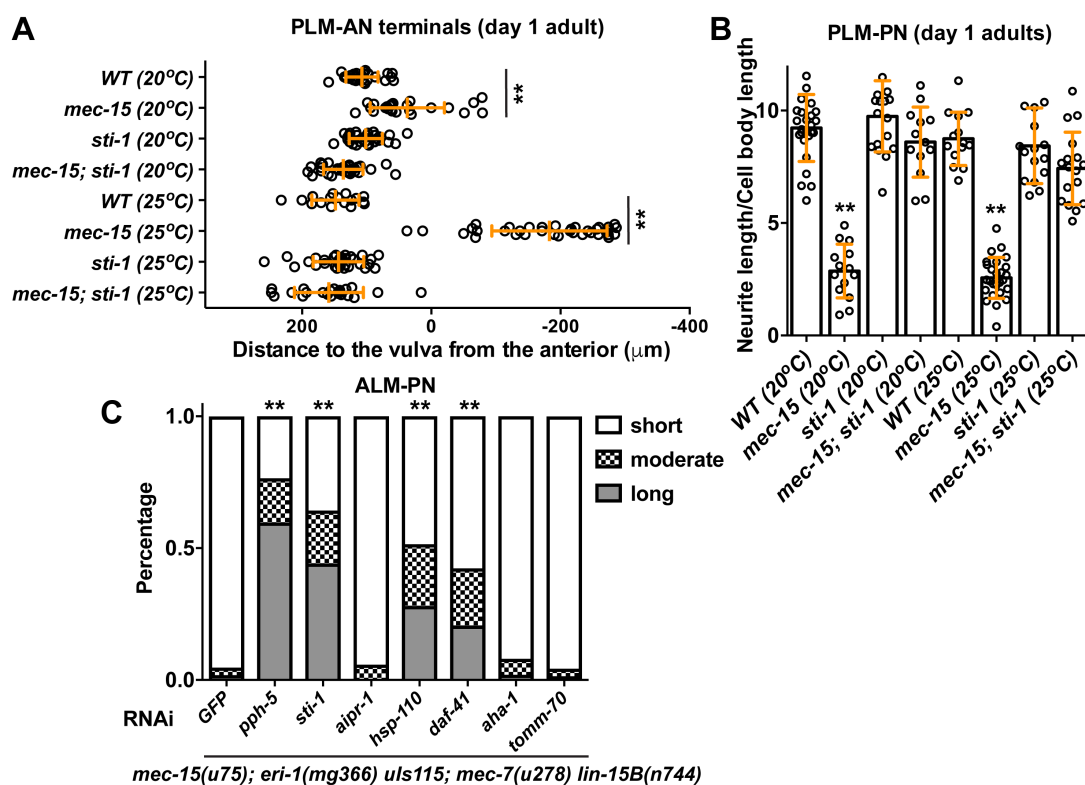


Figure S2. Mutations or knockdown of Hsp90 cochaperones suppress the phenotype of *mec-15* mutants. (A) The distance of PLM-AN terminals to the vulva in wild type (WT) *mec-15(u1042)*, *sti-1(ok3354)* and *mec-15; sti-1* animals at both 20 and 25 °C. (B) The length of PLM-PN in *mec-15(u1042)*, *sti-1(ok3354)* and *mec-15; sti-1* animals at both 20 and 25 °C. Double asterisks indicate statistical significance ($p < 0.01$) for the difference between *mec-15* mutants and the wild type animals. (C) The percentage of ALM cells with short (< 2 cell body length), moderate (2-5 cell body length), or long (> 5 cell body length) ALM-PN in *mec-15; eri-1 uls115[mec-17p::TagRFP]*; *mec-7 lin-15B*; animals fed with bacteria that express dsRNA against the genes indicated. Double asterisks indicate statistical significance ($p < 0.01$) for the difference between the treatment and the RNAi against GFP in a Chi-square test.

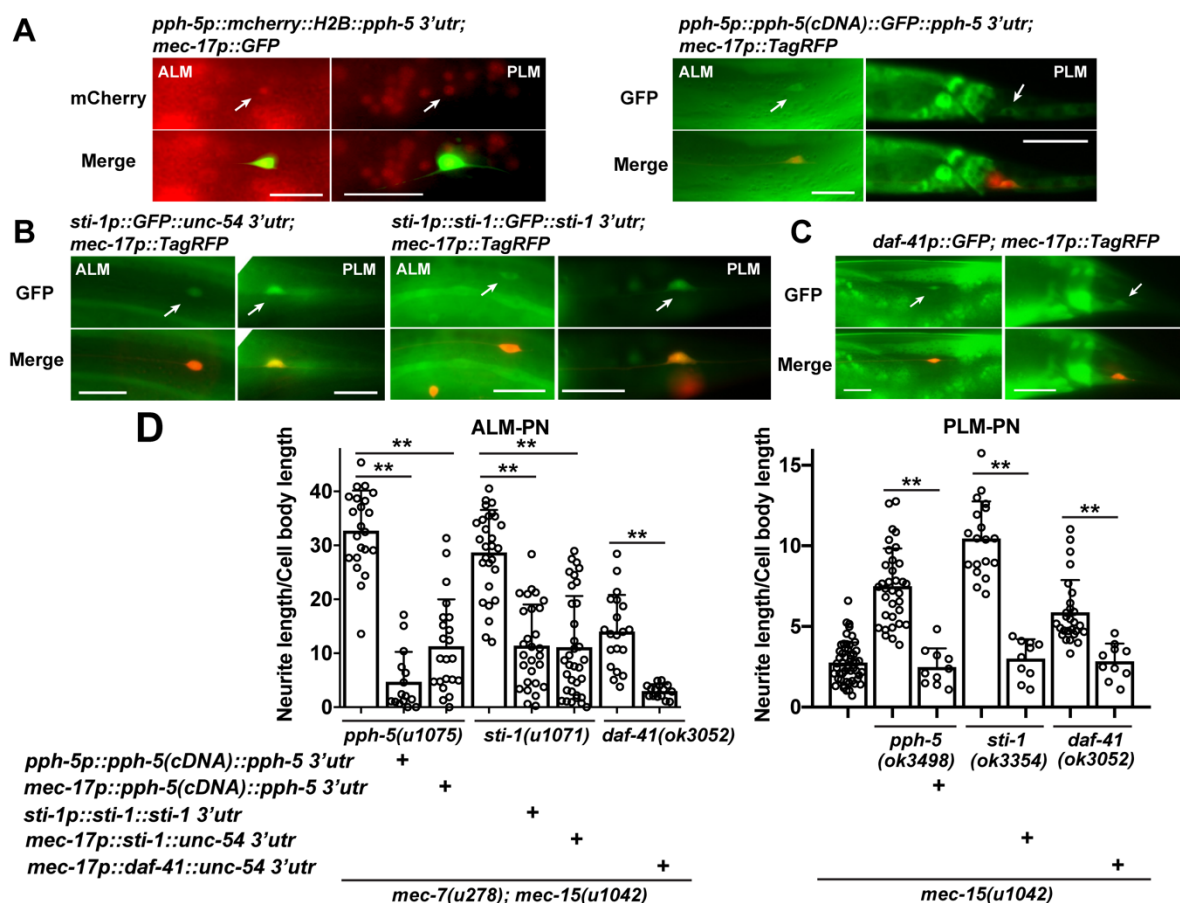


Figure S3. Hsp90 cochaperones are expressed in the TRNs and function cell-autonomously. (A)

The expression of *pph-5* transcriptional reporter *avIs90[pph-5p::mCherry::H2B::pph-5 3'utr; unc-119(+)]* (Richie et al., 2011) and translational reporter *unkEx36[pph-5p::pph-5_cDNA::GFP::pph-5 3'utr; unc-119(+)]* in TRNs. (B) The expression of *sti-1* transcriptional reporter *unkEx52[sti-1p::GFP::unc-54 3'utr]* and translational reporter *unkEx24[sti-1p::sti-1::GFP::sti-1 3'utr; unc-119(+)]* in TRNs. (C) The expression of *daf-41* transcriptional reporter *sEx10796[daf-41p::GFP]* in TRNs. Arrows point to the cell bodies of ALM and PLM. Scale bar = 20 μ m. (D) The length of ALM-PN in the triple mutants that expressed the rescuing transgenes and the length of PLM-PN in the double mutants that expressed the transgenes. Double asterisks indicate statistical significance ($p < 0.01$).

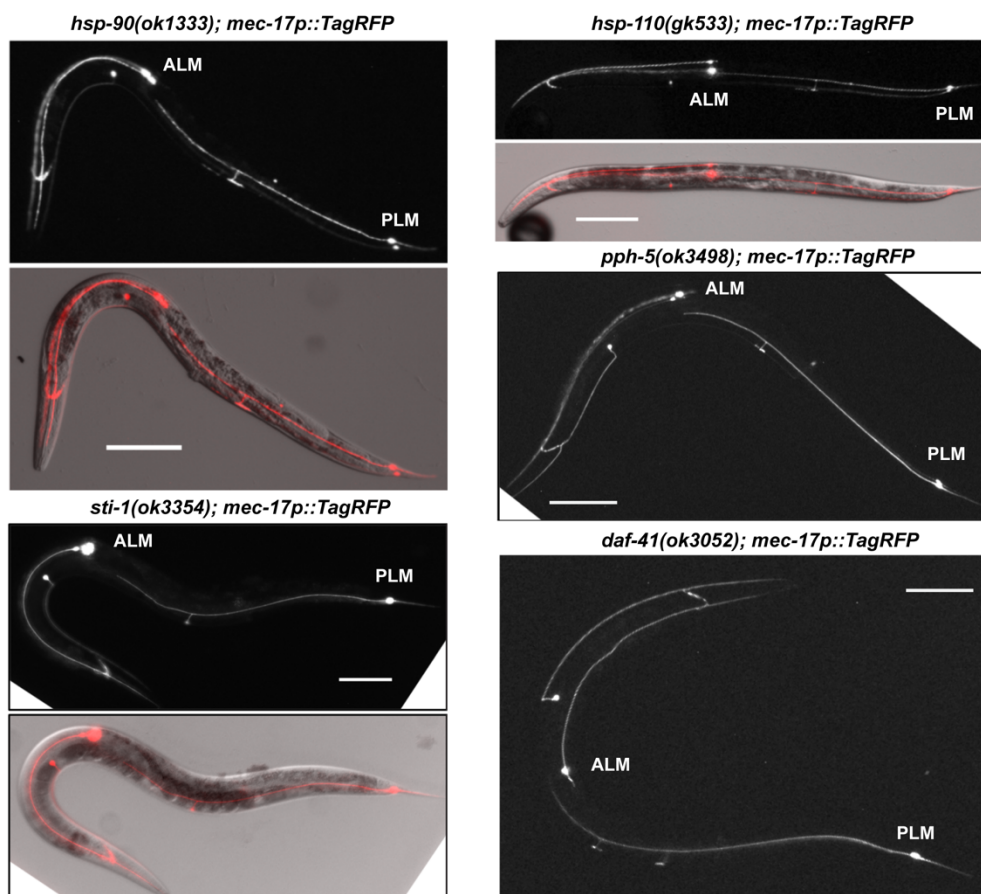


Figure S4. Single mutants of Hsp70/90 chaperones and cochaperones showed normal TRN morphologies. TRN morphologies were visualized by the *mec-17p::TagRFP* transgene in *hsp-90*, *hsp-110*, *sti-1*, *pph-5*, and *daf-41* mutants. *hsp-90* and *hsp-110* homozygotes were the progeny of the balanced heterozygotes *hsp-90/nT1* and *hsp-110/hT2*, respectively. Scale bars = 100 μ m.

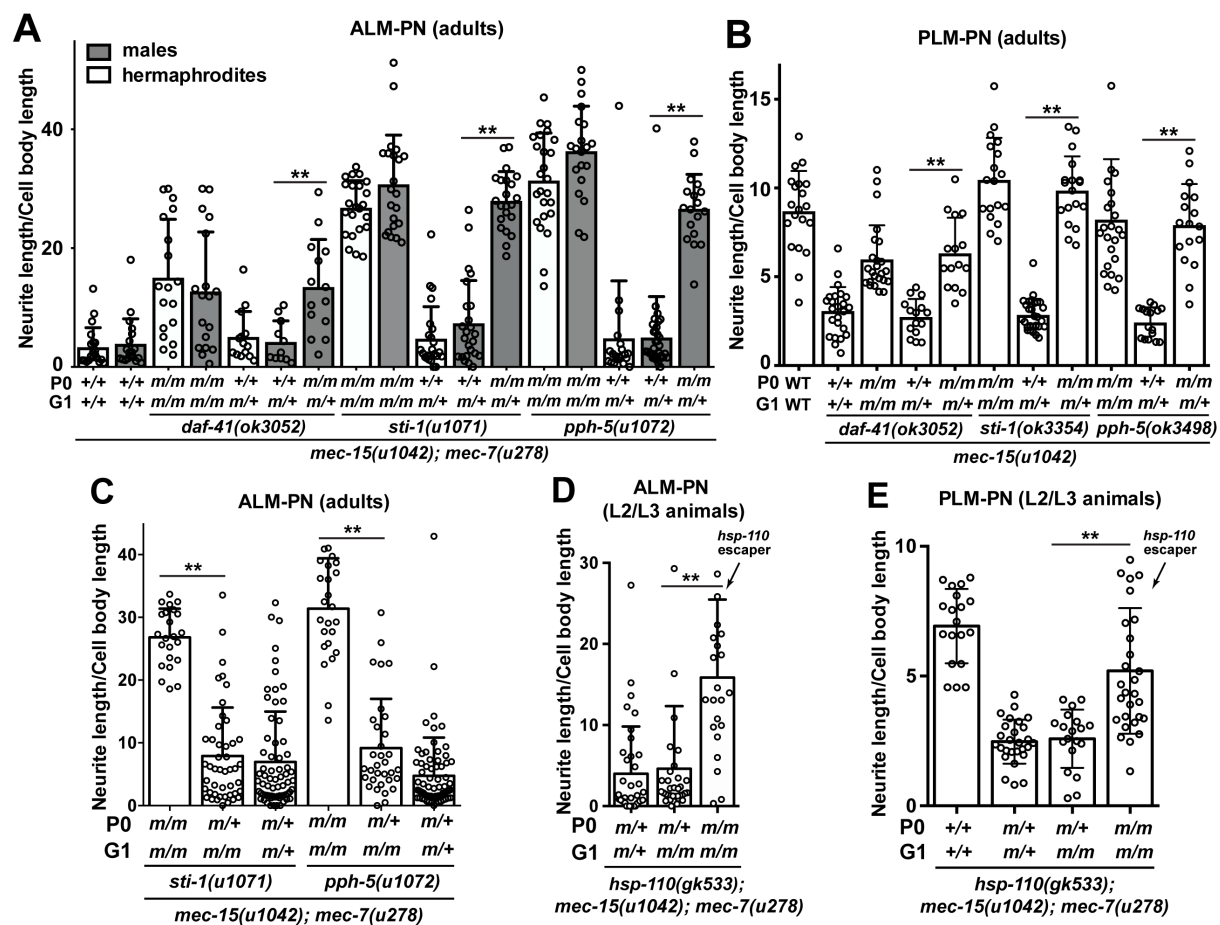


Figure S5. Maternal effects of Hsp70/Hsp90 chaperone and cochaperone mutants. (A) The length of ALM-PN of the heterozygous cross-progeny between *mec-15*; *mec-7* double mutants and their triple mutants with *daf-41*, *sti-1*, or *pph-5* *lf* mutations. P0 indicates the genotype of the mother of the animals (G1 for generation 1) that were examined; “m” represents the mutant allele and “+” the wild-type allele. Crosses to generate heterozygous progeny were repeated three times. (B) The length of PLM-PN of the heterozygous cross-progeny between *mec-15* single mutants and the *mec-15* double mutants with *daf-41*, *sti-1*, or *pph-5* *lf* mutations. (C-D) The length of ALM-PN of the self-progeny of *sti-1*/+; *mec-15*; *mec-7* and *pph-5*/+; *mec-15*; *mec-7* animals and the progeny of *hsp-110*/+; *mec-15*; *mec-7* hermaphrodites and the *hsp-110*; *mec-15*; *mec-7* escapers. (E) The length of PLM-PN of the self-progeny of *hsp-110*/+; *mec-15*; hermaphrodites and the *hsp-110*; *mec-15* escapers. *hsp-110* homozygotes mostly arrested at larval stages.

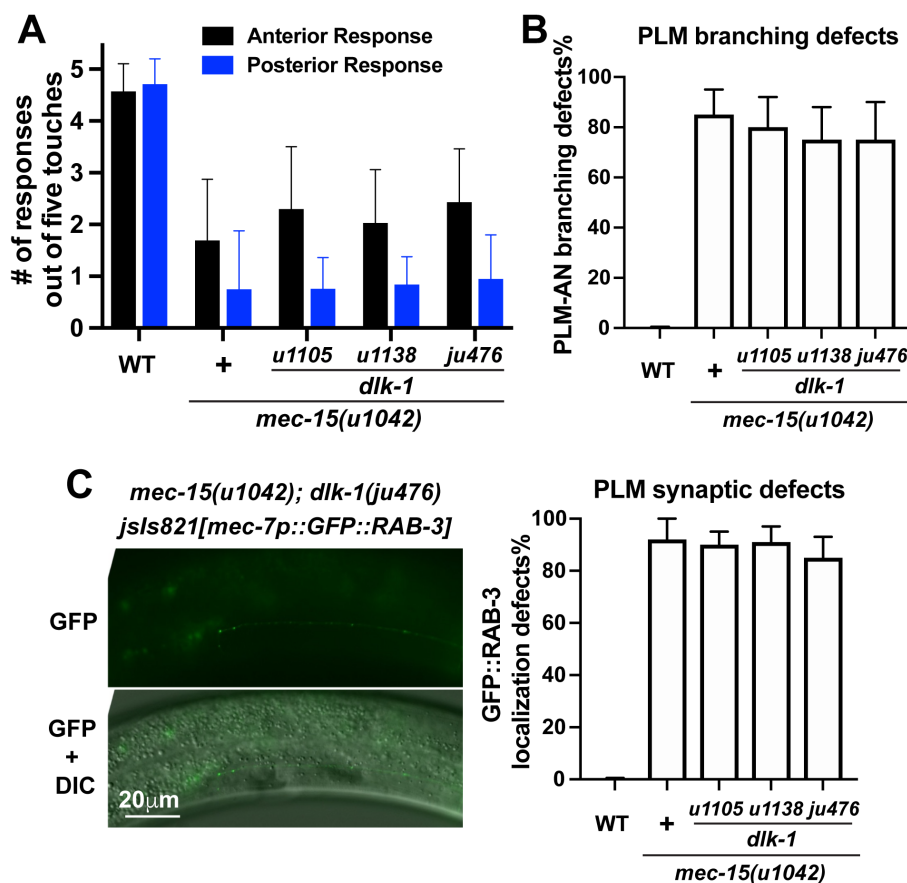


Figure S6. Mutations in *dlk-1* fail to rescue the defects in TRN synaptic development and sensory function of *mec-15* mutants. (A) The number of gentle touch response out of five stimuli in *mec-15* and *mec-15 dlk-1* mutants. (B) The percentages of PLM cells with PLM-AN branching defects in *mec-15* and *mec-15 dlk-1* mutants. (C) Defects of synaptic vesicle (GFP::RAB-3) localization in *mec-15 dlk-1* mutants. The percentage of PLM cells with abnormal GFP::RAB-3 localization in *mec-15* and *mec-15 dlk-1* mutants.

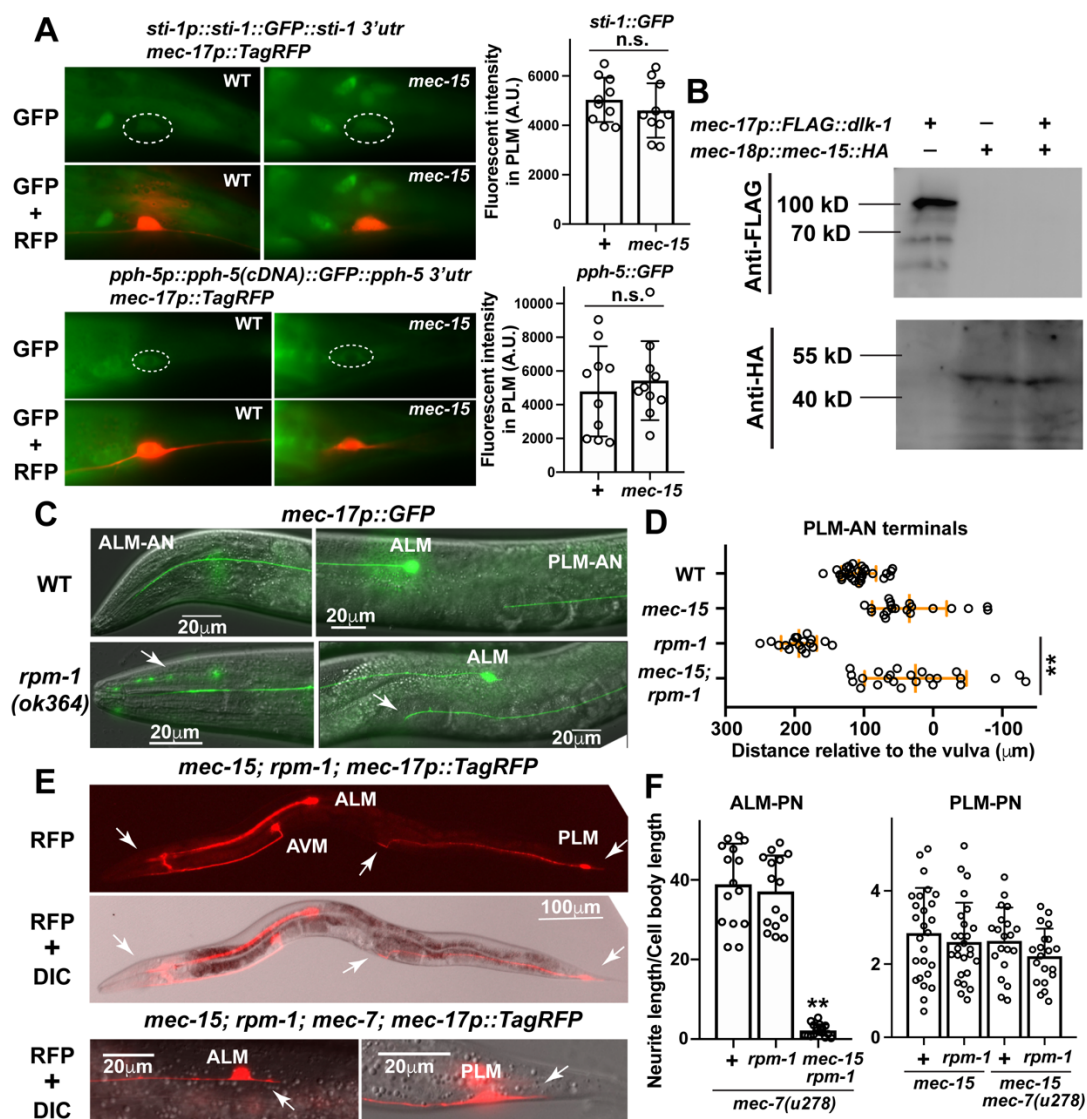


Figure S7. MEC-15 downregulates DLK-1 and is epistatic to RPM-1. (A) Fluorescence of STI-1::GFP and PPH-5::GFP in PLMs of the wild-type and *mec-15(u1042)* animals. Dashed circles enclosed the PLM cell bodies. Quantification of the GFP fluorescent intensity in PLM cells are shown. (B) Western blot with anti-FLAG or anti-HA antibodies with animals carrying transgenes expressing either FLAG::DLK-1 or MEC-15::HA or both in TRNs. (C) Overextension of ALM-AN and PLM-AN in *rpm-1(ok364)* mutants. (D) The distance of PLM-AN terminals to the vulva in *mec-15(u1042)*, *rpm-1(ok364)*, and *mec-15; rpm-1* mutant animals. (E) The shortening of ALM-AN, PLM-AN, and PLM-PN in *mec-15; rpm-1* mutant and the shortening of ALM-PN and PLM-PN in *mec-15; rpm-1; mec-7* animals. Arrows indicate the shortened neurites. (F) The length of ALM-N and PLM-PN in various strains indicated. Double asterisks indicate statistical significance ($p < 0.01$) for the difference between *rpm-1* and *mec-15; rpm-1* mutants.